

DEPARTMENT OF PESTICIDE REGULATION

METHYL IODIDE

EXTERNAL PEER REVIEW PANEL

WORKSHOP

THE PAGODA BUILDING

429 J STREET

SACRAMENTO, CALIFORNIA

FRIDAY, SEPTEMBER 25, 2009

8:15 A.M.

REPORTED BY:

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**ATTENDEES**

**PANEL MEMBERS:**

JOHN R. FROINES, CHAIR  
PAUL BLANC  
KATHIE HAMMOND  
DALE HATTIS  
EDWARD LOECHLER  
THOMAS MCKONE  
RONALD MELNICK  
THEODORE SLOTKIN

**STAFF:**

ELINOR FANNING  
SARAH KOBYLEWSKI

**DPR STAFF:**

MARYLOU VERDER-CARLOS

**PRESENTERS:**

LORI LIM  
NU-MAY RUBY REED

**OEHHA:**

DAVID TING  
ELAINE KHAN

**USEPA:**

JEFFREY L. DAWSON  
ELIZABETH MENDEZ

**ARYSTA:**

BECKY RHODES  
BETH MILESON  
JOHN BUTALA

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**ATTENDEES (CONT.)**

**CALIFORNIA RURAL LEGAL ASSISTANCE FOUNDATION &  
PESTICIDE ACTION NETWORK NORTH AMERICA:**

ANNE KATTEN  
SUSAN KEGLEY

**INTERPRETERS:**

MARIA MUNOZ  
MAURI FITZGIBBON

**INTERESTED AUDIENCE PARTICIPANTS:**

MARILYN LYNDS  
KATHRYN GILJE  
BEN EBBINK  
HUSEIN AJWA  
AMBER WISE  
CAROLINE COX  
JAMES SIMS  
ERIK JOHANSEN  
JIM COCHRAN  
CHRIS VALADEZ  
GINA SOLOMON  
DAVID CHATFIELD  
ELIZABETH MARTIN-CRAIG  
BARBARA LaFave  
ROBERT DOLEZAL  
MANUEL C. CUNHA  
JAMES RANDALL  
PAULA PLACENCIA\*  
GAIL BATESON  
MARTHA GUZMAN  
ALEJANDRA NOLASIO\*  
HORRACIO RAMIREZ\*  
JULIA CRUZ\*  
JOSE AGULAR\*  
FRANCISCO CERRITOS\*  
TOM LA SANDRO  
ENRIQUE HERNANDEZ\*  
DIRGILLIO LOPEZ\*  
SANTIAGO VASQUEZ\*

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**ATTENDEES (CONT.)**

**INTERESTED AUDIENCE PARTICIPANTS:**

MANUEL RAMIREZ\*  
TERESA ESPINOZA\*  
DEROTEO LOPEZ\*  
TERESA DIANDA

(\*Translator being utilized.)

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1 SACRAMENTO, CALIFORNIA

2 FRIDAY, SEPTEMBER 25, 2009, 8:15 A.M.

3 ---oOo---

4 DR. FROINES: We are going to continue the  
5 DPR presentation now. And then OEHHA may show us a  
6 few slides.

7 Is he here?

8 But that will be relatively brief. Then we  
9 will have a half hour for EPA to present. We will  
10 have 45 minutes for Arysta to present. We will then  
11 have Susan Kegley for PAN presenting, and then this  
12 afternoon, in talking to Jim Wells, I got the  
13 impression there may be quite a number of people who  
14 will want to testify at this point. We think, given  
15 the number of people who have an interest in  
16 testifying or presenting material - forget the word  
17 testifying - that we are probably going to limit  
18 people to three minutes this afternoon, but we will  
19 see what kind of crowd shows up. But given what Jim  
20 said, I am anticipating that there will be a number  
21 of people, and so we will go as long as we can go.  
22 And when we are at 4:00, we are closed and that will  
23 be the end of that. And then people who don't have  
24 an opportunity to testify will have to do so either  
25 in writing or when DPR holds a hearing on its

1 registration issue.

2           So that what I'm trying to do is be fair to  
3 everybody in terms of having adequate time to  
4 present their material, but recognizing that there  
5 is an enormous amount of information that is being  
6 discussed and that it makes it difficult. So I  
7 would ask DPR to try and move along as efficiently  
8 as possible. And so let's go from there. At  
9 noontime the panel will meet for lunch and we will  
10 talk about who is going to do what over the next few  
11 months. And so that is that. Here we go.

12           DR. REED: Thank you. Morning.

13           This is the slide where I stopped yesterday.  
14 I think there was a question before I moved on. If  
15 there is any question, if not, I'll go to the next  
16 slide.

17           This is the place where we are talking about  
18 the first mode of action which is fetal thyroid  
19 perturbation due to excess iodide, which causes the  
20 fetal death. So there is two sets of data that are  
21 --

22           DR. BLANC: That may be.

23           DR. REED: That may be; is a possible mode  
24 of action.

25           So this is what I was showing in that if you

1 look at the end, our focus is on really very short  
2 time of exposure. I'm focusing more on the  
3 gestation 23 to 24. You are seeing fetal death  
4 right after the second exposure, which is within 30  
5 hours, with two six hours of exposure. So you see  
6 that it is clear that maternal thyroid was  
7 perturbed, but it wasn't clear in terms of fetal.

8 Slide 32.

9 So in the next slide you're seeing similar  
10 study, except this one would have the side-by-side  
11 sodium iodide study, which is fairly important if  
12 you suppose study was iodide that causes the fetal  
13 death. The difference in protocol is that this one,  
14 the assessment on fetal death, is on gestation day  
15 29, and you're also missing --

16 (Projector mishap.)

17 DR. REED: Will go on. If you need to take  
18 a look on your Slide 33. So, again, the fetal --  
19 maternal thyroid was perturbed. Fetal thyroid was  
20 not at this time. If you look across to the  
21 gestation day 23-26, you are seeing fetal thyroid  
22 being perturbed after four exposures. So gestation  
23 day 23 to 26.

24 But look across to the sodium iodide study,  
25 you are seeing both fetal and with a methyl iodide

1 and sodium iodide you are seeing just as much  
2 problem histopathologically with fetal in terms of  
3 death. You are not seeing a gestation day 29. You  
4 are not seeing sodium iodide causing that much death  
5 as you would with the methyl iodide. So that gives  
6 us an uneasiness about the mode of action,  
7 especially immediately after the second exposure on  
8 this slide and previous slide.

9 DR. BLANC: Of course, all this doesn't  
10 already take into account the fact that we've all  
11 agreed that in any event 20 is not the low effect  
12 level. So just to make sure we remember that from  
13 yesterday.

14 DR. REED: Right.

15 So one more thing I want to point out. All  
16 the fetal data are from the fetuses actually  
17 survived through this. We are not looking at dead  
18 fetuses. And so it's quite amazing that you have  
19 such extent of colloid depletion and hypertrophy on  
20 gestation day 23 to 26. And they're alike. So what  
21 it translates was the concern. After they are born  
22 with such damage, with developmental effect, that  
23 would be since the prenatal developmental toxicity  
24 study ends at gestation day 29. So we don't score,  
25 evaluate, what happened to the fetuses that are so

1 extensively affected.

2 DR. MELNICK: So if you're not seeing the  
3 absorptions with the sodium iodide that you're  
4 seeing with the methyl iodide, are you chasing the  
5 wrong dose metric?

6 DR. REED: And we'll talk about the dose  
7 metrics.

8 DR. MELNICK: The question was: If you're  
9 seeing absorptions with methyl iodide but not to  
10 sodium iodide, are you not chasing the correct dose  
11 metric?

12 DR. REED: That is all together possible.  
13 That is why we are going to progress to the point  
14 where we are actually deciding on the dose metrics  
15 and because the decision on the dose metrics is not  
16 only on the toxicological side of it, but what the  
17 model can do and cannot do, kind of, and the model  
18 validation in terms of whether it validated against  
19 methyl iodide, the parent chemical, or not.

20 So I need to hear from you when we get to that  
21 part.

22 DR. HATTIS: If the model won't give you  
23 the dose metric that you think is best, then why  
24 didn't you fall back on other procedures to  
25 calculate?

1 DR. REED: We do that. Bear with me. That  
2 is something that we grappled with. So that is  
3 great.

4 So this is just a recap because those two  
5 slides could be recapped. Our point of maternal  
6 thyroid picture seems to fit or coordinate better  
7 with the fetal deaths. So here you see a change in  
8 maternal TSH, and in both studies you see the rat on  
9 the first part where fetal part there is no  
10 difference on this first part, gestation date 23-24.  
11 But we are concerned about the persistent of  
12 elevated TSH to gestation day 29.

13 DR. SLOTKIN: I'm sorry, I'm getting a  
14 little confused. If, as it fairly evident from the  
15 last slide, thyroid perturbation is not the cause of  
16 fetal death, what does it mean to then try to pursue  
17 thyroid mechanisms as a way of coming up with your  
18 metric? I understand it is a whole series of slides  
19 now on this, but if this is not the cause of death,  
20 what is the point?

21 DR. REED: Definitely true. One thing is  
22 that because this mode of action is actually the  
23 linchpin for the difference between everybody else's  
24 exposure -- everybody else's risk assessment, and  
25 you will see that if you use fetal thyroid as the

1 mode of action you are going to come up with a very  
2 different HEC. But the reason I am chasing the  
3 thyroid picture and emphasizing on the maternal is  
4 that maternal thyroid perturbation could contribute  
5 to the fetal death, which we don't know. The mode  
6 of action, proposed mode of action was not our  
7 proposal, but it was a proposal out there with the  
8 USEPA and Arysta, is that the fetal thyroid is  
9 perturbed, but it doesn't say maternal is not.

10 DR. SLOTKIN: Is there a literature that  
11 just says that maternal hypothyroid doesn't produce  
12 this fetal death? I have done studies with  
13 propylthiouracil in developing rats, and it doesn't  
14 produce fetal death. It produces gross  
15 abnormalities of brain development and growth and  
16 function, but it doesn't cause death.

17 DR. REED: Right. So that also carries us  
18 to the concern of postnatal effects.

19 DR. MELNICK: Can you go back a slide? So  
20 that if we look again at the TSH, maternal TSH, they  
21 are both the same. So how is that then the  
22 explanation for the iodide in these, change in the  
23 fetus?

24 DR. REED: Uh-huh. Right. I mean -- these  
25 are our sort of questions and issues. The next

1 slide was just a recap of the data, really. Just a  
2 repeat of the data. And the focus is to say that we  
3 didn't think fetal thyroid is the key component for  
4 the early death, but fetal thyroid could have caused  
5 death later on and postnatally, post be a reason for  
6 kinds of neurodevelopmental concerns.

7 DR. FROINES: One problem. The comment  
8 you're getting from everybody about this, what I am  
9 worried about is that we then get into a whole  
10 discussion about HECs when we have said this is not  
11 a relevant pathway.

12 Why should we go to the risk characterization  
13 when nobody's taking seriously the pathways that  
14 you've suggested?

15 DR. BLANC: Just to be specific. What you  
16 mean is risk characterization. John, I think what  
17 you mean or correct me if I am wrong. I understand  
18 you to mean how can we go to risk characterization  
19 using the PK modeling if we don't understand the  
20 mode of action?

21 DR. FROINES: That is correct.

22 DR. BLANC: Because one could go to risk  
23 characterization using cruder safety factors, but  
24 not using modeling.

25 DR. FROINES: Yes.

1 DR. BLANC: Is that correct? Did I  
2 understand you correctly?

3 DR. FROINES: Yes.

4 DR. REED: One thought we have, to answer  
5 that question, or the idea is that you could use  
6 PBPK model or just the PK model to better gauge the  
7 exposure itself and give a default approach of  
8 calculating exposure based on breathing rate and  
9 assuming 100 percent absorption and so forth. So  
10 you can use the model and stop at a certain point.  
11 You don't have to drive it down, all the way down,  
12 to the fetal compartment.

13 DR. BLANC: Why would you use the maternal  
14 thyroid compartment either? What compartment would  
15 you be using?

16 DR. REED: So we don't use the maternal  
17 component either, maternal thyroid component either.

18 DR. BLANC: You would use the whole body?

19 DR. REED: Body, yes.

20 DR. BLANC: You would use the whole body  
21 burden of iodide. How do you know the body burden  
22 of iodide is a good surrogate exposure? You have  
23 elucidated to us what the metabolic pathways are as  
24 we discussed yesterday. So I think everything you  
25 have been presenting to us more and more is

1 convincing, that at least for this outcome one does  
2 not have the data that allows any kind of either  
3 pharmacodynamic or pharmacokinetic modeling.

4 DR. REED: Yes. I hear what you're saying.  
5 We would agree.

6 DR. MELNICK: If you agree, then, can I  
7 suggest that you also provide values based on body  
8 weight scaling to the three-quarter power on  
9 exposure?

10 DR. REED: On exposure, yes.

11 DR. MELNICK: You can provide that because  
12 that is not into here.

13 DR. REED: Yes. I think we did some  
14 calculation already. It's based on our default  
15 approach. So that is the equation that you see in  
16 the document, concentration times breathing rate,  
17 assuming 100 percent.

18 DR. MELNICK: That is not scaling to  
19 three-quarter percent.

20 DR. REED: So the scaling of the animal to  
21 humans will be based on the ratio of the breathing  
22 rate to the body weight, not the body weight to the  
23 three-fourth. So it's in the unit of huminator per  
24 kilogram per day kind of unit. We can certainly  
25 scale back to the three-fourth power on the body

1 weight.

2 DR. HATTIS: If you're going to external  
3 concentrations, essentially what is going on is that  
4 breathing rates go up approximate to the body weight  
5 in three-quarter form. Also, the presumption is  
6 that elimination rates will raise similarly. It is  
7 more or less a wash in terms of external ppm level.

8 DR. REED: Yeah. So with the breathing  
9 rate our rectum human ratio is about 3.5 or so,  
10 defensive. If I'm scaling body weight to the  
11 three-fourth power using the ratio of body rate to  
12 the one-fourth power for rats to humans would be  
13 about four to six. It is not very different, but I  
14 can certainly express that way, yeah.

15 This is summary. We don't need to go through  
16 this again, as a summary of that complicated  
17 information. All I wanted to point out that this is  
18 our conclusion, that the fetal data lack consistency  
19 for this mode of action. Maternal data shows,  
20 perhaps, better concordance, but this is only the  
21 perhaps kind of thing. The uncertainly, as I state,  
22 the fetal data are really from those that survived.  
23 We don't know what happened to those dead ones.

24 Additional data is for making comparisons  
25 between species.

1 DR. HATTIS: Can you go back one slide?  
2 That is a very important statement, the lack of  
3 consistency unlikely. Does staff come up with the  
4 decision about where the point of departure, the  
5 term we would use, and in the interpretation of  
6 appropriate margin of exposure? That really should  
7 be a tight lining when you see a statement like  
8 this. In your effort to characterize the meaning of  
9 exposure, it should then show up in risk  
10 characterization as something that directly feeds  
11 into a choice or recommendation about an acceptable  
12 MOE or a commentary on how the NOEL or LOEL or, what  
13 we would say, a point of departure.

14 DR. REED: Thank you.

15 These are the additional data about species  
16 comparison because there is a appearance that  
17 rabbits are more sensitive. And the question,  
18 whether, how humans could compare to the Rabbits or  
19 to the rats. And so this is a recap of the data  
20 that you have. About 11 percent fetal death at 10  
21 ppm. The rats on the surface looks like it might  
22 not show much of anything regarding fetal death, but  
23 you do have postnatal survival issue. Yesterday on  
24 Slide 15 you also see a body weight issue, and we  
25 talked at length about that. That is at 20 ppm. We

1 are kind of thinking that it's the manifestation of  
2 that effect which caused by methyl iodide that could  
3 be prenatal and postnatal. So if you chop it off at  
4 the prenatal, you might not see the entire picture.

5         These are the data of -- sodium iodide data,  
6 and both of them show survival problem. These are  
7 very severe survival problem. This is pup survival.  
8 This is after birth. In terms of transport, that  
9 was issue with the mode of action. If it is not, we  
10 don't need to go over that.

11         Would that be okay to skip it over.

12                 DR. FROINES: Sure.

13                 DR. REED: We don't want to go through the  
14 other three mode of actions because we want to save  
15 time. We agreed last time not to go over it. I  
16 just want to point out yesterday, I think it was, a  
17 comment made about delay observation or delayed  
18 occurrence that you observed later for after the  
19 exposure. Here is the set of data that you could  
20 have, GSH depletion of 15 minutes afterward. But  
21 way after that, six hours after that, GSH completely  
22 recovered, but that was just coming up afterwards.  
23 So that's important in terms of neurotoxicity.

24                 DR. BLANC: Yeah.

25                 DR. REED: I thought I'd show you this. It

1 is in the document.

2 We are not going to go through this, the third  
3 one. We are not going to go through it. Dale has  
4 some questions about using this type of data for  
5 model of fate evaluations. We do that later or some  
6 other time.

7 Maybe do this, 30 seconds. This is  
8 interesting, what we found in the literature. That  
9 no more rabbit pregnancy patterns; is that the  
10 cholesterol dipped from about day -- at bottom,  
11 about day 21. This is all the way through day 28  
12 gestation day. And we saw that the effects were  
13 just fitting what methyl iodide was doing. Increase  
14 cholesterol, increase fetal dealt, which is late  
15 stage. There is no malformation at all. And  
16 decreasing fetal weight. We don't have anymore data  
17 on methyl iodide to follow that.

18 So the second part that I would go through for  
19 each endpoint is mode of action and then model issue  
20 and then --

21 DR. BLANC: Can we just summarize what  
22 we've just been through? It's important not to lose  
23 the forest for the trees. So what I heard you  
24 present and in your oral comments agreed with what  
25 is, in fact, that there are multiple levels of

1 uncertainty about mode of action of this effect. So  
2 not only did we show yesterday what was initially  
3 taken as a low effect level, was -- as a no effect  
4 level was actually low effect level. We need to go  
5 back even to estimate a departure point. But beyond  
6 that, there is no sufficiently stable scientific  
7 basis upon which to do modeling of the  
8 pharmacokinetic or pharmacodynamic sort.

9 Is that a correct sort of--

10 DR. REED: On the fetal compartment.

11 DR. BLANC: Or the maternal compartment  
12 because you don't know that it is the maternal  
13 compartment. You don't know the metabolism. You  
14 don't know that it is iodide. So, actually, there  
15 is no basis for doing either part of the modeling.  
16 That is what I heard you say.

17 DR. HATTIS: You could do and show the  
18 results of both possibilities. There are three  
19 formal possibilities; either methyl iodide, the  
20 methyl iodide that is the parents or some metabolite  
21 of methyl iodide. And you could -- so you could, in  
22 fact, do total metabolism. You could do total --

23 DR. FROINES: I am not sure that it is that  
24 simple, Dale.

25 DR. BLANC: All it would be is inputs to

1 the model, Dale, since we don't know anything,  
2 basically, is what I've heard. We don't know the  
3 mechanism of action. We don't know what the  
4 measured exposure should be.

5 DR. HATTIS: Basically, we do know that  
6 methyl iodide itself is the post reactive thing that  
7 is going. We know that iodide probably is not the  
8 direct cause of fetal deaths. So I would, in the  
9 absence of that, I would presume either a  
10 concentration of methyl iodide is the most likely  
11 actor causing the fetal deaths. So that is what I  
12 would do as a use of -- as one possible use of the  
13 model, if we thought the model was going to be good  
14 in predicting that, which is not completely clear.

15 DR. FROINES: I actually think that what  
16 you just said clarified your previous comments  
17 because I think your previous comment kind of slid  
18 over that notion of methyl iodide versus iodide, and  
19 I think that is an extremely important point because  
20 it affects everything that we -- every conclusion  
21 that basically follows.

22 DR. MELNICK: It is a little bit of all  
23 what you've been talking about.

24 DR. FROINES: Use the microphone, please.  
25 I feel like a policeman.

1 MR. MELNICK: Because you showed your slide  
2 on cholesterol and that reminded me that this was a  
3 common finding among many of your studies. Elevated  
4 levels of cholesterol and LDL. And I don't see a  
5 risk assessment based on cholesterol. That is an  
6 important endpoint and it is not addressed.

7 Can you provide as well?

8 DR. REED: Yes. Maybe we are sort of  
9 cutting out certain things and certain endpoints  
10 because at the end we are looking at lowest point of  
11 departure, where it would give us the critical point  
12 of departure for risk assessment. And we can  
13 certainly add the cholesterol assessment in the risk  
14 assessment, whether it's a dragging endpoint or not.

15 DR. FROINES: The problem, it is a problem  
16 also, though. If we bring in cholesterol, she  
17 bypassed direct alkylation, also. There is some who  
18 would argue that direct alkylation inhalation,  
19 which, I think, Dale just did, in fact, is an  
20 important pathway. We need to, for the sake of  
21 time, go past things. But for the sake of content  
22 we need to address them.

23 DR. SLOTKIN: I have a point that I would  
24 like to raise about the disconnect between thyroid  
25 status and fetal death. And it looks as though we

1 are going to discard thyroid status. I would  
2 propose that what we should discard is fetal death  
3 as an endpoint. Because in development we always  
4 say fetal death protects you from neurodevelopmental  
5 disorders. And that is not actually a joke.  
6 Because a dose response curve is an inverted  
7 U-shape. And as you get into fetal death, the ones  
8 who survive are the ones who weren't affected.

9       There is a vast literature on hypothyroidism  
10 and neurodevelopment. I don't think anyone would  
11 contest that if you have a hypothyroid mother and  
12 fetus, that there are neurodevelopmental sequelae  
13 even if they weren't specifically studied by Arysta.  
14 It would seem to me you could use thyroid status as  
15 a modeling endpoint all by itself, irrespective of  
16 fetal death, based on the thresholds for thyroid  
17 dysregulation and known dural developmental  
18 outcomes, which would then lower your NOEL or LOEL  
19 vastly from the fetal death. Those numbers are out  
20 there in the literature. I submitted to the other  
21 members of the committee, I don't know, a list of a  
22 half dozen reviews in the last two years on thyroid  
23 dysregulation and neurodevelopmental disorders. It  
24 seems to me that we may be throwing out the right  
25 mechanism while keeping the endpoints.

1 DR. REED: Good point. At the end we are  
2 saying we need to address the thyroid effect, which  
3 we don't have the --

4 DR. SLOTKIN: That could be done because  
5 there is a lot of literature on such degree of  
6 hypothyroidism produces neurodevelopmental delays.

7 DR. REED: I hear you, and I think that is  
8 a very good point, as I said, because that became an  
9 uncertainty for us and actually we can do something  
10 about it.

11 DR. HATTIS: Associated with both of those  
12 is fetal weight reduction.

13 DR. REED: Dale did an analysis on the  
14 fetal weight decline of dosage response.

15 DR. FROINES: We don't seem to have, but we  
16 can get them afterward.

17 DR. SLOTKIN: I sent them a couple weeks  
18 ago.

19 DR. FROINES: A communication problem up at  
20 this table.

21 DR. REED: It is my question to the panel  
22 if we want to go through some model issues. We  
23 don't think running the model is the way to go. We  
24 can cut the model issues out. But, Dale, if you  
25 think there is merit on using the model to

1 characterize the exposure and not the mode of action  
2 then we can go on.

3 DR. HATTIS: I think there is still merit  
4 in potentially exploring the model, but I don't  
5 think we need to take up time today. Basically,  
6 what I want to be sure is if the unchanged methyl  
7 iodide is hypothesized to be one plausible dose,  
8 causal dose, then we want to make sure that the  
9 model is reasonably well-calibrated to predict that,  
10 either peak concentrations or AUCs. And the way to  
11 do that is to use the hemoglobin adduct information  
12 that is in the Sweeney paper in conjunction with  
13 either a measure or an inferred rate like the  
14 constant of the alkylation of the hemoglobin  
15 cysteine. The rat has this hemoglobin cysteine that  
16 other species generally don't. Because we have  
17 these experiments in rats, and we have this  
18 measurement of hemoglobin -- alkylation of the  
19 hemoglobin cysteine, that should be able to be  
20 interpretable in terms of an AUC, if we only have  
21 the rate constant. That could be measured or  
22 estimated from other information that a better  
23 chemist than I could possibly do.

24 DR. REED: We also have adducts in the  
25 rabbits, data on male rabbits, not rat. So whether,

1 how much background we have on the kinetics on  
2 rabbits. We can look into that on later.

3 DR. FROINES: That is important.

4 DR. REED: So what I am going to go through  
5 in terms of model is just -- not the model itself,  
6 not particularly for deriving the HEC for  
7 uncertainty and endpoint yet. Because I just want  
8 to go through some basics of concerns that we have.  
9 So when we review the model, we find certain  
10 parameters that are important and have great impact  
11 on the outcome of the model.

12 One is the alveoli of ventilation rate.  
13 Initially was 12 liter per hour was used. That was  
14 for nonpregnant rabbits. That was because part --  
15 of it is because it fits the measurement data on  
16 fetal iodide. And since the model was initially  
17 designed to better characterize the fetal part,  
18 based on the proposed mode of action, but when I  
19 look at it I go, you know. It has to be  
20 physiologically based, and we wanted to run at 20.  
21 These are the little tweakings that we consider as  
22 important. And actually it fits the maternal data  
23 better than the right QAC.

24 We are also concerned about the model estimate  
25 that results in estimates of higher fetal to

1 maternal ratio. So if you have higher fetal  
2 compartment iodide, and you use that mode of action  
3 as dose metrics, you are going to raise the HEC in  
4 an unsupported way. So we are concerned about.

5 We are also concerned about what the model  
6 shows in terms of fetal thyroid iodide accumulation.  
7 So in rabbits is one thing. But humongous,  
8 humongous amount of iodide is accumulated in humans.  
9 We don't know that integrity of that part of the  
10 model in terms of whether it is true or not. If it  
11 is true, wow, that is a concern. So it shows in the  
12 our document.

13 The fourth point is that we don't know what is  
14 the mode of action. The model was based on fetal  
15 weight of .6 pounds, which is the end of first  
16 trimester. So if you run the model at six pounds,  
17 later stage, how would that make the outcome  
18 different in terms of the parameters you are looking  
19 for? So there is some data here showing that if you  
20 run it at the late stage pregnancy you are going to  
21 change the fetal parameter into almost 50 percent  
22 higher. So that was a concern.

23 Our conclusion is that there is greater  
24 modeling confidence with maternal serum iodide or  
25 methyl iodide, those metrics, which is a better

1 description of what exposure is without considering  
2 any mode of action.

3           So now we're turning into looking at using  
4 PBPK. Just to characterize the amount of internal  
5 dose, the amount of exposure, and compare that -- in  
6 document is says we can compare that to  
7 conventional, straightforward calculation.

8           There is another issue about the dose metrics.  
9 Is whether the model is single date increase or  
10 incremental change. Our sense is that we want to  
11 model for single day because yesterday you saw the  
12 model has limitation of poorer prediction of  
13 measurement data beyond one day. You really cannot  
14 model more than one day. Also, it takes about 13 to  
15 14 days for the study state to be reached. With the  
16 window of vulnerability is only four days.

17           DR. BLANC: What would the translation be  
18 if one assumed that this time period of  
19 vulnerability was correct, at least in terms of time  
20 period? Even if we don't understand the mechanism  
21 of action or the equivalent duration of the time  
22 period of vulnerability in the human fetal  
23 development, is there a cross?

24           DR. SLOTKIN: I don't know the relationship  
25 to rabbits. I am a rat man.

1 DR. SLOTKIN: Let's say it was for a rat,  
2 two day period.

3 DR. SLOTKIN: In a rat you would basically  
4 feel that a day in the life of a fetal rat is sort  
5 of a week in the life of a fetal human.

6 DR. BLANC: Is that taken into account,  
7 just out of curiosity, in your model?

8 DR. REED: The model is mechanically giving  
9 you what you want. And so yesterday we talk about  
10 rabbits born at about end of second trimester. And  
11 I think rats are different in terms of --

12 DR. SLOTKIN: Rats are a little earlier.  
13 Sort of middle second trimester at time of birth.

14 DR. REED: The straightforward answer is  
15 no. But we use the model to gauge the exposure.

16 DR. BLANC: I see.

17 DR. HATTIS: It should also be noticed that  
18 if you have methyl iodide as the relevant cause of  
19 dosimeter, that might well achieve steady state for  
20 approach and given percentages of steady state much  
21 faster than iodide. Iodide takes the longest time  
22 to be eliminated by the urine. I don't know  
23 iodide's half life, but bromide has half life of  
24 about eight days.

25 DR. BLANC: Not in humans. Much longer, I

1 think.

2 DR. HATTIS: You think it is longer?

3 DR. BLANC: Much longer.

4 DR. HATTIS: That is what I remember  
5 studying in '70s or so.

6 DR. BLANC: I think -- I don't want to  
7 stake my life on that, but I believe it is quite a  
8 bit longer.

9 DR. FROINES: Can I stop you here? Can you  
10 hear what he just said?

11 You guys try and do better.

12 DR. SLOTKIN: Half life of bromide in  
13 humans is 12 days.

14 DR. FROINES: Paul, maybe that one is  
15 better for you.

16 (Microphone problems.)

17 DR. REED: If we continue to entertain to  
18 use the PBPK Model, this is what we come with. The  
19 HEC would be .024; and if you use maternal methyl  
20 iodide, it been would be .74, I believe. It is in  
21 the document in table, I think --

22 Lori, please find it.

23 In the document, what I have is using the  
24 eight combinations of dose metrics. So that was for  
25 purposes of discussion. I figure you would want to

1 see that. So that is what it is. What I wanted to  
2 point out is that comparison to our HEC, USEPA's HEC  
3 is 7.4. There is a lot of difference, and that sort  
4 of impacts on the final conclusion of the risk  
5 assessment.

6 Lori just find it. It is Table 8-A4 where I  
7 lined up all the HECs.

8 DR. BLANC: In the appendix.

9 I think you've made the point; the point being  
10 that even -- well, several points I think you made  
11 very clear. One is the areas in which there are  
12 very substantive lack of certainty.

13 And secondly, the fact that, again, you're to  
14 be commended that even taking an approach where you  
15 make certain presumptions which are at least not as  
16 ill-founded as those of the federal EPA. You have a  
17 -- you come up with a value even so that is quite a  
18 bit lower and more public health protective. But  
19 even that is unlikely to be public health protective  
20 enough, given the uncertainties that you have  
21 underscored fairly elegantly.

22 DR. REED: If we are talking about the  
23 sensitive individual later.

24 DR. BLANC: Right. I'm certainly not  
25 worried based on what I'm hearing from you, that

1 having read your document and read your response to  
2 USEPA, that for some reason their argument is the  
3 more sound one. It certainly is not convincing.

4 DR. REED: Move on to the next endpoint.  
5 This is just -- you have in your document, the nasal  
6 effect. Again, I am lining up and starting with  
7 mode of action and what is available, what is our  
8 conclusion on that.

9 It could be coming from direct inoculation.  
10 It could be coming from -- I think proposed. In my  
11 discussion I said that I don't think a clear support  
12 of what mode of action it is. But we do have pretty  
13 good data on glutathione depletion in the nasal  
14 epithelium at 2,500 ppm. And you could also  
15 attenuate that degeneration by replenishing the  
16 glutathione or depleting glutathione. So our  
17 conclusion is that glutathione depletion is likely  
18 an early marker and not necessarily as a key event  
19 to the degeneration, but you could use it as a  
20 marker that is associated with degeneration. And so  
21 in terms of model, if that could be agreed on, we  
22 could use glutathione depletion as a marker as dose  
23 metrics.

24 DR. FROINES: I am just so tempted to -- I  
25 think the notion of a marker is a very interesting

1 one. The issue of glutathione as a causative agent  
2 one should also have on the record so that we are  
3 aware that there are these different possibilities  
4 to explore.

5 DR. REED: That is what I am. So what we  
6 are doing is, to find human HEC we are going to use  
7 the PBPK model to model a level of depletion that we  
8 consider as protective for the possible endpoints  
9 from glutathione depletion. Glutathione depletion  
10 as endpoint, that point is well taken. Not use the  
11 marker.

12 DR. FROINES: That also gives us, I guess  
13 -- let's let it go. What I was going to say that  
14 GST issues are also obviously complicated in terms  
15 of who has it and who doesn't. But let's leave that  
16 for now.

17 DR. REED: That is a part of the sensitive  
18 population. That is great.

19 DR. FROINES: As long as we recognize that  
20 this GST issue is important. The GST issue is  
21 important aside glutathione itself.

22 DR. REED: Yes. That is great. We are to  
23 the last slide in terms of calling out the important  
24 issues. And I really appreciate.

25 So in terms of glutathione depletion or next

1 issue came up was that at what percentage of  
2 depletion do we want to model for. There is a  
3 proposal out there that's saying that 50 percent  
4 depletion is good enough as equivalent of no  
5 observed effect level. And our opinion is that 50  
6 percent is way too high. And the reason is that the  
7 available data within the methyl iodide database, 48  
8 percent of depletion one hour after a hundred ppm  
9 exposure. That is evident with early degeneration.  
10 So we felt 50 percent is just not correct number.  
11 It is not protective enough.

12 We also see about 35 percent depletion at 25  
13 ppm at six hours. We also notice that it's time  
14 dependent. So we need to look at a number of hours  
15 of exposure and not just look at the concentration.

16 At the NOEL of 21 ppm and that NOEL change - I  
17 think we talked about it yesterday. But at 21 ppm,  
18 25 percent depletion; 34 percent depletion at  
19 [unintelligible] also in this region. So our  
20 conclusion is that at 25 percent regional  
21 glutathione depletion is a good marker for no effect  
22 of human nasal epithelium.

23 DR. BLANC: Just to clarify. If you -- and  
24 I thought your argument was convincing about why 25  
25 percent was more protective standard than 50

1 percent. But if you believe that and if at the 21  
2 parts per million there was 34 percent reduction in  
3 one of the sites that was measured, doesn't that  
4 argue against another argument against 21 parts per  
5 million as being a no effect level, since you are  
6 seeing by our own definition an effect?

7 DR. REED: It could be. But it's a little  
8 tricky. I am deriving the 25 percent from this  
9 study, and because we call it no effect, and so I am  
10 saying, okay, what would the percentage of depletion  
11 at no effect level. So I'm saying the model shows  
12 50 to 25, 34. So I couldn't go back and say that 34  
13 proves that 21 is not good enough. I don't know if  
14 you understand.

15 DR. BLANC: In terms of what your  
16 presumptions are. It is something to take into  
17 consideration.

18 A couple other questions. I believe it was in  
19 your documents that you discussed glutathione  
20 depletion at the mitochondrial level as being a  
21 potential mechanism of disease or did I see that  
22 somewhere else, of toxicity? If not, I have  
23 certainly seen that discussed on a theoretical  
24 basis. And if that is -- if there is any general  
25 toxicology literature on that, I think it would be

1 worth addressing it. Because you're not measuring  
2 glutathione at the mitochondrial level. And when  
3 you measure the rebound level, you don't know  
4 whether that rebound is at the expense of the  
5 mitochondria. I think that that is another argument  
6 in favor of the kind of margin of safety you are  
7 talking about.

8 John, do you have any experience in that?

9 DR. FROINES: No. I'm hesitant to take a  
10 position on this, but the -- I think the  
11 mitochondrial issue brings in all sorts of questions  
12 that would take us a long time to discuss. But,  
13 clearly, the mitochondrial issue in terms of  
14 everything from oxidative stress to what other MOEs  
15 one might talk about are relevant, and we should  
16 remember that we are not talking about a NOEL today,  
17 given what we talked about yesterday.

18 And so that -- but the issue of the  
19 mitochondrial issue is one that, to me, deserves  
20 much more investigation. The problem I think that  
21 we'll end up with is that there is not going to be  
22 enough data to really draw conclusions. That is  
23 where the weakness comes. I'm not sure what we have  
24 to work with in that regard.

25 DR. BLANC: I thought this was the one area

1 where I thought you were on a little bit firmer  
2 ground, especially because you weren't committing  
3 yourself to mechanism of action, but committing  
4 yourself to the glutathione reduction as being a  
5 very consistent correlative of toxicity we are  
6 seeing. I thought on that basis for this endpoint  
7 this piece of the document was more logical to me  
8 and, especially, the approach you took in terms of  
9 25 percent rather than 50 percent, which I thought  
10 was a little cavalier.

11           One other very small thing. When you go back  
12 and are doing very fine edits, when I read this  
13 section, reading it quickly I guess, it is really  
14 easy to get tripped up as the reader on the 25,  
15 misinterpreting the 25 percent reduction as  
16 reduction to 25 percent. So if you could put a  
17 parenthetic, e.g., 75 percent remaining, that would  
18 be helpful.

19           DR. REED: Great. Got it.

20           DR. MELNICK: I have three issues which  
21 give me a little discomfort on the glutathione  
22 issue.

23           DR. FROINES: Can I say one more thing  
24 before you go to yours? I am a person who believes  
25 very strongly that chemicals like a methyl iodide

1 end up having significant thiol chemist, whether it  
2 is binding with thiol groups on proteins, whether --  
3 binding with glutathione is one sulfur that we know  
4 happens. But this is a complicated issue in terms  
5 of thiol. Thiol chemistry is obviously -- so I  
6 think that we have to take very seriously the notion  
7 with respect to the mitochondria that there is thiol  
8 chemistry going on with methyl iodide. And Dale  
9 talked about methyl iodide as the key operative  
10 agent. And what I want to say is that I think the  
11 chemistry of methyl iodide and its ability to  
12 potentially bind with sulfidro groups or thiol  
13 groups is something that needs to be taken very,  
14 very seriously. And it takes us into asking the  
15 question: What is the cemetery of concern that we  
16 might have -- we might hypothesize as being  
17 important? And we really -- these documents really  
18 don't address that chemistry. Nowhere.

19 So I would put it as something for further  
20 consideration. And I think the chemistry is going  
21 to end up being interesting. It seems fairly  
22 straightforward to me that we are dealing with  
23 issues that glutathione depletion is a reflection  
24 that some sulfur group is binding with the methyl  
25 group, and that is an issue of toxicity that is

1 potentially mechanistically important.

2 DR. HATTIS: Just want to reinforce that  
3 slightly. That it is not only sulfide groups that  
4 methyl iodide react with. That you can also get  
5 reaction with methionine, another sulfur-containing  
6 amino acid, to get a dimethylsulfonium ion, which  
7 could be a secondary methylate. That was the  
8 concern with methyl bromide a few decades ago. I  
9 don't know that anybody has measured that reaction.

10 DR. FROINES: The chemistry -- we also have  
11 to think about these things in terms of pH. We are  
12 talking about thiol and thiolate, and you're going  
13 to get different levels of reaction depending upon  
14 what the pH of the medium that you find yourself in.

15 So that my point is just to reinforce the  
16 complexity of this issue.

17 DR. MELNICK: I enjoyed your argument.  
18 There are still three issues that are causing me  
19 some concern on using the 25 percent glutathione  
20 depletion as dose metric. One is based on your  
21 figures, the level of glutathione in the olfactory  
22 epithelium of rats is much higher than the olfactory  
23 epithelium of humans. It is approximately four  
24 times higher. So if you deplete by 25 percent,  
25 you're still three times higher in the rat than in

1 the human. And if you want to use that, I can  
2 understand it, but I think you might need an  
3 adjustment for susceptibility because human  
4 olfactory epithelium does have the same level as the  
5 rat.

6         The percent depletion of glutathione with  
7 similar olfactory epithelium and the respiratory  
8 epithelium, that there was no degeneration caused by  
9 methyl iodide in the respiratory epithelium. So  
10 that I think it is a marker, but again not all.

11         Thirdly, when you start to use this for  
12 comparing doses across species, the model, as you  
13 indicated, was based on glutathione data on two  
14 doses, of which one of them didn't fit very well.  
15 Therefore, to me, the model is not reliable for  
16 extrapolations to other exposure levels. So this  
17 gives me a little bit discomfort. I am not sure  
18 which are better, but there are some significant  
19 issues.

20             DR. REED: That is a great point.

21         One thing is that when we come to  
22 interspecies, the first point that you were making,  
23 interspecies extrapolation, there is a  
24 pharmacodynamic factor in terms of response to  
25 certain level depletion that may not ultimately take

1 care of your concern about the low baseline with  
2 humans. But there is a threefold pharmacodynamics  
3 that sort of apply, too, with the last issue.

4 DR. MELNICK: Olfactory and respiratory?

5 DR. REED: Yes. The only comfort that we  
6 have is because we are right at the range for the  
7 model. Model was being evaluated at 25 ppm, and we  
8 were modeling 91. We thought we can sort of make  
9 use of model even though you're right a hundred ppm  
10 off. So that was sort of -- it doesn't fully  
11 address your concern, but I think your concern  
12 should be and we will document it so that it is  
13 clear what are the then uncertainties in this.

14 Thank you.

15 DR. FROINES: Can I raise what I think is a  
16 serious issue? It is after 9:00 right now. And  
17 Elinor just mentioned to me that we have over 20  
18 slides left to go. And we have OEHHA, and we have  
19 all the other people who are waiting expectantly to  
20 testify. And so I don't know how to address this,  
21 but somehow we have to move those 20 slides faster  
22 or eliminate something. I don't -- DPR is clearly  
23 the agency we want to hear from at the this meeting.  
24 That is our job. And so I don't want you to cut out  
25 anything that you consider highly relevant, but also

1 I'm aware that 20 slides and 9:00 create a problem  
2 for us. And so whatever you can do at this point to  
3 speed, to move more quickly will be helpful.

4 DR. REED: I will and Lori will, too,  
5 because later those slides are just what you can  
6 read from the documents in terms of calculation,  
7 real number in terms of risk. And we can cut that  
8 out.

9 Thank you.

10 What this slide I am showing, what is our HECs  
11 and what is USEPA's HECs. The only thing I want to  
12 mention is that you notice that with USEPA' HEC,  
13 this one, nasal effect becomes the lowest HEC of all  
14 the acute HECs, and that impacts the conclusion of  
15 the risk assessment a whole bunch. So I just want  
16 to highlight that.

17 DR. BLANC: Whereas, yours were already an  
18 order magnitude lower because of the other endpoint  
19 you were using.

20 DR. REED: Right. At the end, when we look  
21 at the uncertainty of risk assessment, our focus is  
22 on that lowest one, how uncertain. But USEPA will  
23 be focusing on this one, being able to protect  
24 against all the other endpoint.

25 This is just a graph.

1 Neurotoxicity. We don't know the mode of  
2 action, so you can read about it. We are using  
3 methyl iodide area under the curve in the brain, and  
4 the difference is that USEPA will be using the peak  
5 methyl iodide in the brain. And because the peak is  
6 really within 30 minutes, we feel it is important to  
7 use area under the curve to take care of the -- to  
8 account for the differences between six hours of  
9 exposure in rats and 20 hours exposure to humans.

10 DR. BLANC: This is acute neurotoxicity  
11 which you define as the movement frequency outcome.  
12 We have already yesterday determined that there was  
13 not a LOEL. So the actual number would -- this  
14 again, just to clarify. Again, this is an acute  
15 outcome. We are not talking here about chronic  
16 neurotoxicity because you actually don't have any  
17 studies that are appropriate for that endpoint.

18 DR. REED: Yes.

19 DR. FROINES: I would defer to Ted Shotler  
20 -- Slotkin on this. Sorry, Ted.

21 DR. SLOTKIN: I have been called worse  
22 things.

23 DR. FROINES: But I just want -- I want to  
24 reemphasize that we have this study that we have  
25 been talking about, and we talked about the LOEL,

1 NOEL issue that Paul just raised. But I just want  
2 to add my perspective that we are dealing with a  
3 very important chemical for which there is very  
4 little neurologic toxicity data available to us.

5 DR. BLANC: In animals studies. With a  
6 great deal of neurotoxicity data in human case  
7 report.

8 DR. FROINES: There is a great deal of  
9 human data in human case reports. That is extremely  
10 important in my view and I think every other. But I  
11 also want to say that I wish we had more animal data  
12 because we are dealing with a very small number  
13 here.

14 DR. SLOTKIN: Just a question. Since the  
15 endpoint is acute neurotoxicity, again, there is a  
16 lot of literature about proportional metrics that  
17 you can use to adjust for chronic neurotoxicity  
18 versus acute and for developmental neurotoxicity,  
19 which is even more sensitive than chronic  
20 neurotoxicity in the adult.

21 So is there a way, perhaps, of bringing in  
22 extra factors based on going from acute to chronic  
23 and going from chronic adult to developmental?

24 DR. REED: It is possible. In risk  
25 assessment we use uncertainty factor to take care of

1 that. Except that we have to look at period of  
2 exposure, so we don't extrapolate from acute to the  
3 chronic, so we have to sort of give some  
4 justification consideration for that. There is a  
5 mechanism for doing that. I am not going to show  
6 you, talk about this.

7 DR. FROINES: I do think that this issue of  
8 uncertainty factors should probably come up at some  
9 point. Because --

10 DR. BLANC: She is going to get there.

11 DR. FROINES: I can't see behind myself.

12 DR. REED: I am following your order to  
13 speed through.

14 About uncertainty factor. We have three HECs.  
15 Let's not worry about the numerical value. So by  
16 default we usually apply uncertainty factor of ten  
17 for interspecies differences. And assuming that  
18 PBPK Model is adequate to account for the kinetic  
19 differences in animals, we would be applying a  
20 factor of 3 or 3.16 which is the square root of ten.  
21 We were retaining the inter individual differences.  
22 This is to cover pharmacokinetic and pharmacodynamic  
23 differences within human population. So there is  
24 some sensitive people and so forth. This is not to  
25 say that the factor of ten fully account for

1 sensitive populations. So keep that in mind with  
2 the differences.

3 We are proposing additional uncertainty factor  
4 of ten. I think we went through the issue  
5 extensively. We are considering how to address  
6 neurodevelopmental concerns, and the reason is lack  
7 of data because gestation day 29. And we also see  
8 maternal thyroid effect. And Dr. Slotkin already  
9 mentioned that. So that is our concern.

10 We also are concerned about the fetal thyroid  
11 effect from the histopath and also thyroid  
12 perturbation indication that persisted to gestation  
13 day 29. Without that data, what happened after day  
14 29, we feel uncomfortable. There is -- we are  
15 talking about the difference between methyl iodide  
16 and iodine. In this case there is some postnatal  
17 death with excess iodine, and there is not a whole  
18 lot of dose response data for us to address, but  
19 that's certainly something to consider.

20 And you are going to see in the slide that we  
21 specifically consider excess iodine from methyl  
22 iodide. So that increase total volume for --

23 DR. BLANC: Can you clarify for us on the  
24 very first line the interspecies factor of ten from  
25 a PKPB [verbatim] model.

1 DR. REED: So if we don't have model, we're  
2 using conventional method of calculation, just doing  
3 the adjustment of those between humans and animals.  
4 Then we would apply a factor of ten. If we have  
5 taken care of pharmacokinetic, so we only have  
6 pharmacodynamic left. It is square root of ten.

7 DR. BLANC: Right. So I certainly would  
8 say one of the themes that's emerged this morning is  
9 that there seems to be such a lack of certainty that  
10 I doubt you are going to be able to get a level of  
11 three, and foremost of your calculations you are  
12 going to be using a level of ten.

13 DR. REED: This is even coming from HCP,  
14 direct from PBPK Model.

15 DR. BLANC: I think so.

16 DR. HATTIS: Because we don't know --  
17 first, we have some concerns that whether the PBPK  
18 Model has been -- predictions have been compared  
19 adequately with human data. And second, we are not  
20 quite clear that that's been done for all of the  
21 dose metrics of interest.

22 DR. REED: That is clear.

23 DR. BLANC: Secondly, since you're asking  
24 for feedback, I think that certainly there is  
25 precedent for using additional level of uncertainly

1 factor of ten as you proposed, with the third ten in  
2 this algorithm. And I think that what you have  
3 presented, and our discussions clearly support, that  
4 a level of conservatism. I don't know how others  
5 feel, but panel members should address this at this  
6 point because --

7 DR. HATTIS: The Food Quality and  
8 Protection Act, which was done in the '90s at some  
9 point, does mandate an extra factor often unless  
10 data are sufficient to allay neurodevelopmental,  
11 neurotoxicity concerns. I think that is the case  
12 here.

13 DR. HAMMOND: You are going for three as  
14 opposed to ten.

15 DR. HATTIS: I am saying that the Food  
16 Quality and Protection Act factor of ten is  
17 indicated here.

18 DR. REED: We're referring to this  
19 additional uncertainty.

20 DR. HATTIS: That is what it is.

21 DR. BLANC: I think that would be another  
22 thing there, Dale, on your comments on whether a ten  
23 is more appropriate than three on the top row.  
24 Based on what we are hearing, I would have to say it  
25 is leaning toward ten in most of the situations.

1 DR. HATTIS: Although you could go back to  
2 three for sure if you do a ppm versus ppm  
3 conversion. That is another way of taking into  
4 account if you are doing milligram per kilogram then  
5 full ten is indicated. If you are doing ppm in air  
6 versus ppm in air, that is because of the offsetting  
7 nature of changes in breathing rates and elimination  
8 rates that is equivalent.

9 DR. REED: I think our position slightly  
10 different in that when we do the conventional  
11 calculation and we come up human equivalents, even  
12 though we accounting for the breathing rate  
13 differences, we don't think that fully accounted for  
14 the [unintelligible] kinetic difference in humans.  
15 We still applied the conventional to it. But if it  
16 is coming off from the pharmacokinetic model,  
17 assuming that the model is adequate, we would take  
18 the three off. In that response Paul was saying  
19 that is not adequately addressed, so you need to put  
20 back the ten.

21 DR. BLANC: Yes.

22 John, do you have any comments on that? I'd  
23 be curious to hear them. Did I lose you?

24 DR. FROINES: You didn't entirely lose me,  
25 but I was thinking about something else. But I am

1 trying to figure out how we deal with uncertainty  
2 factors. We are going to have a meeting where we,  
3 where the panel spends the entire time talking about  
4 all the issues that have come up over the last two  
5 days. And, obviously, one of the major issues is  
6 going to be what are the uncertainty factors that we  
7 think are appropriate, relative to what we have seen  
8 from DPR and EPA and Arysta.

9           And so that I'm not sure that this is the  
10 moment when we should get into that. But I can say  
11 without, unequivocally, that given the paucity of  
12 data that we have had to work with, that another  
13 safety factor of ten is absolutely -- seems to me to  
14 be relevant.

15           DR. BLANC: That wasn't what we were  
16 talking about. It is hard because you are looking  
17 at the slide. We talked about the additional safety  
18 factor of ten. And I don't think anybody has argued  
19 about the individual value of ten, the very first  
20 line that ten versus three. And my point is that I  
21 have -- there is such lack of data in terms of these  
22 modeling steps, that I think rather than say we have  
23 taken care of such amount of uncertainty from  
24 modeling when we use the factor of three there, I  
25 think the default position of ten is going to be the

1 most likely one.

2 DR. FROINES: I think there is a larger  
3 issue of uncertainty factors. But in terms of this,  
4 I would argue that ten is the more appropriate  
5 value.

6 DR. MCKONE: I am concerned, basically,  
7 based on what we said yesterday. When you use  
8 uncertainty factors to derive virtually safe dose,  
9 the implication is that you're confident that we can  
10 define a safety dose. I think if we go the  
11 alternative of point of departure with a margin of  
12 exposure, we are not implicitly saying it is safe,  
13 but we argue what would be a reasonable margin of  
14 exposure to give us confidence. And the reason that  
15 is important is that what we have heard a lot about  
16 is our uncertainty. I think when the uncertainties  
17 are large, we are not even sure we know the right  
18 mode of actions, when the uncertainty come into that  
19 level, defining or even implying that we know a safe  
20 dose is a little misleading.

21 To say we use that knowledge to define a  
22 margin of exposure is a little more in line what we  
23 are seeing. We are not saying that it is safe. We  
24 are just saying that we need reasonable assurance  
25 that the dose is low enough to probably be below

1 what we think are a critical point of departure. A  
2 little different approach. This is -- EPA is moving  
3 a bit in that direction. The National Research  
4 Council is trying to push them much harder in that  
5 direction. To the extent we can do that, it would  
6 be more useful, more informative. But, again, that  
7 is sort of late. Coming in later. It really ties  
8 in with the lengthy number of times yesterday we  
9 brought up point of departure in place of NOEL or  
10 LOEL.

11 DR. FROINES: That is part of the reason  
12 why, when Paul asked me about three versus ten, I  
13 was kind of hesitant on it. Because it seems to me  
14 that the issues are larger in some respect than this  
15 specific issue here.

16 DR. MELNICK: I want to reinforce what Paul  
17 said. We have seen enough information presented  
18 here to say that the modeling is not reliable for  
19 the purpose of removing that three from the  
20 pharmacokinetic aspect of value ten.

21 DR. REED: So another alternative is not to  
22 use the model at all and go by what Ron was saying.  
23 Use the conventional calculation and retaining the  
24 ten and ten and the additional uncertainty because  
25 model is uncertain. And if model is uncertain, then

1 there is no merit of using the model that creates a  
2 different set of uncertainties.

3 DR. FROINES: I think that is what you are  
4 hearing. You are hearing that from everybody who  
5 spoke.

6 DR. REED: We can move on. I am going to  
7 show you our concern for excess iodide from methyl  
8 iodide and in terms of total body burden. This is  
9 what we come up with in terms of criteria for iodide  
10 intact. National Academy, IOM, has the recommended  
11 daily allowance and also the tolerable upper bound.  
12 ATSDR set one to 14 days minimum risk level. So  
13 when we look at this and compare to the body burden  
14 of methyl iodide, we are not looking for acute  
15 effect. Because the way our risk assessment falls  
16 out, is that acute point of departure is the lowest.  
17 So people might argue and say, well, this set of  
18 criteria might not be pertinent for single day  
19 exposure, because the acute point of departure is  
20 lowest. We use the lowest point of departure to  
21 show that even at that level and with all  
22 uncertainty factor applied, when the exposure to the  
23 young people, we're using one to three, is  
24 representation. The total body burden would of  
25 concern if we don't have additional factor of ten.

1 DR. BLANC: Also, does this take into  
2 account the rather alarming of water leaching data  
3 that was included as well.

4 DR. REED: These are the route of exposure  
5 that we're worrying about. In my calculation I did  
6 not -- the water, as you see yesterday with 18  
7 milligrams per liter -- I think Dr. Slotkin, was  
8 you, came up with a -- I think in our calculation we  
9 have, like, four, five orders of magnitude. And so  
10 that is a given.

11 DR. BLANC: I think your document needs to  
12 clarify that because it wasn't very clear in the  
13 actual document as written. It seemed to have not  
14 taken that into account.

15 DR. REED: We have 47,000 folds exceeding  
16 or something.

17 DR. BLANC: You didn't talk about that in  
18 the document.

19 DR. REED: Not in the context of extra body  
20 burden. If, we talk about it, we don't need to talk  
21 about body burden.

22 DR. BLANC: It needs to be there, doesn't  
23 it?

24 DR. REED: So we are concerned in terms of  
25 assessing the body burden. We have concern that the

1 sort of background level you get from drinking  
2 water, not from methyl iodide in drinking water, but  
3 table salt, so forth. You can read that in breast  
4 milk there is also iodine, and infants have extra  
5 intake of iodide. Because of that we are  
6 considering this additional iodide from methyl  
7 iodide. This is for inhalation exposure it's --  
8 Dr. Blanc says we cannot even add this component  
9 because it far exceeds. That is end of that.

10 So it is important at this point to bring out  
11 where the sensitive individuals, just for some of  
12 the effects that we noted up to this point.

13 DR. BLANC: I do want to say something  
14 about that. That is a very cursory list of  
15 scenarios for sensitive subpopulation. So I think  
16 it would be worth it for you to revisit that and  
17 just make sure that you -- or else say that these  
18 are very limited examples. We realize there are a  
19 lot of others.

20 One of the things that struck me, given the  
21 use of acetaminophen in the population and it's  
22 known glutathione depletion effect, I would say  
23 anybody who uses acetaminophen could be a  
24 susceptible subpopulation. By the way, I think  
25 susceptible is better than sensitive, which implies

1 allergic mechanism, which you don't need to apply  
2 here. I just use that as a one example.

3 Also, nutritional compromise, which is  
4 probably also important, is a major issue in terms  
5 of glutathione stores, and other chronic illness.  
6 So if you had some not young -- not young person,  
7 normal genetics, but they're getting chemotherapy,  
8 they're going to be glutathione depleted.

9 DR. REED: Thank you.

10 So my last slide. So this is just a sketch of  
11 how uncertainty factors are used. You can use  
12 uncertainty factor when deriving reference  
13 concentration. That is the criteria for what should  
14 be in the air. By using quantitative departure  
15 divided by uncertainty factor. We also calculate  
16 margin of exposure using point of departure divided  
17 by exposure. And so, essentially, if you look at  
18 these two equations, whatever the uncertainty factor  
19 that you would use deriving the air criteria, would  
20 be the uncertain -- would the margin of exposure  
21 that you would want at the end of the risk  
22 assessment to use that as gauge to see if that the  
23 risk is successful or not, based on human health.

24 So this is our calculation. We were using  
25 three and ten and ten and using .24 as the lowest

1 point of departure. It would come up to be 1 ppp.  
2 To know what that 1 ppp is in terms of extra body  
3 burden we did the calculation. It said about 42  
4 micrograms per day. That is using the short term  
5 USEPA exposure from handbooks, breathing rate for  
6 this age group. You all know that there is 2008  
7 exposure factor handbook for children, particularly.  
8 And that one has for short term, that one, and we  
9 talked about this yesterday, activity levels that  
10 make a difference. So if I were assuming that was  
11 12 hours of sleep time and then 12 hours of live  
12 activity, this calculation would be right about 60  
13 micrograms per day instead of 42. So that number is  
14 higher.

15         So in that comparison we are saying if people  
16 are in their background achieving the daily  
17 recommended allowance from the National Academy and  
18 from the tolerable upper boundary, we minus that,  
19 take that component out. You sort of have a space  
20 of 110 micrograms per day. That is rough  
21 theoretical calculation or thinking to gauge whether  
22 this 42 or 60 microgram per day would be excess body  
23 burden. That is not acceptable.

24         So if you use the same thing as ATSTR uses,  
25 same equation, you come to this range. By applying

1 that uncertainty factor we think that we might be  
2 able to stay within the recommended criteria out  
3 there.

4 DR. BLANC: However, as we have seen from  
5 the rest of the discussion, that is not true at all.  
6 Because, for one thing, it is not going to be .24  
7 ppm. It is considerably lower than that. You are  
8 going to be divided by a thousand, not by 300.

9 So, in fact, this is irrelevant. And the fact  
10 we don't know that iodine is the direct metric to  
11 use and, anyway, that would be overwhelmed by the  
12 potential water exposure. So this is sort of not  
13 relevant in this point.

14 DR. REED: The reason I am going over this  
15 is to perhaps receive some comments from you in  
16 terms of methodology. At the end, we would have an  
17 endpoint and so forth. And so we would still need  
18 to go through some kind of evaluation and this is --

19 DR. BLANC: My recommendation would be that  
20 the uncertainty factors are not high enough. The  
21 exposure critical value is way to high because it's  
22 not a NOEL. It is a LOEL. And that I am not  
23 convinced that the endpoint in box number three in  
24 terms of iodide is what you should be using.

25 So that is my feedback.

1 DR. REED: More specifically if I could get  
2 comments on using this upper bound tolerance and the  
3 recommended daily allowance. Is a space where I  
4 could gauge whether any exposure from methyl iodide  
5 would be exceeding sort of safe level as body burden  
6 of iodide to address that issue?

7 DR. BLANC: It doesn't seem that logical  
8 for this endpoint because the mechanism of action  
9 that we are talking about. I think that you  
10 certainly should go through a calculation with your  
11 ground water contamination issue.

12 DR. REED: So the approach for calculation  
13 might be okay to use when I get to groundwater.

14 DR. BLANC: Or if you had some other  
15 endpoint for which you believe the mechanism of  
16 action was the iodide level.

17 DR. REED: In this case, mechanism of  
18 action is a separate consideration. Even if we have  
19 the mechanism of action, even if we have addressed  
20 the neurodevelopmental effect, it is our feeling  
21 that iodide body burden for methyl iodide should be  
22 addressed, anyway. Because there is a standard out  
23 there to say do not exceed this level. And so  
24 methyl iodide is one thing, and it has toxicity. We  
25 need to look at iodide intake.

1 DR. SLOTKIN: This is actually just a  
2 safety check against the known guidelines for iodide  
3 intake as to whether the calculation you come up  
4 with lies within the safety factor for what we know  
5 iodide has to be.

6 DR. REED: Right.

7 DR. BLANC: One thing you might do to your  
8 document, which would be as informative, would be as  
9 a way of underscoring the lack of public health  
10 protection from the proposed EPA numbers as to run  
11 them this way.

12 DR. REED: I see.

13 DR. BLANC: Because you will show the error  
14 is not protected or to come up with a default value  
15 which is no matter what we are working with, if we  
16 don't come out below this, we'd have to say it's  
17 unsafe. Not because of any specific mechanism of  
18 action of inference. We are looking because of the  
19 thyroid, because of iodide burden that you would be  
20 getting.

21 DR. HATTIS: I would just add one caveat.  
22 Because iodide build up over many days or weeks, you  
23 ought to use appropriate time period, residence time  
24 for iodide.

25 DR. REED: That is what I mentioned

1 earlier, that we are using the acute point of  
2 departure because it has lowest number. But so if  
3 you go back and look at document, the chronic and  
4 subchronic are actually higher. So when we get to  
5 longer period of exposure, that is also going to be  
6 an iodide issue, apart from the endpoint issue.

7 DR. BLANC: I understand it better now.

8 DR. SLOTKIN: That's particularly important  
9 because the figure for human iodide half life is two  
10 months.

11 DR. REED: So that is it.

12 DR. BLANC: Thanks.

13 Dr. LIM: If okay with the panel, I am  
14 going to skip through most of my slides, just making  
15 key points regarding how the calculations are made  
16 rather than what it means since the questions about  
17 NOEL, the mode of action and level that is  
18 uncertainly factors that are needed.

19 DR. FROINES: How long do you think you are  
20 going to take?

21 DR. LIM: I only present three slides.

22 DR. FROINES: We will finish. We need to  
23 be able to take a break. And I actually think this  
24 last discussion, I want to emphasize that all of  
25 what Paul was saying and Ted was specifically

1 designed to help in the next phase of the document.  
2 And that I think both people would in essence  
3 compliment you for what you have done at this point.  
4 So I think it -- please feel a positive atmosphere  
5 from this.

6 DR. REED: We do. I actually do.

7 DR. FROINES: Let me just say. I'm sorry  
8 to interrupt. Elinor and I have gotten so far 41  
9 people who want to testify this afternoon.

10 DR. BLANC: Speak.

11 DR. FANNING: Up to 50.

12 DR. FROINES: So we are in serious  
13 difficulty. And there will be no speakers at the  
14 afternoon who will be able to speak longer than  
15 three minutes. And I don't think we are going to  
16 finish. And so I just say that to warn people that  
17 we have a problem that there is no way around it,  
18 because people have to leave. And we want to make  
19 sure that we hear from OEHHA, EPA, Arysta and PAN  
20 And so we are going to just plunge ahead and do the  
21 best we can this afternoon, but it is going to be a  
22 difficult afternoon, I think.

23 That is an important thing for people. The  
24 room is relatively full right now. People can  
25 submit written comments until October 12. And so if

1 one is not able to testify today --

2 DR. BLANC: Speak.

3 DR. FROINES: I keep doing that, I'm sorry.

4 -- speak, please, please get materials into  
5 DPR and then to us by October 27th.

6 So go ahead.

7 Dr. LIM: The three slides I want to show,  
8 this is the first one. We talked about not using  
9 PBPK Model and using the default method. On Page 2  
10 if the Appendix B is a full equation of how you  
11 would calculate the HEC. This is a shorter version  
12 here. Using NOEL just for the breathing rate using  
13 default PK animal factors, if the panel would like  
14 to calculate HEC for acute endpoints, which I did  
15 not do using the B format, the equations provided in  
16 the document for that calculation.

17 And on this slide is equations for the  
18 monitoring exposure and cancer risk equation.  
19 Again, these are also in the Appendix B. So the  
20 monitoring exposures, HEC divided by human exposure  
21 level. That is what I meant by eventually  
22 everything comes down to numbers.

23 DR. BLANC: When you do the human cancer  
24 potency factor risk estimation, will you be using  
25 the -- including the default of factors developed by

1 OEHHA or childhood exposure?

2 DR. HATTIS: This is the extra factor.

3 Dr. LIM: Age adjusted factor --

4 DR. HATTIS: Age adjusted for early month.

5 DR. LIM: We don't have -- I didn't do that  
6 in this document. What I did was I used the adult  
7 exposure level assuming a lifetime exposure with the  
8 person sitting in -- living the same place entire  
9 life time. So the acute default part is the big  
10 component of that lifetime.

11 DR. MELNICK: That certainly isn't like  
12 exposure. That is why there is an adjustment made  
13 on them.

14 DR. REED: We didn't use adjustment factor  
15 because before this discussion we thought that the  
16 weight of evidence in terms of not knowing  
17 genotoxicity, genotoxic mechanism, may not be just  
18 for us to use additional factor for children, which  
19 is three age brackets, factor of ten for the first  
20 part, so forth.

21 DR. FROINES: Can I comment?

22 DR. BLANC: Let her finish.

23 DR. REED: I am finished. So that we  
24 could do that.

25 DR. FROINES: I want to make a point,

1 speaking as a chair of the Scientific Review Panel.  
2 Because the Scientific Review Panel approved the  
3 OEHHA guidelines on cancer risk assessment. And the  
4 children's issue is one area where there is a  
5 designated uncertainty factor applied. So it's  
6 consistent with state policy that such uncertainty  
7 factor be applied.

8 DR. BLANC: So I'm glad to hear that you  
9 will be go back and using that.

10 Thank you.

11 Dr. LIM: And so I just want to show this  
12 slide. This shows Slide 63 from Table 49. This is  
13 how to look at -- what happens with the numbers,  
14 after you give the MOE numbers. So this shows the  
15 three endpoints we looked at for acute and would  
16 have adult, children and infants. So the fetal  
17 endpoint was applied to the adult population with  
18 MOE of 0.8. And from that Dr. Reed's talk, just  
19 --based on what we have, existing NOEL and existing  
20 uncertainty factor that really needs to be 300, at  
21 least, the 0.8. That is how we look at these  
22 numbers.

23 On the far right we talk about the lifetime  
24 risk using the two methods, the threshold method,  
25 which gives you modern day exposure. That is 1,300.

1 If you only use a target of 300, that is okay. Then  
2 implies the cancer risk is okay. But then if you  
3 look at the potency factor of approaching, not  
4 threshold approach, that is only nine times ten to  
5 the minus fifth. The target we want to reach is one  
6 in a million. So, again, that cancer risk is too  
7 high.

8 I just want to put that up so when you look at  
9 these numbers, that is how they are looked at. Not  
10 necessarily to say that we have the right target  
11 point at this point.

12 DR. BLANC: The only other thing I come  
13 back to, I think we have gone through a fairly  
14 exhaustive list of some of the factors you need to  
15 take into account, but, based on our conversation  
16 yesterday, I think it was in the exposure  
17 presentation, not your presentation, by Kathie  
18 brought up the issue of the 90 percent exposure  
19 reduction factor that was used for worker exposure,  
20 had our questions about that. But then following  
21 that, we heard that, in fact, there were a whole  
22 subset of workers for whom there was no respiratory  
23 protection anticipated because they weren't  
24 applicators, they were tarp players with and whole  
25 puncher and so forth.

1           So I think the worst case occupational  
2 exposure scenario needs to be revisited, because  
3 that will change your calculations, even though what  
4 derives your worker bystander calculation is mostly  
5 the flux, which is why your level is so much lower  
6 for drip irrigation. So I am not sure if it will  
7 change your numbers very much. Certainly, I think  
8 that the worker exposure estimates are off by a  
9 factor of ten for a substantive -- the calculations  
10 actually weren't presented for them, but need to be  
11 made without presuming respiratory protection since  
12 none seem to be applied to them.

13           DR. HAMMOND: Just following from that. I  
14 totally agree with -- Paul said this correctly. The  
15 comment I heard also was that respiratory protection  
16 was what the label says, respiratory protection  
17 factor of 90. But any practicing industrial  
18 hygienist says that is not what they actually  
19 deliver. And given it is very hot in the Central  
20 Valley, that's an issue. And the difficulties in  
21 actually having a good respiratory protection  
22 program in an industrial setting, the whole idea of  
23 doing that in an agricultural setting is very  
24 challenging.

25           It is a mistake for those of us responsible

1 for the health of Californians to be thinking about  
2 this protective in a manner that is in ideal  
3 conditions. We need to treat it the way it is  
4 actually going to happen.

5 DR. FROINES: That was brought up  
6 yesterday, and it needs to be reinforced, because we  
7 live in the real world. We don't live in a world  
8 where labels guarantee success. And that we need to  
9 make sure that we have some confidence that there is  
10 going to be effective protection.

11 DR. REED: As a comment, I think my  
12 understanding is that the occupational exposure is  
13 based on the label. So for those who are not  
14 wearing respirators, I think the exposure level is  
15 calculated based on not having a respirator. So I  
16 need to go back and take a look, but I hear your  
17 point. Things have to be addressed in real world  
18 versus --

19 DR. HAMMOND: Let me back up. What I said  
20 yesterday, and I want to make sure we don't forget.  
21 Is at the very, very least I would like to see entry  
22 for -- what the entries for what the concentrations  
23 expected are without a respirator at all. And then  
24 if you are put on respiratory protection, I would  
25 strongly urge you not to use 90 days. To use 90 as

1 a protection factor, that is not realistic. You may  
2 be able to find people who have been working  
3 agricultural workers.

4 Another issue that is of some concern, I don't  
5 know. I'm going back a the exposure thing, is all  
6 of the studies that were done where they measured  
7 workers' exposures were done when the temperatures  
8 were under 25 degrees Celsius. Given the months in  
9 which the applications occur are August, September  
10 and October, it would seem to me that higher  
11 temperatures ought to have been included in that  
12 evaluation. I don't really know what the  
13 temperature is on the methyl iodide concentrations  
14 that one might expect. There might be an  
15 underestimation. I understand you didn't do those,  
16 but at least --

17 DR. REED: Is important. I think  
18 distinction between using air model which you do the  
19 worst case simulation, and that is for the general  
20 public, for the off-site migration. But you're  
21 referring also to the workers.

22 DR. HAMMOND: For both of those.

23 DR. REED: What I mean is that the air  
24 model for general public, the 24-hours area-wide you  
25 have the model that could provide for that kind of

1 consideration by running the model with the most  
2 stable and error, weather conditions and so forth.  
3 But the workers, how they were monitored, the  
4 temperature factor should also be considered as  
5 well.

6 DR. HAMMOND: Right.

7 DR. REED: I don't know if there are data.

8 DR. HAMMOND: But at least one might not be  
9 surprised that air temperature will increase.

10 DR. FROINES: Thank you. Your response is  
11 consistent with --

12 DR. LIM: This is the conclusion of what is  
13 in the document. And that concludes our  
14 presentation. If there are no more questions.

15 DR. FROINES: Thank you very much.

16 We will take a ten minute break. And I can  
17 say that this has been one of the most positive,  
18 pleasant exchanges that I can think of in terms of a  
19 scientific review committee with an agency. So I  
20 compliment you as much as I possibly can.

21 Thank you.

22 DR. MELNICK: Your last sentence says  
23 significant health risks. The value you say is that  
24 we need an exposure reduction 3,000-fold. And after  
25 the discussion here, 3,000-fold is still not enough.

1 Is this feasible?

2 Dr. LIM: I am a risk assessor. How  
3 somebody else takes care of that --

4 DR. FROINES: I think that chairman's  
5 prerogative that asking a question is it feasible is  
6 a question that will come up with the agency later.  
7 And this scientific panel shouldn't get into  
8 feasibility question no matter how interested you  
9 might be in asking the question.

10 Let's take a break.

11 (Break taken.)

12 DR. FROINES: Can we get started, please?  
13 I have talked to the panel members, and we are going  
14 to let the session run until 5:15, so we have a  
15 little bit more leeway. Hopefully, that will give  
16 people an opportunity, but we are going to cut  
17 people off. So everybody be aware that this is  
18 actually -- we are taking it quite seriously in  
19 terms of time.

20 Welcome. I don't have a committee though.  
21 You can start with me, but I am not the most  
22 relevant. I assume that you are presenting and  
23 other people are here for backup?

24 DR. TING: Right.

25 DR. FROINES: Why don't you begin. Ron

1 will be here shortly, I am assuming.

2 DR. TING: Good morning. Thank you for  
3 inviting us to come to this public workshop. My  
4 name is David Ting. I am a senior toxicologist with  
5 the Office of Environmental Health and Human Risk  
6 Assessment for California EPA. The title of my  
7 presentation today is OEHHA's Review of DPR's Draft  
8 Methyl Iodide Risk Assessment.

9 This is where I want to talk about our  
10 department's responsibility and role. OEHHA and DPR  
11 are both part of California EPA. OEHHA is the lead  
12 department for risk assessment. OEHHA has a legal  
13 mandate to provide advice, consultation and  
14 recommendations to DPR regarding human health risk  
15 associated with pesticide exposure. OEHHA reviews  
16 draft risk assessments developed by DPR.

17 This risk assessment was reviewed by a team  
18 of scientists from our department, and most of them  
19 are with me here today. And they can answer  
20 specific questions. OEHHA has submitted written  
21 comments to DPR, and those comments are posted on  
22 DPR website. I am not going to go over all of them  
23 today, and I would only highlight those that we  
24 think are important.

25 I am going to talk about toxicity assessment,

1 specifically on noncancer endpoints and  
2 genotoxicity. I will talk about the exposure  
3 assessment.

4 First of all, noncancer points. In terms of  
5 identification, cancer identification, we notice a  
6 number of adverse health effects. The list is not  
7 comprehensive, but we notice fetal deaths in rabbit,  
8 neurotoxicity in rat, olfactory epithelial  
9 degeneration in rat, change in organ weight and more  
10 weight gain in rat, perturbation of thyroid hormone  
11 regulation in rat, mouse and rabbit, salivary gland  
12 metaplasia in rat.

13 So there is whole range of adverse health  
14 effects. And a number of mode of actions have been  
15 suggested. And they have already been talked about  
16 yesterday and this morning, so I am not going to go  
17 over them again. But it is clear to us that we  
18 don't have all the data to associate different mode  
19 of action with this range of adverse health effects.

20 The critical mode of observed adverse effect  
21 level for noncancer endpoint is the one derived from  
22 a developmental toxicity study, Nemas, 2002. In  
23 this particular study, female rabbits were exposed  
24 to methyl iodide vapor. The 24 animals to a group  
25 and the animals were exposed at 0, 2, 10 or 20 ppm.

1 They were exposed between gestation 6 through 28.

2 They were sacrificed on gestation day 29.

3 DR. BLANC: I think we can speed up this  
4 process. You don't need to read your slides. We  
5 have them on paper, and we have also discussed. You  
6 can just say this slide summarizes what you have  
7 already been told and look forward.

8 DR. TING: I can do that.

9 For this study we noticed many effects, shown  
10 on this slide, and we also used traditional approach  
11 by looking at the NOEL and LOEL, and also do the  
12 benchmark dose modeling actually.

13 For endpoint model we found that the upper 95  
14 confidence level at lower confidence level at 5  
15 percent is around 2 to 3 ppm.

16 DR. BLANC: That is -- just to note, that  
17 finding as acceptable 5 percent fetal mortal issue  
18 is not appropriately safe zone, I would say.

19 Do you have any comment from OEHHA about 5  
20 percent fetal mortality is acceptable.

21 DR. KHAN: We believe that is a bit high,  
22 and we agree with DPR that we can reduce it down to  
23 1 percent. But what we did is we ran the benchmark  
24 dose modeling just to verify that this 2 ppm NOEL  
25 was indeed acceptable if they were to use it.

1 DR. BLANC: We've already decided that it  
2 is not a NOEL; it is a LOEL.

3 DR. KHAN: Well, at the 5 percent extra  
4 risk we're getting a BMCL 05 of 2.7. So that would  
5 be our point of departure, which is loosely  
6 analogous to NOEL. But if DPR were to choose to  
7 take it down to 1 percent extra risk, that would  
8 indeed bring the number further down. We do agree  
9 with what you have to say about this and with DPR's.  
10 If they chose to lower the numbers --

11 DR. BLANC: You would be supportive of  
12 that?

13 DR. KHAN: Yes.

14 DR. TING: I just want to also add that  
15 because of this point we are also recommending  
16 traditional uncertainty factor of ten. You will see  
17 in later slide.

18 Acute exposure is the risk driver for  
19 noncancer endpoint because of the order healthy  
20 effects against lowest known observed adverse level.  
21 And also when you calculate for air concentration,  
22 acute exposure we get to highest concentration.

23 DR. SLOTKIN: I don't actually understand  
24 how studying an acute exposure protects you against  
25 effects from longer exposures or how the NOEL for

1 acute exposure would be lower than that for a  
2 chronic exposure. In fact, for everything I've ever  
3 seen a chronic lower level exposure is as a NOEL.

4 DR. TING: From a lot of chemicals I think  
5 you are right, but for some pesticides I have seen  
6 actually the acute exposure gives you the lowest  
7 adverse effect level. It's based on data that's  
8 available.

9 DR. SLOTKIN: The rabbit study that you are  
10 citing that has the lowest NOEL isn't an acute  
11 exposure.

12 DR. TING: That was an exposure between, I  
13 think, 14 day exposure, but there's also study to  
14 indicate that there is window of vulnerability to  
15 exposures through the hours of [unintelligible].

16 DR. SLOTKIN: That is assuming that the  
17 fetal death is the only relevant endpoint, which I  
18 think we discussed ad nauseam. I think as a  
19 statement of principle is somewhat misleading. I  
20 don't see evidence with methyl iodide that acute  
21 exposure would give you a lower NOEL than a chronic  
22 exposure because we haven't been presented with any  
23 data about lower level chronic exposures.

24 DR. TING: Again, this is based on data  
25 available at this time.

1 DR. SLOTKIN: It would important to note in  
2 a conclusion document that is based on the data, but  
3 there aren't data about chronic exposures and,  
4 therefore, conclusion about whether the acute  
5 exposure protects you from a chronic exposure is  
6 misplaced.

7 DR. TING: I agree with you. There are  
8 data gaps and we tried to make up for that in the  
9 uncertainty factor application.

10 We just have a few points on what we think  
11 about the PBPK modeling. I think this is a very  
12 complex model. It assumes excess serum iodide is  
13 sole mode of action, and we have doubts about that.  
14 It seems that interspecies differences in the  
15 distribution and response to serum iodide that are  
16 not fully accounted for, and are some key parameters  
17 that are based on only a few data points.

18 Because of the reason we already talked about,  
19 we recommend an additional uncertainty factor of  
20 ten. This is on top of the usual default  
21 uncertainty factors because of severity effect  
22 because inadequate testing on animals and because of  
23 the carcinogenicity of this chemical.

24 We think this chemical is clearly genotoxic  
25 because of in vivo DNA damage, adduct formation

1 because of gene mutation and chromosomal damage in  
2 some in vitro studies.

3 Methyl iodide is likely be a genotoxic  
4 carcinogen because of positive genotoxicity data,  
5 because it causes other tumors besides thyroid. So  
6 that the thyroid follicular cell tumors found in  
7 mice and rat may not be solely due to a thyroid  
8 function perturbation.

9 Exposure assessment. Worker exposure  
10 calculation based on standard protection, and we  
11 felt it may not be always achieved in the field, and  
12 we have already talked about that. And we are also  
13 concerned about the protection of the driver and the  
14 assumption that there is a 90 percent protection.

15 DR. FROINES: I want to say one thing about  
16 the carcinogenicity that you went over. I agree  
17 with your conclusions that methyl iodide is likely  
18 to be a genotoxic carcinogen. We talked at great  
19 length about that, but I still think that one of the  
20 things that has not been sufficiently addressed over  
21 the last two days and in the document is that we  
22 haven't talked enough about the chemistry of methyl  
23 iodide. And the chemistry of methyl iodide also  
24 would, I think, strengthen the conclusions that you  
25 developed here, and that the testimony from

1 Dr. Schorer of U.C., Davis, at the hearings where he  
2 discussed the chemistry of methyl iodide, I think,  
3 is very valuable to have in the record. And so,  
4 hopefully, DPR and -- well, I'm getting yes from the  
5 side over here, so I'll leave it at that.

6 DR. TING: This is my last slide. This  
7 concerns that the buffer zone is not wide enough  
8 because workers and bystanders is 152 meters, but  
9 product label half a mile.

10 That concludes my presentation. We'd be happy  
11 to answer any questions.

12 DR. FROINES: Elinor and I are trying to  
13 think about the last slide. We will deal with it  
14 later and not hold you up.

15 So are there other questions?

16 DR. MELNICK: Did you derive a different  
17 risk assessment than DPR?

18 DR. TING: I am not sure what you mean.

19 DR. MELNICK: Risk factor.

20 DR. TING: We did calculate risk factors  
21 for two tumors, and they are in written comments.

22 DR. HATTIS: You recommend applying the  
23 range adjustment factor.

24 UNIDENTIFIED PANEL MEMBER: Yes.

25 DR. MELNICK: Do you come to the same MOE

1 as DPR?

2 DR. TING: We did not calculate the MOEs,  
3 rather we felt it is too detailed. We looked at  
4 factors that go in calculation of MOEs.

5 DR. FROINES: I guess the answer is that we  
6 -- I think what I am hearing from Dale and Ron is  
7 that they would like to have seen that data. Maybe  
8 that is something we can pursue on a different time  
9 frame.

10 Thank you very much.

11 Our next speakers are Elizabeth Mendez from  
12 USEPA and Jeff Dawson: And they will speak for 30  
13 minutes.

14 For the panel, for the panel to recognize when  
15 I say 30 minutes, that means 30 minutes with  
16 questions. We are on a tight time frame.

17 Welcome.

18 DR. DAWSON: Good morning. Thank for the  
19 opportunity to be here to describe how we complete  
20 our risk assessment for methyl iodide. Dr. Mendez  
21 and I are from the EPA Office of Pesticide Programs,  
22 and we are in the health effects division. And  
23 basically, what we have tried to do, based on the  
24 discussion yesterday, is to, for the sake of  
25 brevity, you have a lot of questions, we just took

1 the highlights based on the things that we kind of  
2 discussed yesterday, if that is acceptable.

3 The first thing is a message from our  
4 management, and that is depending -- and I want to  
5 read this into the record.

6 Depending on the outcome of this external peer  
7 review and final risk assessment, EPA may choose to  
8 initiate a reevaluation of the iodide registration.  
9 And if the scientific review panel provides new  
10 information that alters or changes EPA's scientific  
11 analysis, we will include that in this reevaluation  
12 decision.

13 So we are very open to the results and  
14 conclusions of the panel. And this message comes  
15 from highest levels of the agency.

16 We've heard a lot about the potential sources  
17 of exposure and risk. Just to remind everybody, we  
18 are talking about workers in the field and  
19 bystanders getting some various types of exposure  
20 from acute nature, and it can be longer periods of  
21 duration. It stems from methyl iodide being applied  
22 to fields, being volatilized moving off-site to  
23 impact those surrounding the field and also workers  
24 who are involved in the application process.

25 We also look at potential exposure to food and

1 drinking water. One comment on the drinking water  
2 is that our scientists arrived at similar  
3 conclusions. We had concern about the possibility  
4 of drinking water exposure. And in the current  
5 labeling try to implement some risk management  
6 language in there, as directives for users to  
7 prevent drinking water exposure from occurring.

8         There is some additional work being done on  
9 drinking water in Florida that we were interested in  
10 as well.

11         One comment about our risk assessment. All  
12 available documents they are available at this site,  
13 regulations.gov, and that is the docket number.

14         DR. BLANC: Are you saying that you  
15 essentially confirmed the estimated leaching  
16 potential worse case scenario that California --

17         DR. DAWSON: I didn't look specifically at  
18 that number, but we can look at that number, and we  
19 can provide better response that I am able to.

20         DR. BLANC: As far as you know, there is  
21 nothing wrong with that estimate? There is no  
22 critical flaw in that estimate as far you know?

23         DR. DAWSON: I didn't do it. I am not sure  
24 what our position is on the exact -- that particular  
25 estimate. Like I said, I will ask -- go back, and

1 that will be an action that I will look into the  
2 specific value and get back to you.

3           This brings up another topic. As far as our  
4 communication with the panel, clearly we have a half  
5 hour to discuss the issue. So if you come up with  
6 additional questions or comments or thoughts that  
7 you would pose to us, please feel free. We can  
8 develop a mechanism so we can have contact and  
9 communicate about specific issues or questions you  
10 may have.

11           DR. FROINES: I think that is important,  
12 and well look forward to that interchange. We want  
13 as much interaction over the next period of time as  
14 you possibly can to deal with the issues that have  
15 come up.

16           DR. DAWSON: Our only thought is that is a  
17 process that involves the three of us: the panel,  
18 EPA and the DPR folks as well. So just a little bit  
19 of a review. We evaluate a risk under many  
20 conditions. We actually then look at issues around  
21 life stages, for example. We look at varying  
22 durations of exposure. We look at exposure that  
23 could occur from common application that would be  
24 expected with methyl iodide. We look at  
25 emissions data under typical films that are used in

1 agriculture that Dr. Barry discussed yesterday, and  
2 recently we've also had data that has become  
3 available where we are looking at what I would call  
4 higher tech films that reduce emissions, that are  
5 commonly known in the trade as virtually impermeable  
6 films, so we have some research from the use of  
7 those kind of films, and they dramatically reduce  
8 emissions.

9           Then we also looked at doing distributional  
10 analyses using five years of weather data from a  
11 variety of weather stations, sources around the  
12 country to elicit a broad range of potential risk  
13 estimates under various use conditions.

14           A couple comments on the methods -- sorry, I'm  
15 like Roger. I think I'm working on a cold. A  
16 couple of comments on the methodologies that we use.  
17 We use the reference concentration methodology. And  
18 the first bullet just provides all the information  
19 around that document and the policy and the peer  
20 review process that went into policy. Also, the  
21 distributional air model that we used. We had a  
22 peer review under the different scientific advisory  
23 panels in 2004, and this is a link to all the  
24 materials for that meeting. That includes the  
25 issues -- the papers that were developed by the

1 registrant, Arysta, our paper and conclusions of the  
2 panel.

3           Throughout the process, we've had significant  
4 collaboration with scientists from also research and  
5 development, the scientists here at Department of  
6 Pesticide Regulation and various elements of USEPA.

7           A little bit on the exposure assessment.  
8 There was a lot of discussion around worker  
9 exposure. This is the only slide that I have on  
10 workers exposures. I think the data and all those  
11 issues were addressed in sufficient detail  
12 yesterday. Basically, we use essentially the same  
13 data that was described yesterday. We also have one  
14 aspect in our program where we are under  
15 requirements for ethical review of all the human  
16 studies that we use; and these data met our ethical  
17 requirement.

18           We looked at worker risk for all potential  
19 endpoints that we've talked about. I am sure you  
20 have a lot of questions about the endpoints that we  
21 use. The one thing I want to point out from  
22 yesterday, is that what we are finding is that we're  
23 a little bit different on some of the exposure  
24 values. For example, the hole punching and the  
25 planting, what we saw in the result is that, as

1 Dr. Barry described, the emission profile, most of  
2 the emissions are occurring in the first few days.  
3 I went back and looked at the data after discussion  
4 yesterday. On average about, just in first few  
5 days, about 50 percent of material is emitted. And  
6 then it tails off. So you have more emissions over  
7 time. And then for hole punching it is usually done  
8 between five and seven days afterwards. And what we  
9 saw in the data, and I will have some follow-up with  
10 the DPR scientists, but the exposures bear that out.  
11 They are lower for hole punching, and also for  
12 planting aspect.

13 DR. BLANC: It wasn't actually clear to --  
14 DPR was unable to confirm that in test sampling that  
15 was done, although it was clear that it was the  
16 initial application as per routine, whether hole  
17 punching had actually occurred.

18 DR. DAWSON: It did. The hole --

19 DR. BLANC: You verified that?

20 DR. DAWSON: Whenever hole punching was  
21 done, it was always in conjunction with the emission  
22 studies. They did --

23 DR. BLANC: It was incorporated?

24 DR. DAWSON: Yes. Not every one, but  
25 whenever it was done, it was always in conjunction

1 with emission. Sets of emission data that went with  
2 it.

3 DR. BLANC: Let me clarify. You are saying  
4 some of the emissions do not include --

5 DR. DAWSON: Some of the emissions they  
6 didn't monitor hole punching.

7 DR. BLANC: Are those averages in with the  
8 flux estimates?

9 DR. DAWSON: The way we dealt with the flux  
10 estimates is we use each study individually and  
11 presented the risk of such data for each of the  
12 emissions for each study. So the risk managers have  
13 a very broad range based on the -- so we used each  
14 flux study individually and did a series of modeling  
15 calculations based on each individual flux study.  
16 We did average the flux studies together.

17 DR. BLANC: I understood the presentation  
18 that ultimately in the modeling one flux number is  
19 used, depending on what the application scenarios  
20 was.

21 DR. DAWSON: One each application there  
22 were multiple flux studies, like two or three, I  
23 think for the --

24 DR. BLANC: Wasn't only one flux number  
25 applied?

1 DR. DAWSON: No. We presented ranges of  
2 risk estimates for each individual flux study.

3 DR. BLANC: How did the range estimate  
4 differ for the flux studies that you didn't include  
5 the actual, realistic use scenarios?

6 DR. DAWSON: It is indistinguishable.

7 DR. BLANC: What you are saying is that in  
8 those flux studies in which the hole punching and  
9 tarp manipulation were carried, you couldn't detect  
10 a signal?

11 DR. DAWSON: You can't detect a difference  
12 in the overall flux profile because of that  
13 activity.

14 DR. HATTIS: Your agency has been a leader  
15 in application of probabilistic methods to exposure  
16 assessment, both in dietary and to some extent in  
17 the worker. In the dietary case you provide risk  
18 managers with information out to the variability,  
19 out to 99th percentile. What kind of percentiles do  
20 you try to shoot for in these occupational  
21 assessments?

22 DR. DAWSON: The risk manager is provided  
23 with essentially the entire set of distributions for  
24 their considerations.

25 DR. HATTIS: Good answer.

1 DR. DAWSON: I guess the other part of this  
2 is we did identify there were risks associated with  
3 various activities, and we require respirators on  
4 our labeling for various types of workers.

5 DR. BLANC: Not for post-application  
6 workers. We have heard or was that incorrect  
7 information? We were specifically told that after  
8 the application stage, people who did hole punching,  
9 tarp manipulation are not.

10 DR. DAWSON: The risk that we calculate for  
11 hole punchers and the planting, we did require  
12 respirators for. I believe there is little bit of  
13 clarification that needed to be worked on with DPR  
14 about the use of particular monitoring data for  
15 those scenarios. That was our information.

16 The other thing I might mention, based on  
17 discussion yesterday, is for respirators. We always  
18 required OSHA protection program about medical  
19 monitoring and training and cartridge change-outs  
20 every day, every eight hours or if you suspect you  
21 have break through for the user.

22 And methyl iodide is also used in conjunction  
23 with chloropicrin as a warning agent. So that aids  
24 us in kind of lists there in letting people know  
25 when they're experiencing --

1 DR. HAMMOND: Is that a requirement that  
2 all the people there know that? I know the current  
3 process does? Is that a requirement?

4 DR. DAWSON: By the fact -- by default  
5 because they're all mixed chloropicrin, yes. If we  
6 had, for example, consider the use of a product with  
7 a warning agent, we would look at this in a  
8 different perspective. So we would. And some  
9 additional protection.

10 DR. HAMMOND: Has that always been there,  
11 in there?

12 DR. DAWSON: Absolutely. Just a little bit  
13 on what we are doing with our bystander assessment  
14 calculations. We, like I said, we are using a  
15 distributional model, and we are also including the  
16 emissions data that we have with the barrier films  
17 that show the overall reductions in emissions. We  
18 use the monitoring data and whatever is available on  
19 incidence data. So we will do a little checking on  
20 that before this meeting.

21 Just to show the number of iterations of the  
22 modeling that we did. Here is the emissions data  
23 that we talked about yesterday, the eight studies  
24 that Dr. Barry described. We also used three  
25 additional ones with the barrier films that are not

1 on this slide. We looked at weather data for the  
2 anticipated high use area for methyl bromide; two  
3 stations in California, an inland and coastal  
4 station, an inland and coastal station in Florida,  
5 and then a station in Flint, Michigan. We did the  
6 iteration as for variety of difference, application  
7 rate, and so on.

8 The model was iterated around 4,000 times. It  
9 has a series of outputs. So the actual number of  
10 outputs are much greater. All that information was  
11 considered as part of the risk management decision  
12 making process.

13 This is an example of what the weather data  
14 looked like. Here in California we use the site in  
15 Bakersfield in inland and we used a Ventura County  
16 site. And in actuality we also use a site in  
17 Bradenton, Florida, which consistently predicted the  
18 furthest buffer distances, and that played a large  
19 role in our decision making process for this  
20 chemical. And this is just a graphical example of  
21 what the five years of weather data from one station  
22 looks like. This is just showing hot wind vectors  
23 over a five-year period and shows the prevailing  
24 wind directions and the magnitude of those  
25 directions.

1 DR. HAMMOND: In inland California those  
2 prevailing directions changes by season, different  
3 seasons. You wouldn't want to use a yearly average  
4 for those seasons?

5 DR. DAWSON: We look at the issue of  
6 seasonality as well. The modeling output presented  
7 on monthly basis, and you can look at the  
8 differences.

9 There are just different ways of compiling the  
10 information that comes from this. So they're  
11 basically three different approaches that we use for  
12 compiling the information. This represents four  
13 days out of five years of weather data in each set  
14 of calculations. So this is the field and this is  
15 the set of receptor points included in the model  
16 around the field. So for each of the five years'  
17 worth of days, it's calculating a profile, a  
18 concentration profile, if you need some part of  
19 concentration of concern. Then one way of compiling  
20 this is by over five years creating a distribution  
21 of all points around these isoputs. That is called  
22 the home field analysis.

23 And then there are others ways where you just  
24 take the furthest data points for each of the days;  
25 that is called the maximum buffer approach. And

1 then within the set of receptors around this ring,  
2 it actually provides air concentrations and a  
3 distribution of specific ring distances around the  
4 field. You can also use those directly and  
5 calculate more of the exposure. So the risks are  
6 presented in three different ways. The whole  
7 distribution of exposure outputs are included in  
8 that.

9 DR. MCKONE: Has this ever been done for  
10 situations where you go to the next adjacent farm  
11 and assume that there are multiple fields treated or  
12 the pattern of use that --

13 DR. DAWSON: That is one of the issues that  
14 we're looking. That is one of slides coming up. We  
15 haven't calculated that yet. But one of the ways we  
16 dealt with that is by -- whenever we establish  
17 buffers, so they are not overlapping. And so that  
18 is one way of dealing with it. This is an area of  
19 concern for us, and we are definitely looking at the  
20 issue of multiple sources within the area, also  
21 better ways to model and predict ambient  
22 concentrations from multiple sources within  
23 communities, because I know that was one of the  
24 discussions yesterday.

25 DR. HATTIS: I imagine many farmers want to

1 apply the material at similar times.

2 DR. DAWSON: We have had a lot of  
3 discussion with the growers about the implications  
4 of that, as far as the ambient conditions of the air  
5 and their application processes. Certainly is a big  
6 issue.

7 One other thing I want to bring up after  
8 discussions yesterday, there was a lot of discussion  
9 around mass balance issues and where -- what happens  
10 to the remainder of the emitted materials. So we  
11 have engaged - and this document was published  
12 about a year ago - a document that looks at the  
13 factors that are occurring subsurface, in the  
14 subsurface soil, and related to the field conditions  
15 that can impact emissions. And these key factors  
16 that we're finding are on types of tarps.

17 So in our empirical monitoring data you can  
18 see the area films we have included as far as the  
19 amount of emissions, the kind of field conditions  
20 present, how growers prepare the field, the amount  
21 of moisture in those fields, level organic matter,  
22 the soil types and so on. Then the use of soil  
23 amendments, for example, irrigation after  
24 application to keep emissions down or the use of  
25 barriers agents like sodium thiosulfate. Because

1 one the points we're using where there is actually a  
2 process that appears to degrade certain fumigants at  
3 the barrier level, between the soil and the  
4 atmosphere. Also, the impact of various application  
5 methods. And we're engaged in revising this  
6 document, and we're looking at adding additional  
7 data. We received some comments, and we're looking  
8 at various modeling options, and so on.

9 DR. FROINES: I had a simple question.  
10 It's my understanding, and I could be wrong, but  
11 methyl iodide is being used in 47 states around the  
12 United States. And so I would hope that you're  
13 checking data from those experiences that relate to  
14 these particular issues.

15 DR. DAWSON: We are, and we are also  
16 looking at this in a broader range, across all the  
17 fumigant chemicals. We want to understand these  
18 kinds of processes for all these different  
19 chemicals.

20 DR. FROINES: And Elinor thought that you  
21 mentioned, made a comment, that you were also  
22 looking at, quote, incidences to date. Does that  
23 mean that there have been incidences to date?

24 DR. DAWSON: I actually had a slide on  
25 that. I went back and called some of the folks in

1 Florida where it's been predominantly used. There  
2 has been no incidences to date that we are aware of.  
3 I had our epidemiologist in our groups to go back  
4 and look at the data sources that we are having, and  
5 we have not found any evidence of any kind of  
6 incidence since this was put to the market.

7 DR. MELNICK: Does the data that you have  
8 been collecting, is it consistent with your model  
9 predictions?

10 DR. DAWSON: The data is actually used to  
11 -- it's an empirical modeling approach, so the  
12 emission profiles are used as a basis for modeling  
13 approach. But, yes, we find, basically, the same  
14 conclusions that Dr. Barry talked about yesterday  
15 were you would be able to predict for those -- you  
16 would be able to predict.

17 DR. MELNICK: With multiple weather  
18 conditions?

19 DR. DAWSON: You could go back and  
20 predict.

21 DR. MELNICK: I know predict. Does the  
22 data support the predictions?

23 DR. DAWSON: Yes.

24 DR. MELNICK: With the variable weather  
25 conditions?

1 DR. DAWSON: Yes. Again, I would  
2 recommend, if you have questions on that, to go and  
3 look at the SAP document in 2004. There is a lot of  
4 discussion with that issue.

5 DR. FROINES: That doesn't sound quite  
6 current. 2004 to 2009 is a five-year difference.  
7 That seems like a problem.

8 DR. DAWSON: We can provide more  
9 information about this, this specific approach as to  
10 how he calculated the flux and so on. But I think  
11 that would answer your question.

12 DR. MELNICK: Has there been any detection  
13 in groundwater?

14 DR. DAWSON: Not that I am aware of. But,  
15 again, I will look into the groundwater issue,  
16 because I personally didn't have hands on doing  
17 that, and I want to make sure I give you the right  
18 answer. So I we will follow up and give you the  
19 right answer.

20 DR. FROINES: You have eight minutes left  
21 before this closes, so we are taking their time. So  
22 please make sure that your questions are relevant.

23 DR. HAMMOND: Was temperatures one of  
24 parameters you considered in this model?

25 DR. DAWSON: Absolutely.

1 DR. HAMMOND: Is there an attempt that it  
2 is not very important?

3 DR. DAWSON: It is very important. But it  
4 is not one of ultimate drivers, but it is important.

5 Just a little bit. For longer time exposures  
6 we compared -- we looked at the ambient air fate for  
7 methyl bromide, similar to what California did. We  
8 did not do a modeling approach, but we are very  
9 interested in looking at the modeling approach that  
10 they've done. We're actually doing a scientific  
11 advisory panel meeting for non-fumigant pesticides  
12 that could be volatile in December, and we are  
13 looking at a variety of methods to do that. That  
14 could be something that we are going to make  
15 improvements in the risk assessment over time. And  
16 I'm aware that there is monitoring data for  
17 community-base exposures that are going to be going  
18 on in Florida, and we are going to be looking at  
19 that data very closely.

20 DR. FROINES: Will that be methyl iodide?

21 DR. DAWSON: Yes. And the issue of  
22 incidence. To the best of our knowledge, to date  
23 around 17,000 acres have been treated with methyl  
24 iodide with a limitation of 40 acres per  
25 application. That means that there's been at least

1 been 425 individual application events with no  
2 reported incidence.

3 I will turn it over to Liz. Thank you for  
4 your time.

5 DR. MELNICK: What do you mean by no  
6 incidence?

7 DR. DAWSON: We have no report of any  
8 problems stemming. They could range from tearing  
9 from the chloropicrin component or to more serious  
10 effects. It depends how they are reported. It  
11 means no reports even even of any kind of slight --

12 DR. MELNICK: No incidence doesn't mean  
13 there wasn't something happening.

14 DR. HAMMOND: Is it an active or passive  
15 monitoring, where you're actively seeking or just  
16 waiting to hear?

17 DR. DAWSON: Reported to us.

18 DR. HAMMOND: No active effort to go out  
19 and the find it?

20 DR. DAWSON: The individual growers. Again  
21 --

22 DR. HAMMOND: You are waiting for a report?

23 DR. DAWSON: We will provide more  
24 information on the details on how this works.

25 UNIDENTIFIED AUDIENCE MEMBER: That is

1 basically no report of incidence.

2 DR. FROINES: Where did that come from?

3 This is not that public meeting. Please.

4 DR. DAWSON: Liz.

5 DR. MENDEZ: I think I have about six  
6 minutes left, so I am going to try and go through  
7 these very quickly. In light of the discussions  
8 that we had for the past day and a half, we will be  
9 taking all of the considerations that the panel and  
10 the recommendations that the panel is making through  
11 the process. So we just wanted to give you a 20,000  
12 foot view of where we are right now, and taken from  
13 air.

14 Before I say anything more, I want to say that  
15 this is the result of the evaluations conducted by  
16 dozens of EPA scientists in the office of pesticide  
17 programs and National Center for Computational  
18 Toxicology and other areas in ORD.

19 We have an overview, we have an extensive  
20 toxicology database with four studies required by 40  
21 CFR part 158 inhalation route. This compound has  
22 been classified as a non-use pesticide and as such  
23 it did not typically require to have this entire  
24 data set. So it's different from what we normally  
25 would expect from a nonfood use.

1           We have in addition to the four studies,  
2 mechanistic studies, we have PBPK modeling and  
3 observational human study. And just a brief word  
4 about the observational human study. That was no  
5 deliberate exposure to anyone of methyl iodide. The  
6 intent of that study was to determine what is the  
7 distribution of iodide between the fetal compartment  
8 and maternal in human, because in the rabbit it  
9 appears that the fetal compartment accumulates  
10 fourfold higher than the mother.

11           A hazard assessment was conducted for all  
12 durations of exposure. There was emphasis on acute  
13 bystander assessment.

14           We used CRC methodology developed the Office  
15 of Research and Development at EPA to derive human  
16 equivalent concentrations for all but acute  
17 scenarios. Those metrics and parameters were  
18 derived using a PBPK Model were used for acute  
19 scenario. We did not require a developmental  
20 neurotoxicity study, and we are not -- it is not  
21 likely -- it's been classified by the Cancer  
22 Assessments Review Committee as not likely to be  
23 carcinogenic in humans at doses that do not alter  
24 thyroid hormone homeostasis.

25           DR. SLOTKIN: We have a compound that is

1 documented as being neurotoxic, which case study is  
2 of human exposure show the most persistent and most  
3 sensitive effect is neurotoxicity and it's  
4 developmentally toxic. Explain to me how you came  
5 to the conclusion that DNT was not required.

6 DR. MENDEZ: At this point in time, our  
7 deliberation was that because this is a thyroid  
8 toxicant we wanted to chase the thyroid hormone  
9 perturbations because it is a critical initial step  
10 for thyroid toxic compound and neurodevelopmental  
11 problems.

12 DR. SLOTKIN: The fetal toxicity data show  
13 very clearly that it is not the thyroid disruption  
14 that is causing fetal toxicity. And I think there  
15 is a vast literature about organo halogens and  
16 developmental neurotoxicity above and beyond that of  
17 thyroid effect. So to me it doesn't make sense that  
18 if you have a neurotoxic compound and it is  
19 developmentally toxic that you wouldn't ask for a  
20 DNT study.

21 DR. MENDEZ: Another consideration that the  
22 EPA scientists took into account is the fact that  
23 the DNT is a fairly blunt instrument with empirical  
24 measure, and we felt for -- we all felt that perhaps  
25 going for the hormone levels would be a bit more

1 relevant.

2 DR. SLOTKIN: That makes the presumption  
3 about mechanism that is, in fact, backed by the  
4 available data. And I would submit to you blunt  
5 instrument is better than selecting no instrument at  
6 all.

7 DR. LOECHLER: Regarding your final  
8 statement there. I have your 2005 report. And on  
9 Page 24 you say:

10 Though there are abundant data  
11 suggesting that iodomethane induces  
12 thyroid follicular cell tumors through  
13 an anti-thyroid MOA, the fact that  
14 iodomethane has been shown to have  
15 mutagenic properties precludes the  
16 exclusion at this time of mutagenicity  
17 as a contributing factor in thyroid  
18 tumor genesis. (Reading)

19 Do you still stand by that statement on Page  
20 24?

21 DR. MENDEZ: What we have determined is  
22 that while we cannot rule out on the mutagenicity of  
23 it, we felt that the operative mode of action is  
24 thyroid perturbations.

25 DR. LOECHLER: I have one other question

1 from this document as I have the microphone here.

2 On Page 3 --

3 DR. MENDEZ: Before you continue, I just  
4 want to let you know there is a newer document from  
5 2007. That you may --

6 DR. LOECHLER: Okay.

7 DR. MENDEZ: I just wanted for the record  
8 for you to know that.

9 DR. LOECHLER: I will get it.

10 You can tell me if this sentence is still in  
11 that document, perhaps. It says: The majority of  
12 neoplastic lesions - I won't read the whole sentence  
13 - were observed as the terminal sacrifice unlike  
14 tumors induced through a mutagenic MOA.

15 Can you provide to me some references or  
16 documentation that support that particular view? I  
17 would like to have those documents.

18 DR. MENDEZ: I have to get back to you, but  
19 I believe, I am not a hundred percent sure, but I  
20 believe that we softened that language in the 2007  
21 document.

22 DR. LOECHLER: That seems prudent. Why  
23 don't you send me the documentation. I would love  
24 to see it.

25 DR. MENDEZ: I will.

1 DR. MELNICK: The study of sodium iodide,  
2 which is obviously not an alkylating agent, induces  
3 the same thyroid perturbations, but did not induce  
4 thyroid tumors.

5 Does that make you question your fourfold?

6 DR. MENDEZ: It was -- the sodium iodide  
7 study was a short-term study.

8 DR. FROINES: We're going to have to move  
9 on.

10 Thank you.

11 DR. MENDEZ: I can go to the very end. I  
12 can go to the very last one.

13 DR. FROINES: Please, please. I apologize  
14 for acting like this.

15 DR. MENDEZ: We understand it.

16 I just want to summarize where we are right  
17 now with the risk assessment. We thought that we  
18 had robust and extensive evaluated toxic database.  
19 We calculated acute HECs for three endpoints of  
20 concern: The nasal lesions HEC report with 4.5; the  
21 fetal loss HEC, 7.4 and 23; and the neurotoxicity  
22 HEC with 10 ppm.

23 Risk calculations were done with all three of  
24 these HECs and provide to risk management. And as  
25 we just discussed, we have classified this as not

1 likely to be carcinogenic in humans at doses that do  
2 not alter thyroid homeostasis.

3           In light of the brevity of this presentation,  
4 I encourage the panel to please avail themselves to  
5 call or E-mail us and we'd be more than happy to  
6 continue these discussions.

7           DR. BLANC: One of the problems with your  
8 presentation is that it reiterates your comment to  
9 DPR and presumes that we weren't privy to those  
10 comments. It would be far more helpful to hear what  
11 your response was to what you heard the last couple  
12 of days. And in future communication I think that  
13 would be far more useful. I would have to say that  
14 the consensus of our discussions in the last day and  
15 half completely go against every single one of the  
16 bullets in your summary slides. Some of them -- I  
17 think the level of strengthening opinion is more  
18 nuance than others.

19           But clearly I don't believe there is a person  
20 sitting at this table who believes that your  
21 toxicity database was, in fact, robust. And we  
22 identified significant things that are lacking,  
23 most saliently the comments made a few minutes ago  
24 about developmental neurotoxicity, but also chronic  
25 neurotoxicity data, including that we completely

1 disagreed with the basis and method for achieving  
2 every single one of your acute endpoints, and that  
3 there was absolute disagreement, as you heard just a  
4 couple minutes ago, with the last bullet.

5 Now, I understand this is just recapitulating  
6 what you previously stated. What is important to us  
7 is for you to reflect back on what you learned and  
8 heard in the last day on these things.

9 DR. MELNICK: Because of your response to  
10 my question, the chemical I meant was a potassium  
11 iodide instead of sodium iodide. That was a  
12 two-year study which did not produce thyroid tumors.  
13 It is a non-alkylating agent.

14 Does that question your fourth bullet in terms  
15 of mode of action?

16 DR. MENDEZ: You know, I have to go back  
17 and look at that study. I haven't looked at it in a  
18 while.

19 DR. MELNICK: It was published in 2000.

20 DR. FROINES: During the next couple of  
21 months, while we're working, we would -- what I want  
22 to do is promise you that we'll follow up with you  
23 to basically address the issue that Paul raised. So  
24 we can, basically, all be on the same page in terms  
25 of our general understanding of the issues.

1 Thank you very much.

2 DR. DAWSON: Thank you for having us.

3 DR. FROINES: Your next panel will be from  
4 Arysta. What I would like you to do, first  
5 introduce yourself and your fellow speakers. And we  
6 can get started. This will be a 45 minute  
7 presentation.

8 MS. RHODES: Yes, sir. I am very happy to  
9 be here today and appreciate you all wanting to  
10 listen to our position. My name is Becky Rhodes. I  
11 am head of regulatory affairs for Arysta Lifescience  
12 for North America which we are headquartered in  
13 Cary, North Carolina, a neighbor of yours.

14 DR. SLOTKIN: As long as you're not a Tar  
15 Heel fan.

16 MS. RHODES: I'm an N.C. State fan.

17 DR. SLOTKIN: That's even worse.

18 MS. RHODES: Before I get into my  
19 presentation, I would like to introduce our esteem  
20 panel. We chose to bring all of your experts today.  
21 I know our time is limited, but, please, they can  
22 address any of the questions that I have heard you  
23 all put forward during the course of this last day  
24 and half. I just do want to say to you that we tell  
25 people that Midas is the most comprehensively

1 studied chemical in agriculture. More than 100  
2 different scientists have worked on or studied  
3 iodomethane and its effects, and we have included  
4 that list of scientists in your packet. Hopefully,  
5 you have received from the Department of Pesticide  
6 Regulation this morning a booklet that looks like  
7 this. We have included some documents here that we  
8 heard you fellows request and lady requesting. And  
9 hope this will be of value to you.

10 If you give me just a second. Can you hear me  
11 in the back of the room?

12 On my left is Dr. Beth Mileson. Dr. Mileson  
13 is a toxicologist and co-editor of a special issue  
14 of *Inhalation Toxicity* which has a special section  
15 on methane. We've also included a copy of this in  
16 your packet. The yellow Post-it notes where the  
17 sections starts. You all mentioned an interest in  
18 having peer review journal articles, and most of the  
19 data that you have been discussing has been peer  
20 reviewed and published in this journal. I'll offer  
21 this unless it is against the law, but if guys don't  
22 want to take this back, if you'll put it back  
23 together and give us a business card we'll be happy  
24 to Federal Express it to your office. I carry a  
25 large load of paper myself, but if that is against

1 the law, I don't want to do that.

2 Next to Beth is Dr. Richard Reiss. He  
3 actually did develop the ProFume model. He is  
4 principal scientist and exponent in engineering and  
5 consulting.

6 Next is in the back here is Lisa Sweeney,  
7 Dr. Lisa Sweeney. She developed a pharmacokinetic  
8 model that you all have been discussing. She is the  
9 senior scientist with toxicology excellence for risk  
10 assessment.

11 We have at the table Andy Newcombe. Andy is  
12 charter chemist and principal scientist with LFR,  
13 environmental fate expert.

14 And then we have Shan Brooks at the end. Shan  
15 is our technical sales manager in Florida, and has  
16 hands-on experience with the total of approximately  
17 17,000 applications. He manages the stewardship  
18 program there, qualifies the applicators and can  
19 answer any of your questions about hands-on that  
20 came up earlier in the presentations.

21 And last but not least, you have me. As I  
22 said, I am Becky Rhodes. One thing I would like to  
23 make sure the panel understands, and I know we have  
24 a lot of interesting parties in here, about working  
25 on a farm. I was raised on a peanut farm in rural

1 North Carolina. I have my Master's degree from  
2 North Carolina State University in crop science. My  
3 husband and I still operate a Christmas tree farm.  
4 My entire life has been devoted to agriculture, as  
5 has been a number of people in our company.

6         When I was growing up, we were a true family  
7 farm. When I was eight years old, I had to carry my  
8 own row chopping peanuts. I'll just say one thing  
9 last about that. It was a proud day for me when I  
10 came home from graduate school and said to my  
11 father, "Hey, I learned about this new herbicide  
12 that is registered." And I never really thought  
13 he'd take me seriously, but here today he was using  
14 it. We didn't have to chop peanuts anymore. Just a  
15 point of emphasize that there is a place for  
16 pesticides, and we want to make sure your  
17 discussions are balanced.

18         Iodomethane was developed as a methyl bromide  
19 alternative. By I believe it is Jim Sims - I just  
20 met him a few minutes ago. Never met the gentleman.  
21 So if you guys want to ask him any questions, I'm  
22 sure he would be glad to talk to you - at the  
23 University of California, Riverside. And this was  
24 in 1991. University of California system granted a  
25 license to Arysta in order to commercialize the

1 product. And you may know that methyl bromide has  
2 been in wide use for decades and is currently being  
3 phased out under the Montreal Protocol because it is  
4 an ozone depleter. Iodomethane is not. ArystaTrace  
5 was proud to be awarded the prestigious EPA Ozone  
6 Layer Protection Award earlier this year, which was  
7 given by the Office of Air Radiation for the  
8 Stratosphere Protection Division of EPA.

9 I would like to briefly touch on what we call  
10 the EPA review. And, of course, we just heard from  
11 the folks at EPA. I want to make sure everybody is  
12 aware that the initial data package for this  
13 compound was submitted to both EPA and DPR  
14 simultaneously in 2002, which is seven years ago.

15 Iodomethane was registered by EPA in October  
16 2007. Our label is the toughest label in the  
17 fumigants industry. We have the most comprehensive  
18 stewardship and in-field training program of all  
19 fumigants on the market. We work extensively with,  
20 as I mentioned, over a hundred scientists, with  
21 representatives from U.C. Davis, U.C. Riverside,  
22 University of Florida, University of New Mexico and  
23 USDA. We do all of our data development through  
24 contract with laboratories such as PTRL West here in  
25 California, rural research labs, just to mention a

1 few. I mention that because I hope that would give  
2 you an extra edge of comfort. This is not inhouse  
3 Arysta data. I think you are aware that, as  
4 yourselves, all scientists want to maintain their  
5 integrity. So it is not just Arysta; it is a  
6 hundred people who looked at things and helped us  
7 make good decisions.

8           It is worth noting that EPA said when  
9 registering Midas that it had been one of the most  
10 thorough analyses ever completed on a new pesticide.  
11 I believe that is still on their website.

12           You all mentioned earlier that this compound  
13 is registered in 47 other states. That is  
14 absolutely true. We are very proud of that. We are  
15 hoping to be able to add these other three,  
16 particularly the state of California. We are still  
17 under review in Washington. We actually took our  
18 package out of New York because they wanted some  
19 commercial information that we just can't feel was a  
20 priority because it was a very small market for us.  
21 We will resubmit in New York just as soon as the  
22 opportunity presents itself. And, of course, we are  
23 here today to facilitate the process of obtaining a  
24 registration in California.

25           As has already been mentioned, we have

1 17,000-plus acres already in use in Florida, Maine,  
2 Michigan, Virginia, North and South Carolina and  
3 Georgia. Without incidence. I am glad that point  
4 has already been made.

5 DR. BLANC: Just to correct the record. It  
6 was without reported incidence. That is a critical  
7 difference and update. I think that is rather  
8 important to be clear about.

9 MS. RHODES: I think I am making my own  
10 noise here. Excuse me just a second. I seem to be  
11 having your problem.

12 Thanks for making that point, Dr. Blanc.

13 This real world experience has proved that  
14 iodomethane can be and is used safely in a field.  
15 Because iodomethane is chemically similar to methyl  
16 bromide, is every bit as effective, the growers use  
17 30 to 50 percent less pounds per acre than they did  
18 methyl bromide. Also, growers who use an effective  
19 fumigant can reduce the amount of compounds used  
20 throughout the season. That means fewer pesticides  
21 in total in the field throughout the entire growing  
22 season. We like to call that the right foundation.

23 You've seen various pictures of applications,  
24 so I felt compelled to show one of ours. But I have  
25 to say this may be the most important thing that we

1 might like to talk about.

2 DR. LOECHLER: It sounds like you have some  
3 data with respect to how the use of methyl iodide  
4 and some other fumigants decreases the use of other  
5 pesticides subsequently. Is that something you  
6 could provide to us?

7 MS. RHODES: Let me check into that and see  
8 exactly. It's basically commonly known in  
9 agricultural circles what a fumigant does; it  
10 removes the weed seed, the diseases that are there  
11 and insects. It gives the crop a head start so we  
12 don't have to put applications on immediately after  
13 planting.

14 Iodomethane is applied by professional,  
15 certified applicators licensed by the state. And in  
16 this state I understand it is absolute 100 percent  
17 all professional licensed. In addition, the state  
18 certification applicators must be qualified by  
19 Arysta. This is a label requirement and it is a  
20 company requirement. This includes classroom  
21 training specific to our product and our safety  
22 requirements. This training has to be repeated  
23 annually in order for the applicator to be able to  
24 purchase the product.

25 Once Iodomethane is sold, we work with the

1 applicators to ensure that their equipment is  
2 properly calibrated, applicators understand the  
3 label requirements and that applicators, farm  
4 workers, and bystanders will all be protected within  
5 the label.

6           Just go over briefly. I believe you all  
7 talked and asked some questions to make sure. The  
8 application is made to soil when there is no crop  
9 present. Injections - you hear a lot bout shank  
10 injections. Shanks would be placed on this tool bar  
11 about 12 inches apart with injections made six to  
12 12 inches beneath the soil surface. The tarp or  
13 this plastic is immediately applied in one  
14 integrated process immediately after the  
15 application. These two processes work in tandem.  
16 Work to minimize the amount of material that leaves  
17 the soil surface. Not only is that protective of  
18 humans, but that is better for everybody because we  
19 want it in the zone where it is going to do its  
20 work.

21           Next thing I want to mention is that the field  
22 is then immediately covered by a tarp and monitored  
23 for 48 hours, which is the restricted entry interval  
24 period. Buffer zones are clearly delineated around  
25 the outside of the field based on the label and have

1 to be monitored. It is the certified applicator's  
2 responsibility to stay on site within the line of  
3 sight of the application at all times and to see  
4 that the buffer zone integrity is maintained.

5 Our commitment as Arysta to safety is to farm  
6 works, neighbors and bystanders because, after all,  
7 we are all neighbors, aren't we. Arysta's position  
8 is that we have a shared goal of safe and effective  
9 use of iodomethane. We believe we have that shared  
10 goal with EPA, with DPR and with this panel.

11 Our existing label, as approved by EPA,  
12 affords industry leading protection to workers,  
13 bystanders, communities; and it is our position that  
14 DPR's Draft Risk Characterization Document is  
15 unnecessarily conservative. Our work is based on  
16 solid science, and with your permission we would  
17 like to cover the important areas and explain the  
18 basis of our science.

19 We have six key issues where we believe DPR's  
20 document is overly conservative. You talked about  
21 -- I will try to make it brief. DPR selected the  
22 NOEL 2 ppm in rabbits for a developmental toxicity  
23 endpoint instead of 10 ppm NOEL as a point of  
24 departure. DPR did not accept the weight of  
25 evidence for the developmental toxicity mode of

1 action; thereby selecting an inappropriate dose  
2 metric or human equivalent concentrations.

3 DPR recommends an uncertainty factor of 300,  
4 which is ten times more than EPA, and we believe is  
5 unnecessary. DPR indicated a developmental  
6 neurotoxicity study with a data gap. EPA determined  
7 available data are adequate, and we concur. DPR  
8 used chronic oral iodide intake to assess potential  
9 risk from acute iodomethane exposure. We believe  
10 this is inappropriate. DPR used simplistic  
11 screening level methods for exposure assessment  
12 instead of state-of-the-art probabilistic exposure  
13 assessment methods that were just discussed.

14 This is basically our version of the summary  
15 of the same data you've seen in different forms, but  
16 this is how -- the clearest to me to understand. We  
17 have three acute toxicity endpoints of concern:  
18 nasal lesions, transient neurotoxicity and  
19 developmental toxicity. At every point that EPA had  
20 for occupational and bystander is quite a bit higher  
21 than the one chosen or derived by DPR.

22 What I would like to point here is the EPA HEC  
23 for developmental toxicity is 23 ppm and .22 ppm for  
24 DPR. This is a hundredfold different, which is an  
25 extra hundredfold safety factor.

1           Like to briefly touch on nasal lesions.

2           DR. SLOTKIN: Could you back up to neurotox  
3 issues? What is the rationale behind just doing  
4 transient neurotoxicity when the human exposure data  
5 indicates that the problem is chronic neurotox, even  
6 with acute toxicity exposure that the neurotoxicity  
7 developed later, involves specific regions of the  
8 brain? I would also like to hear a comment from you  
9 about something that came up yesterday.

10           In reviewing your documents, we heard  
11 yesterday that there was no evidence for any  
12 histopathological changes in the brain. But I just  
13 checked the series of articles that you provided us  
14 with, and there is nothing at all about what was  
15 studied or when or where in the brain this was  
16 examined. It seems to me that the neurotoxicity  
17 endpoints are critical here.

18           MS. RHODES: As a matter of process,  
19 Mr. Chairman, I will give two choices. I will  
20 either hand the question to one of our expert panel  
21 now or we do have a slide that deals with our  
22 opinion of the neurotoxicity situation later on. I  
23 will do whatever you would like.

24           DR. FROINES: If you have a slide, why  
25 don't we wait until we come to it.

1 MS. RHODES: Would that be acceptable to  
2 everybody? Okay.

3 The key for identifying the NOEL for nasal  
4 epithelial degeneration was a 13-week study in rats.  
5 The development of nasal toxicity is dependent upon  
6 substantial sustained depletion of glutathione,  
7 based on mechanistic studies. Literature data  
8 indicates GSH depletion must be greater than 50  
9 percent, and it must be sustained for damage. Nasal  
10 toxicity HEC for the PBPK Model, used by EPA and  
11 Arysta, relied on 50 percent. DPR, on other hand,  
12 as you have seen, relied on dose metric of 25  
13 percent GSH depletion. This results in a difference  
14 in HEC of about 2 HEC or 2 HEC over conservatively.

15 Arysta's position that the preponderance of  
16 data in literature supports EPA's conclusion of and  
17 use of 50 percent depletion.

18 Transient neurotoxicity. Another example of  
19 conservatism is found in the derivation of the acute  
20 neurotoxicity HEC. The exposure of rats to high  
21 levels of iodomethane for six hours resulted in an  
22 anesthetic-like transient neurotoxicity reflected in  
23 the reduced motor activity and reduced body  
24 temperature. All neurologic effects were  
25 short-lived and no neuropathology was detected.

1 EPA, Arysta and DPR are all in agreement as to the  
2 NOEL. However, the dose metric identified by EPA  
3 and Arysta, as the basis indicates, is the  
4 steady-state brain concentration of iodomethane.  
5 DPR uses the cumulative measure of iodomethane  
6 exposure or the area under the concentration versus  
7 time curve, which produces a significantly more  
8 conservative HEC with a three HEC difference as  
9 evidenced in the data in the table.

10 DR. MELNICK: Why do you prefer a  
11 steady-state level to what was the half life of  
12 methyl iodide in the brain?

13 MS. RHODES: I would like to hand this  
14 question over to Beth.

15 Do you understand the question? Could you  
16 restate it?

17 DR. MILESON: I think I do. Yes, I do  
18 understand the question.

19 We selected the steady-state brain  
20 concentration as the dose metric of interest here  
21 because it's a very short-lived effect. It's a --  
22 you know, quickly occurs and there is a fast  
23 recovery. I am not certain of the half life of the  
24 parent compound in the brain, but I can find that  
25 out for you.

1 DR. SLOTKIN: Based on lipid solubility,  
2 you would expect the brain concentration to be  
3 higher than plasma concentration at all time --

4 DR. MILESON: I don't doubt that. I would  
5 have to look that up, actually. And to try to -- I  
6 might ask you to -- I think this is the point you  
7 wanted to address your question, Ted. The first  
8 question that I sort of remember you saying or  
9 prefacing your question is that human data indicated  
10 that neurotoxicity is a chronic problem in humans.  
11 But we haven't seen any human data here today. But  
12 my impression in just a second is that most of the  
13 human data are from case studies of a single high  
14 dose exposure, and those effects developed over a  
15 long period of time.

16 DR. SLOTKIN: That is correct.

17 DR. MILESON: That is not really a chronic  
18 exposure.

19 DR. SLOTKIN: I didn't mean chronic  
20 exposure. I meant the chronic time course for the  
21 development of the neurological symptoms, as opposed  
22 to symptoms that appeared during -- immediately  
23 after the exposure. And the reason that is a  
24 concern is that later appearing deficits are  
25 consistent with neuro degeneration as opposed to a

1 local -- as opposed to an immediate anaesthetic-like  
2 effect.

3 DR. MILESON: I think what we see in humans  
4 is like a massive exposure of poisoning, but I don't  
5 think we see recovery in those humans as we do in  
6 the rats, where we see a transient effect. So you  
7 see a poisoning --

8 DR. SLOTKIN: I'm sorry, what you are  
9 seeing is just an immediate effect. That has  
10 nothing to do neuro degeneration. Neuro  
11 degeneration doesn't show recovery, obviously. We  
12 don't regenerate our brains the way we do our  
13 livers. So we are talking totally different things  
14 here.

15 My point was that the case study of acute  
16 poisoning shows late developing, irreversible brain  
17 damage. And yet I haven't seen anything that says  
18 that you guys followed your rats with your acute  
19 effects and shown me that this isn't a neuro  
20 degeneration later on or late appearing behavior or  
21 neurological problems.

22 DR. MILESON: I think they were tested  
23 maybe seven days and 14 days after exposure.

24 DR. SLOTKIN: I would like to see the data.

25 DR. MILESON: I would be happy to get that

1 to you. I want to sort of let you know that all of  
2 these studies that we talking about have been peer  
3 reviewed by DPR scientists, and those reviews are  
4 available for you should you ask for them.

5 DR. SLOTKIN: I need to see the data rather  
6 than someone else's review.

7 DR. MILESON: We would be happy to give you  
8 any report.

9 DR. FROINES: I just have to say that I  
10 think that the term "transient neurotoxicity" is  
11 probably not the best term. I don't think we are  
12 talking about transient neurotoxicity. And I think  
13 I am -- what I am saying is the same thing that Ted  
14 is. So that to use the word "transient" implies  
15 something different to me than what we actually see  
16 in the human case studies.

17 DR. SLOTKIN: This isn't really  
18 neurotoxicity as an endpoint. That is neurologic --  
19 this a behavior effect during the exposure period.  
20 Neurotoxicity involves something that I think more  
21 of a long-term than that.

22 DR. MILESON: What should we call this  
23 endpoint? Should we discount --

24 DR. SLOTKIN: I think to call the endpoint  
25 neurotoxicity and say that it is negative gives an

1 inappropriate cast to what was actually measured?  
2 You haven't really -- for anything that I have seen  
3 you haven't really assessed whether there is  
4 neurotoxicity. There are published things besides  
5 the case reports, such as study reports with  
6 cerebellar or granular cells that say that this  
7 compound actually causes neurotoxicity; that is cell  
8 destruction of neurons. That is not the same thing  
9 as an anesthetic-like effect on your behavior. I  
10 don't think that when you go for surgery, and if you  
11 get an inhalation anesthetic, that you would say  
12 that while you were anesthetized that's a neurotoxic  
13 effect. I think the semantics here are just wrong  
14 for what the endpoint is.

15 DR. MILESON: I would also submit that you  
16 can get toxicity in cells very easily that wouldn't  
17 necessarily indicate that neurotoxicity would occur.  
18 If you use a cell system, you elicit a  
19 neurotoxicity.

20 DR. SLOTKIN: Cell systems are less  
21 sensitive to neurotoxicants than in vitro.

22 DR. MILESON: We can talk about this  
23 anytime. So let's not bore the audience.

24 DR. BLANC: It's not boring at all.

25 DR. FROINES: I mean, this is -- I know we

1 have a time problem. Believe me, I know. This is  
2 one of the fundamental issues in this entire two-day  
3 discussion. Without question, the case reports in  
4 humans were really quite devastating when you read  
5 them. So I think somebody who makes his living  
6 dealing with issues like Ted does is obviously very  
7 focused on the interpretation.

8 MS. RHODES: If it's okay, I support your  
9 proposal. Either DPR can provide the report data.  
10 I think it is consistent for us to actually do what  
11 we'd be glad to do. You advise us. We are  
12 transparent. Any data that you --

13 DR. SLOTKIN: I appreciate that.

14 MS. RHODES: I am going to move long to  
15 your key issues. Go right to our key issues. I've  
16 already gone over the point, which is that we  
17 disagree on the NOEL. The rabbit developmental  
18 study was dosed at 0, 2, 10 and 20 ppm on gestation  
19 day six to 28. I bet you've all got that memorized  
20 by now. And I guess I support the point of end  
21 process.

22 And so finally, my dose groups were  
23 statistically significantly increased in late  
24 resorptions, decreased percent viable fetuses per  
25 litter, increased post-implantation loss, and

1 reduced offspring body weight compared to control.  
2 The dose rate, the 10 ppm group, however, had no  
3 statistically significant difference in no late  
4 resorptions, no effect on percent viable fetuses and  
5 no effect on post-implantation loss. The only  
6 significant effect found in this dose level was  
7 statistically significant decrease in female  
8 offspring body weight only, and was described by the  
9 study director as equivalent.

10 So it is Arysta's position that the NOAEL is  
11 10 ppm.

12 DR. MELNICK: We had a lot of discussion  
13 yesterday in terms of picking NOELs as well as  
14 benchmark dose. And it seems like the sense of the  
15 committee was that the benchmark approach was more  
16 appropriate.

17 Are you comfortable with that?

18 DR. FROINES: I think that record is clear  
19 on this issue, because we talked about it for  
20 literally hours. And so to now go back to the same  
21 issue, we are going to disagree, perhaps. And so  
22 it'd be better if we went ahead and didn't kind of  
23 relive old, yesterday's history.

24 MS. RHODES: I can assure you we will  
25 disagree. I know we don't agree with this

1 statement. I support moving on.

2 DR. FROINES: I want to give you the best  
3 chance possible to address issues that haven't --  
4 the record is clear on what this panel thinks is  
5 important from the discussion, and so I'm repeating  
6 myself. Go ahead.

7 DR. LOECHLER: I'm wondering if it is  
8 appropriate to ask them to provide some kind of  
9 written documents with greater detail as to why they  
10 think that the approach that was recommended  
11 yesterday is still inappropriate, after they heard  
12 the discussion? Could they provide us with a  
13 written document that we can go through those  
14 arguments?

15 DR. FROINES: I don't want to assume a role  
16 that I don't have any authority. So I think the  
17 answer to the question comes from Arysta, and we  
18 can't require anything.

19 DR. LOECHLER: Not require.

20 DR. BLANC: Let's go forward.

21 DR. FROINES: He is asking a question.  
22 Would Arysta be interested in providing information?

23 MS. RHODES: I think I will answer that in  
24 the same way I did about the data. Our  
25 understanding, this is Department of Pesticide

1 Regulation interaction with you, and we will take --  
2 have to take our guidance from them, as how they  
3 want to handle any questions that bring up any  
4 additional information that you require.

5 DR. FROINES: I think that is good.  
6 Everybody would be satisfied with that.

7 MS. RHODES: I will move on now to talk  
8 about a phased fate study that we did rather than an  
9 exposure study. This was conducted to determine the  
10 window of sensitivity and to better characterize the  
11 mode of action for the effect of the late fetal  
12 absorption.

13 I just take a little bit opportunity to walk  
14 people through the graphic. All of the graphics  
15 were dosed at 20 ppm dose. The X axis shows percent  
16 late absorption which range from 0 to 20 percent.  
17 The control is a group of rabbits that, of course,  
18 were not treated. The high bar was statistically  
19 significant in dosing in gestation day six through  
20 28. Each bar represents a group, distinct group of  
21 rabbits, that were dosed at these distinct time  
22 frames. And the point of this slide, and I know you  
23 all discussed it, is that there is an increase  
24 resorption in GD 23 to 24, 25 to 26, and really  
25 nothing happening at other points, which led us to

1 the conclusion that the window of sensitivity is  
2 gestation day 23 through 26 for developing rat  
3 fetus.

4 DR. BLANC: Just, maybe your technical  
5 people can answer this. But going back to the  
6 previous slide, another way of interpreting the data  
7 is that there is a synergistic effect with the  
8 exposure that occurs between day six and day 22  
9 which makes the 23 to 24 and 25 to 26 effect bigger.  
10 Or is the contention that it simply 23 through 26,  
11 if you added those together that would equal the  
12 other? What would be the interpretation? It could  
13 be interpreted either way.

14 DR. MILESON: We have interpreted the data  
15 to indicate that, if the rabbits were exposed for  
16 those four days of 23 to 26, we would see the full  
17 significant increase in late absorptions.

18 DR. BLANC: Thank you for clarification.

19 MS. RHODES: This is a graphic which  
20 represents -- let me back up, start over. I would  
21 like to state that iodomethane is readily  
22 metabolized to release iodine as you all have  
23 discussed, in the form iodide. It has been widely  
24 known for decades that pregnant rabbits are more  
25 susceptible to the intake of excess iodide. So we

1 examined the entirety of the developing rabbit fetus  
2 thyroid available in the literature compared to the  
3 window of sensitivity of iodomethane, the same point  
4 we just discussed, which is gestation days 23 to 26  
5 identified here in the orange.

6         The rabbit iodomethane window sensitivity  
7 occurs just as the rabbit fetal thyroid is rapidly  
8 accumulating iodide. Thyroid hormone production is  
9 beginning. Thyroid globulin is starting to  
10 accumulate in the thyroid follicle. This suggests  
11 excise iodide in the rabbit fetus may alter thyroid  
12 function, resulting in late resorptions. This  
13 preliminary information is providing the basis for  
14 two additional studies to further evaluate the  
15 effects of iodomethane exposure to pregnant rabbits  
16 and to establish the mode of action, which brings us  
17 back to the .2, which is that DPR did not accept the  
18 weight of evidence for developmental mode of action.

19         The postulated mode of action for this effect  
20 is that late resorptions in iodomethane in exposed  
21 rabbits are a direct effect of increased fetal  
22 plasma iodide. We emphasize fetal because DPR, of  
23 course, is using maternal plasma iodide.

24         I am sure all of you are familiar with this,  
25 so I won't belabor it, too. It was an evaluation of

1 the mode of action for developmental toxicity  
2 conducted by EPA, DPR and Arysta, as outlined in  
3 mode of action framework developed the International  
4 Program on Chemical Safety. This evaluation is  
5 performed using these eight steps. We have already  
6 shown you our postulate and mode of action. We  
7 intend to go over some key events, and we will  
8 outline alternative MOAs.

9         The first key event mode of action is that  
10 iodide is concentrated in fetal rabbits compared to  
11 the doe rabbit in both the control and treated  
12 rabbits with iodomethane. For example, in control  
13 rabbits the fetal plasma iodide level is  
14 approximately nine times what it is in the mother  
15 and in the controlled rabbits and was found to be  
16 six times higher. So I wanted to be clear, because  
17 not everybody in the audience might quite understand  
18 this. It's taken me a bit of time. But, basically,  
19 the fetal, developing fetal rabbit has nine times  
20 more plasma iodide than the mother. Fetal rabbits,  
21 as I mentioned earlier, are known to susceptible to  
22 excess iodide.

23         This is a graphic representation which shows  
24 the fetal rabbit's plasma compared to the maternal  
25 rabbit's plasma. And these are matched pairs of

1 animals, so mother to baby. On the left of side is  
2 representations of human plasma iodide; and note  
3 that there is no such concentration.

4 I believe you were all talking a little bit  
5 about these two studies a little bit earlier. The  
6 next key event for postulated mode of action is that  
7 excess iodide in the fetus produces effects on the  
8 developing thyroid of the rabbits fetus. As we  
9 demonstrated earlier the two [verbatim] days of  
10 sensitivity are gestation days 23 through 26.  
11 Excess iodide causes inhibition of thyroid hormone  
12 synthesis, resulting in an increase in thyroid  
13 stimulating hormone and follicular cell hypertrophy.

14 All of these effects were observed in the  
15 fetal rabbits exposed to 20 parts per million in the  
16 MOA studies. An additional intravenously  
17 administered sodium iodide study produced plasma  
18 levels that were lower than the 20 ppm iodomethane  
19 exposure. Similar effects on thyroid structure and  
20 function were observed in rabbits exposed to sodium  
21 iodide. But the effects were less severe as would  
22 be expected from lower plasma iodide levels.  
23 Therefore, it is Arysta's position that fetal iodide  
24 is the most appropriate dose metric for risk  
25 assessment.

1 DR. SLOTKIN: Just a second. It is true  
2 that the levels are lower with the sodium iodide,  
3 but the elevation of fetal TSH is exactly the same  
4 which would indicate to me that you have hit the top  
5 of the dose response curve for the iodide effect, if  
6 it is, in fact, on thyroid homeostasis.

7 MS. RHODES: Is there a question you would  
8 like us to address?

9 DR. SLOTKIN: It is a comment on whether  
10 the right metric here in terms of figuring out if  
11 you screwed up fetal thyroid hemostasis. It is just  
12 the serum iodide level or TSH level. Because it is  
13 true that the sodium iodide produces less of an  
14 increase in fetal serum iodide than the methyl  
15 identify, but it gave the same exact increase in  
16 TSH, which says to me that functionally, as far as  
17 the thyroid homeostasis, the two treatments were  
18 equivalent. Yet the sodium iodide didn't result in  
19 fetal death.

20 MS. RHODES: I'm sort of the referee. I  
21 don't know if you got a question? Or do you, Beth,  
22 have a question?

23 DR. FROINES: It is a question.

24 DR. MILESON: A quick response. I would  
25 say there are endpoints that were measured in

1 addition to TSH that are in the article in the  
2 *Inhalation Toxics Journal*, and those include  
3 histopathology, the incorporation of colloid and  
4 follicles and things like that. So we look at other  
5 endpoints in addition on TSH. You might look at  
6 some of the pictures in there of histopathology.

7 DR. SLOTKIN: I'll do that.

8 DR. MELNICK: Your emphasis is on the fetal  
9 rabbits susceptibility to iodide. Fetal rabbits  
10 susceptible --

11 THE COURT REPORTER: Can you talk into the  
12 microphone, please?

13 DR. MELNICK: Your focus is on the  
14 susceptibility of the fetal rabbits to iodide. Are  
15 fetal rabbits susceptible to alkylating agents?

16 DR. MILESON: I don't know. There is a  
17 long history of susceptibility in fetal rabbits to  
18 iodide. I have to look up their susceptibility to  
19 alkylating agents.

20 DR. FROINES: That is an issue which I  
21 think Ron would be interested in hearing about at a  
22 later date.

23 DR. MELNICK: The point is you're pursuing  
24 one pathway. One multiple pathways may be involved  
25 or it may be increased pathway. It seems to me

1 there is a need to distinguish. And one approach  
2 they used was sodium iodide as non alkylating agent,  
3 which the expectation would be a similar response if  
4 the effects are consequence to elevation in TSH.

5 MR. RHODES: Thanks for your comment. Beth  
6 is taking notes, and we will as we discussed the  
7 process.

8 DR. FROINES: I think your point is very  
9 well-taken and relevant. I also think it  
10 demonstrates a problem that we've been talking about  
11 for a few days; and that is there may be a vacuum in  
12 the data that we have to answer some of these  
13 questions. And that itself is a problem because it  
14 leaves us with trying to draw conclusions based on  
15 very limited information.

16 DR. HATTIS: I want to emphasize we are  
17 open to further arguments on this subject, in  
18 particular, because it is a very key portion of the  
19 argument, deciding what the appropriate mode of  
20 action is.

21 DR. SLOTKIN: Just as a matter of record.  
22 I just looked at Table 10 in the paper you referred  
23 to, and I don't see any difference in the  
24 histopathology between methyl iodide and sodium  
25 iodide in that table.

1 DR. MILESON: Maybe there aren't any good  
2 pictures, or something.

3 DR. SLOTKIN: I'm looking at numbers where  
4 you compile follicular cell hypertrophy and colloid  
5 depletion, and there is basically not much  
6 difference between the methyl iodide and sodium  
7 iodide. So just want to make sure the statement you  
8 made to me was accurate.

9 DR. FROINES: Let's move on. We are down  
10 to seven minutes, but we will run over another five  
11 minutes to make sure that -- because of the panel,  
12 again.

13 MS. RHODES: And my slowness of speech from  
14 the south. Thank you, sir.

15 Just quickly, just take this. These are three  
16 modes of action, alternative modes of action that we  
17 considered and rejected. I would point out there is  
18 a - -- what we call a white paper. It is called  
19 comments that were designed to come to you as an  
20 expert panel. That is basically a run-through of  
21 all of these type of things, mode of actions that  
22 you can look at at your leisure, if you can find any  
23 leisure now that you decided to take on this  
24 project.

25 In conclusion, the developmental toxicity MOA

1 was evaluated and identified using IPCS framework by  
2 EPA, DPR and Arysta. DPR performed MOA and  
3 determined that the data was insufficient. EPA and  
4 Arysta concluded the weight of evidence supports the  
5 MOA for fetal loss in iodomethane exposed rabbits as  
6 a direct effect of increased fetal plasma iodide.  
7 The MOA supports use of the fetal plasma iodide as  
8 the dose metric for extrapolation from rabbits to  
9 humans. And EPA and Arysta use this dose metric for  
10 extrapolation. DPR does not in their risk  
11 characterization document.

12 We are going to switch gears a little bit here  
13 and start relating a little more too the PBPK Model.  
14 And I would remind you that Dr. Lisa Sweeney is here  
15 and she can explain anything that you want to ask  
16 about it.

17 DR. FROINES: I think given the constraints  
18 that we won't, but yesterday we agreed that  
19 Dr. McKone and Dr. Hattis and Dr. Sweeney could talk  
20 off line to deal with any questions that might  
21 emerge.

22 MS. RHODES: That would be fine. Just send  
23 me the bill.

24 Okay. So I'm just going to move through this.  
25 EPA and Arysta assumed the human fetus is equally

1 sensitive to iodide as the rabbit. So for the mode  
2 of action of fetal rabbit loss due to excess fetal  
3 iodide, this is the assumption: The difference  
4 between humans and rabbits is that the human fetus  
5 does not concentrate iodide in the plasma compared  
6 to the maternal circulation as the rabbit does. And  
7 this has been reported in the literature by Cottino,  
8 et al. The human fetal maternal iodide ration is  
9 approximately one in women, Rayburn, et al., which  
10 is Dr. Bill Rayburn from the University of New  
11 Mexico, which is a peer review publication,  
12 conducted a human study, we are going to talk about  
13 in a second here, that proved that.

14 I want to be very, very clear and echo what is  
15 listed. Is that basically rather than just rely on  
16 data from the literature to determine whether human  
17 fetus concentrate iodide in their plasma compared to  
18 the maternal circulation, Arysta teamed up with  
19 Dr. Bill Rayburn, University of New Mexico Medical  
20 Center to measure iodide levels in maternal serum  
21 samples and in cord blood. I want to emphasize that  
22 these are routine blood samples taken in normal  
23 delivery ,and they are held in case there is  
24 something wrong with the baby and they are  
25 discarded. This study protocol was approved by the

1 University Institutional Review Board, and at no  
2 time were any women treated with any iodomethane. I  
3 just want to be clear about that. This study  
4 included collection and measurement of samples from  
5 29 premature births, 92 term births. And the bar on  
6 the right is a compilation of all.

7         And the message is that ratio worked out to be  
8 1.2 times between the fetal blood and the maternal  
9 blood, which pretty well supports the literature.  
10 It is this number was used the in PBPK modeling.

11         DR. BLANC: I think you better do something  
12 because even with the extra minutes, you are only  
13 two-thirds of the way through your slides. Right?  
14 You are going to have to either skip some slides --  
15 you should choose the slides that are the most  
16 important to you. We have your slides here, but  
17 otherwise it will be more than a full hour. And I  
18 think that will be unfair to some of our other  
19 presenters.

20         I think the most useful thing of your time  
21 would be not necessarily, as I said, exactly what I  
22 said to EPA ,not necessarily to reiterate the points  
23 you have already made in the document that have  
24 submitted, but to -- California has seen your  
25 response to their findings -- but to go beyond that

1 and say what you feel is new or your response to the  
2 response or some context like that.

3 MS. RHODES: I think I'll ask Dr. Froines  
4 to respond to that. You have just indicated to me  
5 that you would extend our time because of the  
6 questions.

7 DR. FROINES: I said I'd add another five  
8 minutes. That is 50 minutes. So I think that  
9 somehow we have to be fair to the rest -- to any  
10 people who want to make a presentation. And I  
11 appreciate your very special circumstances. You are  
12 the manufacturer of the product. But I think,  
13 therefore, Paul is right, that if you can select  
14 those things that you think are the most relevant,  
15 that would be helpful.

16 MS. RHODES: I call on you, Mr. Chairman,  
17 to extend to us the courtesy because we were  
18 confirmed just today by you that we had a full 45  
19 minutes. By my watch I have not gotten there when  
20 the comments were made. And I had not hit the end,  
21 so I would like your indulgence, please. I could go  
22 on and cover a little bit more stuff, or we can talk  
23 about the fact that we are going to use of our five  
24 minutes talking about whether there is five minutes.  
25 That is how it looks to me.

1 DR. FROINES: Go ahead.

2 MS. RHODES: Thank you very much.

3 Appreciate that. Sorry, Dr. Blanc, I take what you  
4 are saying in consideration.

5 So the message here is that -- I've go over  
6 this with you already, so I will drop to the bottom  
7 only line.

8 DPR based its HEC on the maternal plasma  
9 iodide level, dismissing the evidence supporting the  
10 fetal plasma iodide as the appropriate dose metric,  
11 which means using the 10 ppm NOAEL instead of NOEL  
12 instead of the two.

13 The next key issue is the uncertainty factor,  
14 and fact that DPR is indicating a data gap at  
15 chronic versus acute. We believe the uncertainty  
16 factor is not necessary because data submitted in  
17 PBPK modeling identify iodomethane exposure levels  
18 that do not cause effects to fetal or maternal  
19 thyroid. Basing the HEC on fetal iodide level, the  
20 offspring are never exposed to excess iodide, thus  
21 preventing adverse effects on the thyroid and  
22 neurodevelopmental effects. Therefore, DNT testing  
23 is not necessary.

24 I'd like to point out in the interest of  
25 brevity that we believe use of a chronic iodine,

1 upper tolerable, upper intake level is inappropriate  
2 for acute exposure.

3 Our last key issue would be to address the  
4 simplistic screening methods used for the exposure.  
5 And any questions you have, I would like to direct  
6 to Dr. Reiss because he did develop the Perfum  
7 Model, and I really encourage you to take advantage  
8 of his expertise while we are here.

9 I will quickly run down because there have  
10 been a lot of questions. There is a white paper in  
11 your packet, which is a compilation of all the 11  
12 flux studies. Just to clear the record, we did take  
13 personal occupational measurements out of six of the  
14 field trials. There has been a little confusion  
15 about that. All of those trials happen to be  
16 conducted in California, where iodomethane air  
17 concentrations were measured for workers within  
18 tasks. The workers included applicators, shovelers,  
19 tarp monitors, tarp punchers and planters. And you  
20 can read the results. And we reported them in parts  
21 per million. We believe that was what we heard  
22 yesterday as preferable.

23 DR. HAMMOND: Excuse me, point of  
24 clarification. That is the range that you reported  
25 is the range after you corrected and applied a

1 respiratory protection factor.

2 THE COURT REPORTER: I have to know who is  
3 talking, sorry.

4 DR. HAMMOND: Kathie Hammond.

5 THE COURT REPORTER: No, no,

6 MS. RHODES: Dr. Rick Reiss.

7 DR. REISS: This prior -- you will see in  
8 the next slide. We corrected for when presenting  
9 the applicator.

10 DR. HAMMOND: I thought I saw higher  
11 numbers.

12 DR. REISS: I will have to check.

13 MS. RHODES: I was going to let you present  
14 the next slide if you're going to go there. EPA  
15 estimated exposures using the maximum measured  
16 values across the six studies that we just  
17 discussed. DPR instead chose to subset the data  
18 several ways. I remember you all talking about it  
19 yesterday about concerns about that subsetting.  
20 They subsetted by application method and job  
21 category and then performed their statistical  
22 extrapolation. The bottom line of this slide, the  
23 four lower bars are the actual measured values from  
24 the field and real life conditions. And that's what  
25 we think that the estimation should be based on.

1 DPR's extrapolation as stated that this number  
2 is three times greater than anything that was  
3 measured in any field study.

4 Is there a question?

5 DR. HAMMOND: Just noting that the  
6 respiratory protection factor isn't monitored.

7 DR. REISS: With the higher number, you  
8 must always -- probably extrapolated values compared  
9 to the real value.

10 MS. RHODES: Did you get the answer you  
11 were seeking?

12 DR. HAMMOND: I heard his answer.

13 MS. RHODES: We move quickly to bystander  
14 exposure. We have conducted 11 bystander studies  
15 and we believe our flux studies, most were conducted  
16 in California. Each one provides flux estimates for  
17 two weeks after the application. I recollect you  
18 all discussed that yesterday.

19 The mass balance shows 80 to 100 percent of  
20 applied volatilized.

21 DR. HAMMOND: I do have a comment to this  
22 one slide. As far as I know, we don't have the  
23 actual input data to that flux model. I would be --  
24 I couldn't find that in anything that we have. I  
25 would really like to have the actual values that

1 were measured.

2 DR. REISS: That is fine. We can provide  
3 that to you.

4 DR. HAMMOND: Thank you.

5 DR. LOECHLER: Just to comment. Those  
6 weren't the numbers that I remember being reported  
7 yesterday. I don't remember any number at 100  
8 percent volatilized. The numbers -- the first  
9 number we heard was 50 percent with the increase,  
10 but it is 30 to 80 or something like that. I have  
11 them noted.

12 DR. REISS: Let me clarify. There may have  
13 been some confusion in what was presented. There is  
14 generally two numbers that are provided:  
15 volatilization of the first day, the first 24 hours.  
16 And my recollection is that range is between 30 to  
17 60 percent. But then when we do a mass balance,  
18 where we look at the volatilization over the entire  
19 two weeks of measuring period. That is generally  
20 about 80 to 100 percent of the material that we can  
21 account for. I would note, given the error in  
22 methodology, you can't differentiate that a hundred  
23 percent.

24 MS. RHODES: If there's not another  
25 question in this area, I'm going to skip forward to

1 the summary piece, which is we'd just like to make  
2 the point that DPR's methodology that they use for  
3 exposure is at odds with the recent National Academy  
4 of Science's recommendation, which is that Arysta's  
5 estimates can be most fully characterized by  
6 performing probabilistic analysis when possible and  
7 by presenting the range of possible risk estimates  
8 rather than by reporting a single point estimate  
9 that we saw yesterday.

10 I need to get a couple choices. I've got a  
11 little bit carcinogenicity and a little bit on  
12 environmental fate. I know that is of keen interest  
13 to the panel.

14 DR. BLANC: It there is nothing different  
15 in your position on the carcinogenicity to what you  
16 commented onto the California Department of Food and  
17 Agricultural, your comments to them, then I would  
18 just skip that.

19 MS. RHODES: There is quite a bit more. I  
20 do not believe, correct me if I am wrong, John, I  
21 don't think that carcinogenicity was emphasized very  
22 much in the Arysta characterization document in the  
23 first draft, and I don't think we've ever actually  
24 done too much about it responding to it, other than  
25 if you reviewed the - what we call the - white paper

1 July 9th paper. It is your call, whatever you would  
2 like me to do.

3 DR. FROINES: Well, you have five minutes.  
4 That is it.

5 MS. RHODES: I will use it to my best  
6 advantage that I can, then. I am looking to see  
7 what I think that is going to be. Give me two  
8 seconds here.

9 I'm going to just cover this and close it and  
10 hit the environmental fate.

11 DPR revised risk characterization document  
12 concluded that there was -- that iodomethane should  
13 be considered as a potential human oncogen because  
14 of tumor formation in male rat brains, astrocytoma,  
15 mouse uterus/cervix, rat and mouse thyroid. We  
16 believe that this is not a problem. In both cases  
17 the tumors are not seen to be caused by iodomethane.  
18 And I'd love to go through it in a little more  
19 detail, but you have the slides.

20 But basically the effect for astrocytomas have  
21 always been historical controls for laboratory  
22 within the concurrent controls and within the  
23 breeder controls. As far as the uterus/cervix  
24 combined, just like to make sure that you are aware  
25 that independent pathology working group chaired by

1 Dr. Jerry [unintelligible] found that mice fibromas  
2 were not caused by iodomethane. The incidence of  
3 fibromas in the high dose group was a function of  
4 the unusually low control incidence, which you will  
5 notice happens to be zero. The working group also  
6 noted that precursor lesions that would be expected  
7 in the animals were not found and that uteran and  
8 cervical fibromas were not found to the two-year rat  
9 study with iodomethane.

10 In the interest of time I will just close this  
11 part out by saying the changes that were seen are  
12 altered thyroid [unintelligible], hyperplastic  
13 change, thyroid tumor result, hyperplastic changes  
14 in thyroid cell architecture, elevated TSH levels,  
15 diminished T3 or 4 levels, that are all indicative  
16 of altered pituitary thyroid function and matched  
17 the criteria for thyroid follicular cell  
18 [unintelligible] established by the EPA risk  
19 assessment form. Tumor associated with altered  
20 thyroid pituitary function are associated with  
21 chronic reduction in thyroid hormones and an  
22 increase in thyroid stimulating hormones.

23 The RAF concluded that sustained perturbation  
24 of thyroid hormone homeostasis is an MOA for tumors  
25 formed under these circumstances, and their margin

1 exposure is similar nonlinear effect assessment be  
2 used for low dose extrapolation.

3 I would just like to point out that I may have  
4 failed to introduce John Butala. Would you raise  
5 your hand? He would be the person I would seek out  
6 to explain that position.

7 DR. MELNICK: I believe that the EPA cancer  
8 risk assessment guidelines for mutagens don't allow  
9 strictly a non-mutagenic mode of action.

10 MS. RHODES: John, would you like him to  
11 rephrase question? Did you hear it well enough?

12 MR. BUTALA: I heard him. What we are  
13 looking for in this -- if you're referring to the  
14 risk assessment form guidelines, the fibroid tumors,  
15 they lay out several possible causes, if you will,  
16 just the way the mode of action's termed. And  
17 certainly one of those is a genotoxic etiology. One  
18 of them is -- the one we referred to here which has  
19 the characteristics that Ms. Rhodes just read off to  
20 you on the slide. It does not include a genotoxic  
21 component. Hence, the nonlinear extrapolation of  
22 low dose. That is what we are referring to.

23 DR. MELNICK: I know what you're referring  
24 to. I'm saying it is somewhat inconsistent with the  
25 USEPA guidelines of considering the mutagenic mode

1 of action.

2 MR. BUTALA: Are you referring to the  
3 mutagenic mode of action in general or specifically  
4 for these rodent thyroid follicular cells?

5 DR. MELNICK: In general. And as mentioned  
6 earlier, there is a potassium iodide two-year study  
7 which did not produce rodent tumors.

8 MR. BUTALA: I think the way we can resolve  
9 that, and we can do that off-line, is to simply  
10 compare the two EPA documents and one specific for  
11 the rodent follicular cell tumors and then the more  
12 general guidelines.

13 MS. RHODES: Just quickly. I am going to  
14 move us along, if it is all right. If you guys can  
15 get together or if you got -- we can provide some  
16 more information. Be glad to.

17 DR. FROINES: I want to say one thing. We  
18 have a list of papers on mutagenicity that is this  
19 long. And you have four papers in your document.  
20 And what I would like to have happen over the --  
21 between now and as we move the process on is for we  
22 will provide you with what we have so you know what  
23 we are thinking are the positive and negative  
24 mutagenicity studies. If you could respond so that  
25 we have a sense of why there is such a significant

1 difference between what we found and what you  
2 reported. And so I would like to try to get  
3 resolution to the intellectual issues associated.

4 MR. BUTALA: If I may. I've had that  
5 thought myself over the last day and a half  
6 listening to you. I think that is the way to go  
7 forward. We have to compare these studies, you  
8 know, essentially for methodological differences,  
9 and look at the best and come up with the weight of  
10 evidence for what it means. I think -- I like your  
11 idea.

12 DR. FROINES: I think the communication  
13 will be -- I know I'm promising people's time here,  
14 so I may get murdered when I walk out of here. But  
15 the fact of the matter is I think communication,  
16 approved solid communication, is going to be the  
17 best approach. And it goes to the issue that Ron  
18 was raising, indirectly.

19 DR. LOECHLER: You write -- this is the  
20 bottom right corner of this slide. Reported as  
21 positive for DNA alkylation, however, interference  
22 from the novo synthesis appears to have occurred.  
23 What does that mean?

24 MR. BUTALA: The test compound with C-14  
25 labeled methyl iodide did turn up or were detected

1 in tissue in the animal after administration. The  
2 question is, well, were they true adduct formations  
3 of a methylation event at the typical sites, or was  
4 there something else? And what we are saying is we  
5 have evidence of this, that the C-14 that is  
6 incorporated was not incorporated into specific  
7 sites along DNA or proteins or even RNA as a result  
8 of alkylation, but instead as a result of carbon,  
9 just carbon, that happened to be radio labeled as  
10 well. Incorporated into the carbon pool of the  
11 cells. That is what I'm talking about.

12 DR. MELNICK: Excuse me. There are  
13 published studies. I think you're referring to one,  
14 [unintelligible] laboratory, which they identified  
15 by using mass spec as methyl 1. That is not from  
16 the carbon pool.

17 MS. BUTALA: That is not from the carbon  
18 pool, but I think there is additional information as  
19 well that suggests there is.

20 DR. MELNICK: They look at the total C-14  
21 and they looked at specific adducts which they  
22 identified. There were two adducts.

23 DR. FROINES: Not to break up this  
24 lovefest. This issue of de novo synthesis is also  
25 in the DPR document. It's a problem in the DPR

1 document and it's a problem here, obviously, as you  
2 can tell. So, again, this is something that we need  
3 to communicate with DPR. We need to talk to OEHHA  
4 about. We need to communicate with you. And so I  
5 don't want this issue of de novo synthesis left  
6 hanging because it gets asserted. What we need to  
7 do is to try and develop the science to answer the  
8 question as best we possibly can.

9 DR. LOECHLER: I would ask for the same  
10 thing I asked a few minutes ago. If you would be  
11 willing to provide a written document that goes  
12 through your arguments and analysis, I would  
13 appreciate receiving that document for  
14 consideration. If that's possible.

15 DR. FROINES: As far as I'm concerned from  
16 our standpoint, of course, that is a very logical  
17 request. And Arysta will obviously take into  
18 consideration and get back to DPR, and DPR will get  
19 back with us, and we will go from there.

20 MS. RHODES: I support your suggestion  
21 area, Dr. Froines, that you all share with us what  
22 you have, and perhaps that would help us know what  
23 to do next. You obviously have information that we  
24 don't have.

25 DR. MELNICK: This issue is totally

1 incorrect. The paper is published. It is  
2 Gansewendt paper of 1991 which identifies specific  
3 adducts. They looked at the total label and  
4 identified specific adducts. It's undeniable.

5 MR. BUTALA: I think additional  
6 incorporation in C-14 was identified as well.

7 DR. FROINES: I am going to close because  
8 what you will do is you will make a coherent  
9 response and he is going to come back. So this is  
10 issue we can resolve off line.

11 MS. RHODES: With that advice, I am going  
12 to move quickly to fate, environmental fate.

13 DR. FROINES: I'm afraid we have to stop.  
14 We've gone an hour and five minutes. Admittedly,  
15 this hurts you. But I think it is -- at this point  
16 if you would sort of draw your conclusions we would  
17 -- that would be appropriate.

18 MS. RHODES: I guess all the way to my very  
19 last conclusion. Other than the fate area, we  
20 covered everything. Just wanted to say in  
21 conclusion that we believe DPR risk characterization  
22 document is excessively conservative and has taken  
23 the most conservative options available at the every  
24 decision point. Overly conservative assumptions. I  
25 would just like to put an idea in your mind. If

1 they don't have benefits, they are not added  
2 protection. They do have a cost that also has to be  
3 balanced. One cost of that could be the extension  
4 of the critical use exemption for methyl bromide, an  
5 ozone depleter. I would like to remind you it would  
6 not be used except to get critical use exceptions  
7 because there is no alternative.

8           Increased use of non [unintelligible]  
9 pesticides as we talked about earlier, loss of  
10 production of California crops. And we believe the  
11 small farmers will be hit the hardest. Increased  
12 imported fruits and vegetables from countries with  
13 minimum health and safety standards, minimal  
14 pesticide enforcement. Additional safety margins  
15 will not increase safety and do not have a benefit.  
16 They do have a cost in making the valuable tool, a  
17 replacement for a known ozone depleter to onerous to  
18 use.

19           I just ask you to consider all the data we  
20 presented or we are happy to provide to you to make  
21 your decision. Appreciate your time.

22           DR. FROINES: Thank you very much. Thank  
23 you very much for participation and have everybody  
24 come with you.

25           I just want to make one comment. I want to

1 disagree 100 percent with what you have there.  
2 Because this panel, as far as I am concerned, is  
3 here to look at the science and only the science.  
4 We have no policy. We have no economics. We have  
5 no political science. We have no sociologists. We  
6 are not -- we have no knowledge nor mandate to  
7 address issues that take us outside the toxicology,  
8 epidemiology and other scientific factors.

9           So I would say that this panel has to look at  
10 only the science and go from there.

11           DR. BLANC: Can I ask a quick question? If  
12 you could point us in the right direction in the  
13 published volume of *Inhalation Toxicology* or  
14 somewhere else in materials that we wouldn't  
15 necessarily have seen. Just for our edification.  
16 Dr. Hammond commented last time a very interesting  
17 question. It is: What is the mechanism of  
18 lethality to nematodes of the methyl iodide? How  
19 does it kill the target pest?

20           MS. RHODES: I am not sure we have anybody  
21 -- we have somebody in the audience that may -- that  
22 might be able to answer that question.

23           DR. BLANC: Don't take time now. Just  
24 provide us some data just for whatever insight we  
25 could -- for example, if it killed nematodes by

1 methylating them, that would be of interest for us  
2 to know.

3 MS. RHODES: That is a question we can  
4 answer for you fairly easily when we get the right  
5 person to the microphone. In interest of your main  
6 objective here.

7 Again, thank you so much.

8 DR. FROINES: Thank you very much. I don't  
9 want to in any way think that -- I think it is  
10 really important for this panel to focus only on the  
11 technical issues associated with this. I want to  
12 assure that that is what we do.

13 MS. RHODES: Thank you.

14 (Break taken.)

15 DR. FROINES: Susan, please introduce  
16 yourself -- however you want to do it.

17 MS. KATTEN: I am Anne Katten, from  
18 California Rural Legal Assistance Foundation, and I  
19 am an industrial hygienist by training. And thank  
20 you for having me.

21 Just wanted to emphasize the central coast, in  
22 particular, is a patchwork of labor intensive crops  
23 as the whole state agricultural areas so.

24 DR. FROINES: Can I stop you. On two  
25 separate occasions, one with EPA and one with

1 Arysta, Paul raised an issue of can you focus on  
2 things that we don't already know. And so please  
3 try and do that.

4 MS. KATTEN: As you know, residential and  
5 schools are close to fields. I think DPR has made  
6 many reasonable assumptions in estimated exposure.  
7 One that hasn't been mentioned is that they assume  
8 to calculate to the maximum label rate. That is  
9 really good and really important. And also I would  
10 differ with the manufacturers. I think that the use  
11 of the 95th percentile exposure to compensate for  
12 the very small data sets for applicators is  
13 critical.

14 There are some assumption, though, that  
15 underestimate worker exposure, and I'll go right  
16 into them. First, we have talked about using the  
17 tenfold protection factor for respirators. First,  
18 if the regulation is followed, you can use either a  
19 quantitative or qualitative testing. Of course,  
20 there is no number check with qualitative. It you  
21 use quantitative, there is no guarantee that when  
22 you finish shoveling all day in the hot sun you're  
23 going to get the same protection factors.

24 Also, among violations in California that are  
25 documented, personal protective equipment violations

1 and pesticide handler training are the top two types  
2 of violations. So there is no guarantee people are  
3 going to get their testing. Get their cartridges  
4 changed out where they're supposed to and other such  
5 things.

6 In addition, Dr. Nicas and Dr. Neuhaus  
7 recently conducted a -- published a study about  
8 respirator fit, and this was a review, a statistical  
9 review of the studies where they looked at  
10 respirator fit during the workday. I think most of  
11 them were looking at concentrations inside and  
12 outside the respirator. Five of seven of these  
13 studies, the protection factor was below or close to  
14 five. And based on this, they recommended that a  
15 protection factor, an assigned protection factor,  
16 should be reduced from ten to five. Because right  
17 now it is not just DPR, EPA, OSHA are both using  
18 assigned protection factor of ten. They also found  
19 that protection factors were smaller in studies  
20 involving gases and small particles.

21 DR. HATTIS: Were these agricultural  
22 studies or industrial?

23 MS. KATTEN: They were industrial, styrene,  
24 cement, stuff like that.

25 DR. HATTIS: They've never actually studied

1 an agricultural --

2 MS. KATTEN: Not to my knowledge.

3 DR. HATTIS: Probably not likely to be good  
4 data.

5 MS. KATTEN: Right. Good point. It is  
6 likely to be lower because of factors we've already  
7 discussed.

8 Then I tried to put in the data -- I use data  
9 from the appendix on Volume 2 on Page 73. And this  
10 is just to point out that if you have the -- if  
11 these -- here are the exposures for the applicator  
12 and tarp monitor and the shoveler have been adjusted  
13 for tenfold protection factor. So, obviously, their  
14 exposures are going to be a lot higher without that  
15 and be way, way higher than these levels.

16 Another point where I think DPR under  
17 estimated, and is they assumed applicators are  
18 exposed only three months. We know there are  
19 applicators who work for large farms who travel up  
20 and down the coast. There two additional months of  
21 fumigation in the Ventura area, so that brings it to  
22 five months. And if you did a more detailed  
23 analysis, including the Central Valley, it could be  
24 even longer. We also don't think that an eight-hour  
25 workday is a reasonable worst case estimate for

1 agriculture. Because pesticide applicators and  
2 other agricultural workers, they work seasonally.  
3 So they are going to take any hours they can. They  
4 are not entitled to overtime pay rate until after  
5 ten hours of work a day. And irrigation workers who  
6 move around a lot of fields, they are going to get a  
7 lot of exposure. They are completely exempt from  
8 overtime payment.

9 DR. FROINES: What was the last? You went  
10 too fast.

11 MS. KATTEN: I think I covered all those  
12 points. The last one is just -- they are also  
13 exposed during transit to and from work and at home,  
14 which actually DPR did accommodate for somewhat,  
15 maybe not enough.

16 Then I also think there can be significant  
17 dermal exposure. During those drip applications the  
18 routine duties include repairing the drip lines and  
19 tears in the tarp. And actually, if you look at the  
20 label, which I will show next, the directions say  
21 you are not allowed to wear gloves because the  
22 gloves can trap the vapors next to your skin, if the  
23 chloropicrin or methyl iodide, and cause burns that  
24 way. And then the MSDS, both says use gloves and  
25 use Viton, which is a very expensive type of glove

1 that might be provided. But it later says not to  
2 use gloves, which I will show.

3 I would also say if one does look at dermal  
4 absorption, they should assume 100 percent since  
5 there isn't any data available. I believe the risk  
6 assessment makes no other kind of estimate.

7 DR. HATTIS: What do you mean by 100  
8 percent?

9 MS. KATTEN: I believe there is a complete  
10 data gap on dermal absorption. So in the absence of  
11 data, I would assume that all of the chemical -- I  
12 would like to see that assumption, that all is  
13 absorbed. That is all I mean.

14 DR. HATTIS: Are you talking relative to  
15 loading that is in the document?

16 DR. FROINES: I think she is saying, given  
17 the fact that the document says zero, that your  
18 alternative is a hundred.

19 MS. KATTEN: Well, it is 100 percent of  
20 --there was a calculation from the vapor, and they  
21 assumed it was negligible. There was some sort of  
22 estimate that the absorption would be 40 percent  
23 based on some analogy to some other chemical. I  
24 don't remember all the details. I just thought  
25 there that you don't much data, you should assume

1 absorption rate, I should have said there, of 100  
2 percent. You all have more experience to make that  
3 the decision than I do.

4 DR. HAMMOND: The point you're making, if I  
5 hear it correctly, is that we should not neglect  
6 dermal absorption. It is an important route of  
7 exposure, and I agree, also. The document focuses  
8 more, as you said, on gas things. This is something  
9 people look at, gas vapor phase absorption  
10 throughout your body. But on top of that there is a  
11 direct contact that one might have in doing the  
12 tasks necessary. Where those happen, one should --  
13 your suggestion which I assume is a hundred percent  
14 absorption right from where the contact is.

15 I think what we are missing -- you have the  
16 concept of how much contact there is, but we don't  
17 have that. We have to make assumptions, or one  
18 should make such assumptions.

19 DR. FROINES: I think Kathie is exactly  
20 right. And the confusion was that the quantitative,  
21 the 100 percent number.

22 MS. KATTEN: I should have said rate.

23 This is the instruction on the label about not  
24 wearing gloves, and then MSDS in blue where it says  
25 don't wear gloves and where it says do wear gloves

1 and then it says don't wear gloves.

2 DR. FROINES: Could you go back for a  
3 second?

4 MS. KATTEN: Then also the case study have  
5 been alluded to, and I believe this is one that most  
6 of you have reviewed where there was a man who had a  
7 regular job, an industrial job, where he tanks the  
8 methyl iodide to a truck. And he was wearing a  
9 chemically protective suit and air-supply  
10 respirator. So inhalation exposure at the end of  
11 shift, he saw that there was a breach in the sole of  
12 this suit, the foot, and then later, after work,  
13 after he took a shower he noticed a rash. He  
14 fainted. He went to the ER. He had more severe  
15 skin symptoms develop and then he developed memory  
16 and other neurotoxicity problems. Which I know has  
17 other implications, too, but has implication that  
18 you can get a little bit of dermal from, like, when  
19 you're changing the canister or something, and it  
20 will be a big effect.

21 DR. HAMMOND: I was unclear, still not  
22 clear. The breach is in the sole of the suit. But  
23 he had shoes on. So, I mean, my question is how  
24 much exposure was that and what would that have  
25 been? We don't know. I am trying to think it

1 through. He was wearing shoes, I assume, and he is  
2 wearing shoes is possible that some liquid got on  
3 the sole of the shoe as maybe just vapor phase in  
4 there. Unless there was a spill or what -- that was  
5 the other question. I saw no indication of a spill.  
6 At one level it is the type that should have been  
7 enclosed and should be nothing without the  
8 protective suit. All I can figure out, as I try to  
9 figure what scenarios is happening there. I don't  
10 think we have enough information. It seems like it  
11 must have been a very tiny exposure.

12 MS. KATTEN: I have all those same  
13 questions.

14 DR. HAMMOND: Does anyone know about this  
15 case? I thought you knew the case.

16 DR. FROINES: I think that you are  
17 absolutely right, that the health effects that got  
18 emphasized in the report were really excellent and  
19 relate to all the things Ted has been talking about.  
20 But that in the beginning of the report the  
21 description of what actually happened was not very  
22 well not and complete as one might have.

23 DR. BLANC: They specify that he wasn't  
24 wearing an outer boot over the protective suit. So  
25 he had -- whatever shoes he had on, they were inside

1 this. Even though he wasn't barefoot inside the  
2 protective suit, and then there was a breach in the  
3 foot area of the protective suit, and there wasn't a  
4 boot on around the protective suit. I think the  
5 point -- you are using it to make the point that in  
6 the exposure scenario where there is little data to  
7 suggest a substantive inhalation exposure and  
8 exposure was to the skin, either from liquid or from  
9 vaporized material, there was a significant health  
10 effect.

11 Isn't that the point you're trying to make?

12 MS. KATTEN: Right. And it wasn't detected  
13 right away.

14 DR. FROINES: There was one point I would  
15 make. That is that so often with -- and this  
16 happens in industrial general all the time. So  
17 often when you see reports of case studies, one  
18 always assumes that the exposures were off the  
19 charts. And that they there are no such things as  
20 case studies with low exposure, and that assumption  
21 may be right much of the time, but it always not  
22 right all the time.

23 DR. LOECHLER: That was the point I was  
24 going to raise. This is possibly indication of a  
25 particularly sensitive individual. The reason why

1 we need these extra factors of ten in these kinds of  
2 deliberations.

3 MS. KATTEN: Other limitations that I am  
4 not sure about, the total significant, I noticed  
5 that most the measurements of the application  
6 personnel were five to six hours long. You don't  
7 have any idea of what the short peak exposure could  
8 be in that period of time. And then they didn't  
9 measure the chloropicrin exposure. It would be good  
10 to know this, of course. And then I don't know if  
11 this is significant, but they used the 71 kilogram  
12 body weight where some of the bystander workers were  
13 much lower weight.

14 And then, just switch gears, but I had heard  
15 about this monitoring of the wells in Florida. I  
16 did check. I called around some Florida EPA folks.  
17 I want to let you know that the monitoring had been  
18 planned in Florida, but it hasn't been initiated  
19 yet. So there isn't any data there yet.

20 I have additional concern about the iodide  
21 because many rural wells don't get any monitoring.  
22 It is connected with one farm or something. And  
23 then the smaller systems rarely get monitored, so  
24 detecting a problem would be much harder.

25 Thank you.

1 DR. KEGLEY: My name is Susan Kegley. I am  
2 a Ph.D. organic chemist. I work with PAN as a  
3 consultant chemist. PAN is a national organization  
4 that we are also exploring with a number of other  
5 groups that is pesticide action networks to promote  
6 sustainable methods of pest control to try to get  
7 rid of the most toxic pesticides that are currently  
8 in use. I would like to begin with a quick  
9 overview. I am not -- I have a lot of slides that  
10 you guys have already talked about. Basically  
11 chemical profiles, something about use trends and  
12 how that might affect what goes on with methyl  
13 iodide and some comments that you haven't hit for  
14 the health risk assessment and exposure assessment.

15 We talked about the paper has been cited.  
16 Basically, methyl iodide is an alkylating agent, and  
17 actually several studies that show that it alkylates  
18 DNA. Isolated DNA has been extracted and analyzed.  
19 So, basically, it gets into the cell, and there is  
20 obviously a lot of possibilities for what goes on  
21 there.

22 DR. FROINES: As a chemist, I want to say  
23 thank you very much. You are the first person to  
24 put up a chemical structure.

25 DR. KEGLEY: Few regulatory facts. It's

1 been on the California Proposition 65 list of  
2 carcinogens since 1988. It is listed by NIOSH as a  
3 potential occupational carcinogen and EPA classifies  
4 it for industrial powers as a hazardous air  
5 pollutant. If you use this chemical in industrial  
6 or academic lab -- well, in industrial lab in  
7 particular, you have to fill out reams of paperwork  
8 to be able to receive small amounts of this material  
9 into the environment.

10 Chemists who are working with this chemical in  
11 the lab have double gloves, syringes for  
12 transferring uses you are very, very careful with  
13 it. It is well-known among chemists that it is not  
14 a molecule to play around with. We use it in  
15 chemical reactions because it works. It alkylates  
16 very effectively.

17 This is just another application. It is just  
18 remarkable that the guys over there doing the  
19 shoveling don't have any respirator protection.  
20 This is a methyl bromide application. So I want to  
21 spend a little bit of time on this one. Because it  
22 illustrates a point that is relevant to methyl  
23 iodide.

24 Basically, fumigant use in California has been  
25 approximately the same, at about 30- to 35,000,000

1 pounds per year since about 1988; and 2007 is the  
2 latest data set we have. You can see that the  
3 different mix of fumigants has changed over the  
4 years. You can see the methyl bromide phase out  
5 coming in, but it's kind of stagnant in the last few  
6 years. More chloropicrin is being used. Probably  
7 the most remarkable part of that graph is Telone,  
8 the brown line. And in 1988, about 16,000,000  
9 pounds of Telone was being used a year. In the  
10 1991, air monitoring occurred that showed levels of  
11 Telone in the air which is also a Proposition 65  
12 carcinogen there were way above levels of concern.  
13 It got pulled immediately from use by DPR. They  
14 work closely with the registrant, which is  
15 AgriSciences, to develop application methods to put  
16 less Telone in the air. They reintroduced in 1995  
17 under very restricted use conditions. It can only  
18 be used in a few counties. Limited number of pounds  
19 per year per township could be used, and it couldn't  
20 accrue use every year. And these restrictions --

21           So there was a great deal of public scrutiny,  
22 like methyl iodide is getting a great deal of public  
23 scrutiny now, and it was pulled. And slowly over  
24 time all of those protections have eroded.

25           I guess the point I would like to make, I

1 know you don't want to talk about restrictions, if  
2 it does get registered, the chemical falls under the  
3 tent. Even if it is registered for just a few uses,  
4 there is no public scrutiny afterwards. It is just  
5 a discretionary decision on the part of DPR. And  
6 you can see what's happened with Telone. In 2007,  
7 9.9 million pounds were used -- 9.4. As far as I  
8 know, the analysis that caused it to be pulled in  
9 1991 has not been redone yet.

10 DR. HATTIS: Following up on this. I know  
11 John is going to be terribly unhappy for me to ask  
12 this question. We and DPR are constrained by the  
13 legislative framework that they are operating under  
14 to evaluate chemicals in uses one by one. Have you  
15 considered the possibility of expanding the  
16 framework to allow multiple chemical comparative  
17 analyses for comparative technology or control that  
18 might be more informative from a risk management  
19 standpoint?

20 DR. KEGLEY: I am not understanding. Have  
21 I considered?

22 DR. HATTIS: Have you or your organization  
23 thought of the possibility of restructuring the  
24 basic framework for analysis?

25 DR. KEGLEY: For risk assessment?

1 DR. HATTIS: Yes.

2 DR. KEGLEY: It would require new  
3 legislation, and, yes, we thought about that. It  
4 would be quite a bit to get that passed through the  
5 Legislature.

6 DR. HATTIS: They are otherwise occupied, I  
7 would think.

8 DR. KEGLEY: There is a lot of other  
9 chemicals and air pollutants that were separate  
10 mandates that should be combined.

11 DR. HATTIS: I would agree with that.

12 DR. FROINES: The irony is -- we have to  
13 stop this risk management discussion, but the irony  
14 is the Scientific Review Panel under AB 1807 is  
15 taking up chloropicrin next month. And so we have  
16 this irony going on where we are dealing with methyl  
17 iodide on the one hand and simultaneously we are  
18 dealing with chloropicrin and never the twain shall  
19 meet. Any logic whatsoever would tell you that that  
20 is not necessarily the best way to have approached  
21 this issue, but that is the way it is going.

22 DR. MCKONE: The Academy study, the science  
23 industry, has a whole chapter on what is called a  
24 solutions based risk a assessment as opposed to  
25 chemical-specific assessment. We have asked for

1 exactly this and outlined how it can be done. It  
2 might be a starting point.

3 DR. FROINES: We have to move on.

4 DR. SLOTKIN: I have a specific question.  
5 Refresh my memory, if it is Prop 65 chemical,  
6 doesn't that mean that all we have to work with is a  
7 LOEL rather than NOEL? We have to apply an  
8 additional tenfold safety factor?

9 DR. KEGLEY: For carcinogen analysis it is  
10 not LOEL, NOEL. They use a linear --

11 DR. HATTIS: For the cancer, but there is a  
12 provision for reproductive developmental toxicant  
13 that doesn't require an extra --

14 DR. SLOTKIN: And extra ten for a LOEL as  
15 opposed to a NOEL on top of everything else.

16 DR. HATTIS: It isn't obvious to me, and  
17 this is my legal opinion, my legal understanding  
18 speaking as a geneticist, it isn't obvious to me  
19 that Prop 65 comes into the pesticide.

20 DR. SLOTKIN: I was just curious. If it is  
21 listed as a Prop 65 chemical, that might be  
22 something that we need to consider in our  
23 deliberations.

24 DR. KEGLEY: So why are we all concerned  
25 about this? Because you can't easily integrate the

1 fumigant under a circumstances where you're putting  
2 volatile chemicals in the ground in the summer in  
3 the Central Valley through the protocol. And so  
4 here are some instances that have occurred. A lot  
5 of times hundreds of people are evacuated from their  
6 homes and have to leave. It usually happens in the  
7 evening when the weather, meteorological conditions  
8 stabilize and you get inversions. A lot of these  
9 incidences happened or some of the incidences  
10 happened because of mistakes that applicators made.  
11 Those will happen. The last one, in Yerington,  
12 Nevada, in 2007, chloropicrin. Twenty-four people,  
13 workers working in an adjacent field about a half  
14 mile away, were all taken to the hospital with  
15 symptoms of poisoning.

16         The reason you see chloropicrin and metam  
17 sodium and not methyl bromide and Telone is because  
18 metam and chloropicrin are both irritant gases. It  
19 gets in your nose and you can be poisoned. Methyl  
20 bromide, you might feel like you're getting a flu.  
21 You might have a headache. You might just feel bad.  
22 You are getting something else. Telone, the same  
23 kind of things. Methyl iodide certainly falls under  
24 the category with methyl bromide and Telone. You're  
25 not going to know about poisoning.

1           Just to say we're very pleased with DPR's risk  
2 assessment for the most part. It is very  
3 comprehensive. It is thorough. It is well  
4 documented, and they found a lot of papers that we  
5 haven't found before. They have tried hard to use  
6 science-based decision making, based on research  
7 that has been done. We generally agree with a lot  
8 of DPR's analysis. We think there are some areas  
9 where it can be improved. And a lot the health  
10 studies -- I would say whoever looks -- having that  
11 real, raw data is going to be critical here. It's  
12 just some things you can't get out of reading, even  
13 DPR's assessment of the study.

14           The survival rates of the control groups were  
15 lower than that of the treated groups. That is kind  
16 of weird. And excess deaths at 60 ppm in rats  
17 occurred during months five and six of the study.  
18 Engineering corrections and changing cage placements  
19 stopped the mortality. Well, what is going on  
20 there? Is it a study you can trust at all or should  
21 it just been throw it out all together? Then it is  
22 not clear if all the animals were examined and all  
23 the tumors reported in the study. And there are  
24 number that died before termination. And in the DPR  
25 document it said two pituitary adenomas and

1 undetermined accounted for literally all deaths.

2           Were those two pituitary adenomas included in  
3 the table? I couldn't really tell. And I would say  
4 that whoever is looking at that data should look at  
5 it very closely.

6           I am not going to talk about the first two  
7 bullets. The third one, basically, the radio in the  
8 study showed that  $^{14}\text{C}$  ends up all over the body.  
9 What is going on there is a lot of potential for  
10 damage there and many ways that can be affecting  
11 things.

12           Here is some examples, and I won't -- since  
13 you have this slide, I won't go into too much.  
14 Basically, want you guys to think about cancer and  
15 potencies. Here are some comparison numbers.  
16 Methyl iodide kind of falls into the formaldehyde  
17 and propylene oxide and all those kinds of things,  
18 from the DPR risk assessment.

19           Inhalation studies. We're really concerned  
20 about the lack of parallelism between the studies  
21 used for the rats and the rabbits and humans. So  
22 you've got six hours a day, five days per week. And  
23 exposure nothing, exposure nothing. You've a long  
24 break over the weekend. During the rest phases you  
25 have chance to -- the animal has a chance to

1 replenish glutathione, exert iodide and start  
2 cellular repair. In contrast, here is the real  
3 data, some real data from Telone application. And  
4 you see that you get this spike that lasts for quite  
5 a long time. Actually about two and a half days.  
6 So almost three days. This is totally unrelated to  
7 this.

8         And then I think I am going to -- well, I will  
9 submit some other comments. I think there is one  
10 thing more important. You talked about that. You  
11 already talked about that.

12         The exposure assessment. I would recommend  
13 that whoever is looking closely at the exposure  
14 assessment, go read -- not just the summary of SAP  
15 concern in the document on Perfume. There is some  
16 really interesting things in the actual transcript.  
17 But basically there is a lot of uncertainty in this  
18 model and the SAP had concerns. The question is:  
19 Were those addressed in the current model? I can't  
20 tell from the data that I have access to. But if  
21 you guys do have access you can check this. And  
22 most importantly, the flux. Which flux profile do  
23 you use? There were a bunch of studies done. Were  
24 they done at 90 degrees in the Central Valley or  
25 were they done at, you know, 60 degrees on the

1 Central Coast? That will make a huge difference in  
2 the flux profile that will effect the result that  
3 you get out of your model. The panel flag that as a  
4 real issue.

5 Background issues. Applications has come up,  
6 too. More things, basically.

7 DR. BLANC: Clarify one thing about that.  
8 This is something that Dr. Hammond brought up.  
9 There is a bit of diversion, if I understood  
10 correctly, the flux model statistically attempts to  
11 put in the model different temperatures for how that  
12 would impact the actual measures that they have. As  
13 opposed to that, the occupational exposure data,  
14 which were done at certain conditions, is not put  
15 into a probabilistic model which tries to reestimate  
16 the observed occupational data. If it was not a 60  
17 degree day on the coast, but rather it was a 95  
18 degree day in Ventura. That is our understanding of  
19 at the moment. Are you trying to imply something  
20 different about the probabilistic --

21 DR. KEGLEY: It is a bad calculation  
22 method. If you look at what conditions that study  
23 was conducted under.

24 DR. BLANC: Doesn't the extrapolation then  
25 model into it what would our measured have been like

1 if it was a different meteorological condition?

2 DR. KEGLEY: That's the thing to check. I  
3 can't. I don't have access to the model. And is  
4 there input for temperature?

5 DR. BLANC: We were told yes, but there was  
6 not input for the temperature for the occupational  
7 exposure data.

8 DR. KEGLEY: I would think that it wasn't  
9 in there earlier.

10 DR. BLANC: We were not actually dealing  
11 with the Perfum. We are dealing with the actual  
12 model, the flux models that California used, which  
13 was not the Perfum, I don't think.

14 DR. FROINES: They didn't use Perfum.

15 DR. MCKONE: Those are just transport  
16 model. You have to back backwards for the flux.

17 DR. BLANC: I think your point about  
18 temperature may not be so relevant.

19 DR. KEGLEY: It is worth looking at,  
20 whoever is looking at that.

21 DR. HAMMOND: Along that line, I would  
22 point to your earlier slide that showed general  
23 consumption inhalation studies. You have the  
24 northwest and southeast. They look quite different  
25 in the first. And I don't know the temperatures

1 those recorded at. My question that popped into my  
2 mind: Why are the different? Is it because they  
3 were different temperatures that was happening?

4 DR. KEGLEY: The same field, but the wind  
5 was blowing from a different direction. This was  
6 not --

7 DR. HAMMOND: I thought Washington and  
8 Florida.

9 DR. KEGLEY: Condition with the wind  
10 direction.

11 So there's been an issue with enforcement and  
12 inversion conditions really do cause this stuff to  
13 concentrate in the lower levels of the atmosphere  
14 where people are. I am not sure ICSTC3 accounts  
15 well for calms during emergencies. And Cal Pup was  
16 put together to start addressing these kinds of  
17 issues. Air Mod does a better job of that. I would  
18 want some attention paid to that.

19 DR. FROINES: Susan, you have to --

20 DR. KEGLEY: Averaging is really important.  
21 There is an incident in there you can take a look  
22 at. I just added this set of slides, which you have  
23 in your things, just shows you the distribution of  
24 applications over the year. You can see that there  
25 are some area like Ventura that just stay dark the

1 whole time or pretty much. And you can see there is  
2 several months in this; that has already been shown  
3 to you.

4           There is a couple of issues that have happened  
5 in the real world, and that is when you -- DPR did  
6 calculations for 40 acres and a 400-foot buffer  
7 zone. They want a smaller buffer zone so they  
8 fumigant fewer acres at a time.

9           This 54 acre block in Los Angeles took two  
10 months to be fully fumigated. You can see the  
11 blocks they divided it up into, which means you have  
12 subchronic exposure issue going on instead of just  
13 an acute.

14           DR. FANNING: Sorry, does the panel have  
15 that supplied?

16           DR. KEGLEY: No. I added this one. Yes, I  
17 will I give you that one.

18           I just want to say be careful. There is a lot  
19 of averaging going on. And that is a list of some  
20 of the things being averaged. I wanted to give a  
21 very quick example. Here is an exposure assessment,  
22 Table 5, and they give you one number for each  
23 incidence for each location, one number for each  
24 distance. So 15 meters you would have concentration  
25 of 3.4 micrograms per liter per 24 hours. What

1 does that mean? That you get one concentration?  
2 There has to be some averaging going on. If you  
3 have --

4 I am using imaginary data here. It is pretty  
5 realistic. I just wanted to have this example.  
6 You've got a situation where you've got fumigation.  
7 Your concentration is really high to begin with and  
8 it drops over time. You get more in the downwind  
9 direction, and downwind direction here is mostly  
10 east and north. Let's take the downwind direction.  
11 And the maximum concentration is 25 parts per  
12 million. Again, if you average that over 24 hours,  
13 you get 7.36. You are not averaging the spike that  
14 might be several hours long. That may make a pretty  
15 big difference in terms of glutathione depletion or  
16 iodide exposure.

17 Now one thing that has been done in the past  
18 and may be done here - I can't tell because the data  
19 aren't there - is that all of the north, south, east  
20 and west sampling are averaged together for each  
21 point on the curve. So it is a whole field  
22 concentration. And so you are not seeing that peak.  
23 So this has been done in Washington data looking at  
24 methane sodium fumigations. What does that mean?  
25 That means if you average, the average, you got our

1 levels way down by a factor of seven and a half or  
2 so different. I can't tell. I can't evaluate. I  
3 don't have the raw data. You guys need to take a  
4 look at the averaging issue.

5 DR. HATTIS: What this means, you have more  
6 or less linear dose response relationship. The  
7 averaging doesn't hurt you a whole lot. But if you  
8 have a highly nonlinear dose relationship, then you  
9 care -- and a short time of action, then you care  
10 very much about these fluctuations. So it really  
11 interacts strongly with what you think the mode of  
12 action is in the right.

13 DR. KEGLEY: The 25 parts per million was  
14 measured three to six hours after.

15 DR. HATTIS: If you think glutathione  
16 depletion can be reversed over a period of hours, it  
17 is important than that interacts with the -- you are  
18 thinking that is a long-term accumulating risk,  
19 genetic risk, you are talking about. It is less  
20 important, except it synergizes.

21 DR. KEGLEY: I won't take the time to do  
22 it, but I have the federal registrant, the  
23 guidelines when EPA should require the DNT test.  
24 Everyone have those guidelines?

25 DR. FROINES: I think we can say that that

1 was a lot of new data, new information that you  
2 provided. That is, I think, quite useful.

3 So thank you.

4 DR. KEGLEY: Thank you.

5 DR. FROINES: We are going to take a break  
6 for lunch and decide our future.

7 DR. BLANC: Can you give the audience some  
8 sense of when we are going to reconvene?

9 DR. FROINES: I would say 1:40. It is  
10 going to take us time to get upstairs and  
11 everything.

12 (Luncheon break taken.)

13 ---oOo---

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1 AFTERNOON SESSION

2 ---oOo---

3 DR. FROINES: We are ready to go. We have  
4 an awful lot of people who want to speak. And at  
5 the outset I want to apologize for the limited time  
6 you will have to speak and whether we even make it  
7 through, and so we will just have to see.

8 What I would like to do now is to announce the  
9 names of the ten people who will line up here, I  
10 guess. And then we will call them and then what?  
11 And then they will have a three minutes a piece and  
12 we will give one minute warning and we will go to  
13 the next ten.

14 UNIDENTIFIED AUDIENCE MEMBER: Are you  
15 going to have additional time for translation?

16 MS. KOBYLEWSKI: It will be the same time  
17 translation I am told. So they will be translating  
18 as the person speaks.

19 UNIDENTIFIED AUDIENCE MEMBER: The time it  
20 takes for a translation, the person will be speaking  
21 in Spanish and the translator will speak into the  
22 microphone, translate that in English and the person  
23 standing next to them.

24 MS. KOBYLEWSKI: I will announce the first  
25 ten people: Marilyn Lynds, Kathryn Gilje, Ben

1 Ebbink, Dr. Husein Ajwa, Amber Wise, Caroline Cox,  
2 Dr. James Sims, Eric Johansen --

3 DR. FROINES: I want to emphasize one  
4 point. There are obvious time limitations. Every  
5 word that is said is going to be on the transcript.  
6 And we will go through the transcript and read every  
7 single person's presentation. So that no one need  
8 feel that they are not -- their voices are not being  
9 heard. I will make it a personal commitment to all  
10 of you that these presentations will be heard in  
11 their entirety.

12 MS. KOBYLEWSKI: Two more. Shirley  
13 Batchman and Jim Cochran.

14 The first person is Marilyn Lynds.

15 MS. LYNDS: My name is Marilyn Lynds. I am  
16 representing Pesticide Watch, and I'm a spokesperson  
17 for Moss Landing Heights community.

18 THE COURT REPORTER: You have to speak into  
19 the mike.

20 MS. LYNDS: I read your mission statement,  
21 which is to protect human health and the environment  
22 by regulating pesticide sales and use and by  
23 proffering reduced risk management. I was very  
24 gratified to hear. I do not see a way that you  
25 could allow methyl iodide to be registered for use

1 as a pesticide in California with that as your  
2 mission statement.

3           You have all heard about all the suspected and  
4 known dangers of the pesticide methyl iodide this  
5 morning. Like the other three fumigant pesticides,  
6 methyl iodide can cause neurological damage, fetal  
7 insult, including miscarriage, thyroid cancer. The  
8 list goes on. Methyl iodide is in every way just as  
9 or more dangerous than its predecessors to human and  
10 animal life. And unlike the others, it is water  
11 soluble. Putting waterways, acquirers, wells at  
12 risk for contamination.

13           I cannot imagine a scenario where a sane  
14 person would agree that growing nematodes  
15 prestrawberries, a luxury crop takes precedence over  
16 healthy and safe drinking water.

17           Spokespeople from pesticide companies and  
18 certain agencies make assurances to the public that  
19 every precaution will be taken and that there is no  
20 real risk to workers and neighbors. My community  
21 seeks such assurances in the fall of 2007 when the  
22 farmer across the street from our homes used  
23 chloropicrin and methyl bromide on land that had  
24 never been fumigated in its 80-odd years in  
25 agricultural production.

1           We were contaminated time after time. Tarps  
2 blew off. No one was notified. The application was  
3 violated by use more acreage than was allowed. They  
4 applied the fumigants during -- in convergent  
5 layers. And the truth is even though those did  
6 happen, fumigants are going to drift. It is their  
7 nature. Drift may not occur during ever single  
8 application of the fumigant, but inevitably there  
9 will be drift. The only laws fumigant are the laws  
10 of science and nature. When a fumigants are used  
11 next to homes, schools and work places, people are  
12 left with the awful choice of stripling for cover up  
13 to weeks at a time on 48 hours notice over the  
14 course of weeks or months, or take the chance of  
15 becoming very sick.

16           What's more, like Moss Landing, many places  
17 were methyl iodide will be used for irrigation near  
18 sensitive waterways. Moss Landing is next to the  
19 Salinas River, Coho Slough, Elkhorn Slough as well  
20 as being at the heart of Monterey Bay Sanctuary.  
21 The farm in question is 100 feet from a snowy plover  
22 nesting site and home to many endangered species.

23           Now, when I learned that Moss Landing has been  
24 designated a test site for methyl iodide, we feel  
25 that we have suffered enough. Nobody from my

1 community is anti farm or patently anti-pesticide.  
2 Our subdivision was created over 50 years ago by the  
3 original farmer owners.

4 MS. KOBYLEWSKI: Five seconds.

5 MS. LYNDS: It is simply a matter of  
6 fairness. Pesticide companies and farm owners make  
7 a profit while communities and wildlife pay the  
8 price with their health, their environment and value  
9 of their homes with no benefit. Please do not  
10 register methyl iodide.

11 And I have some letter and petitions that I  
12 would like to present to you.

13 DR. FROINES: I want to make sure that  
14 everybody in this room is clear on what is actually  
15 happening here today. The people who are sitting  
16 around this table do not have any relationship  
17 whatsoever with the department of pesticide  
18 regulation. We are an independent group of  
19 scientists from around the country who are reviewing  
20 the risk assessment prepared by the Department of  
21 Pesticide Regulation. They will determine the  
22 future of methyl iodide in California. And what we  
23 are doing is making critical scientific comments on  
24 the risk assessment that has been developed.

25 So that we are not part of the decision making

1 process. That will be the role of the leadership of  
2 the Department of Pesticide Regulation. And so what  
3 you've got here is a group of scientists who are  
4 going to do the best we can to address the issues  
5 that are relevant to the methyl iodide decision.

6 MS. LYNDS: Should I give them to somebody  
7 else?

8 DR. FROINES: She is with the Department of  
9 Pesticide Regulation. She may not want everything  
10 that people are going to give her, but we'll start  
11 out that way.

12 MS. GILJE: Kathryn Gilje on behalf of Ted  
13 Schettler. Chairman Froines and distinguished  
14 members of the panel, I would like to begin by  
15 offering my sincere and deep gratitude for your  
16 willingness to invest so much time in reviewing the  
17 science behind methyl iodide and your commitment to  
18 review process that is vigorous, transparent and  
19 marked with integrity.

20 The recommendations that you make as part of  
21 this panel will significantly influence how the  
22 State of California moves forward with its decision  
23 on methyl iodide. As such, as residents of the  
24 State of California we place a portion of our future  
25 in your hands. I thank you for understanding the

1 weight of that responsibility and for being willing  
2 to bear it.

3 My name is Kathryn Gilje, and I am the  
4 director of Pesticide Action Network. I'm here  
5 today at the urging of thousands of PAN supporters  
6 who are very concern about the possibility that  
7 California may register a new chemical that carries  
8 with it known and unknown health and environmental  
9 threats. We are concerned for the health of  
10 California's children, farm workers and water in  
11 particular along with the health of those living in  
12 the communities where fumigation may occur each  
13 season.

14 In my brief time today with the members of the  
15 panel, I want to offer you some material from one of  
16 the scientists on our board of directors who is  
17 particularly concerned about the potential  
18 registration in California, Dr. Ted Schettler.  
19 Dr. Schettler has a medical degree from Case Western  
20 Reserve University and a Master's in public health  
21 from Harvard University. Dr. Schettler is  
22 submitting his own letter to DPR as part of this  
23 process and this letter covers many more points than  
24 I can cover in a brief time today.

25 I will read a brief statement related to

1 developmental neurotoxicity which is a big concern  
2 of his. I quote from his letter: The mechanisms by  
3 which methyl iodide exerts its neurotoxic effects  
4 are not completely understood. However, it is clear  
5 that glutathione depletion is an important  
6 contributor to the casual pathways leading to  
7 neurotoxicity. Several studies conclude that  
8 glutathione depletion alone leads to neurotoxicity.  
9 In these studies depletion of glutathione prior to  
10 methyl iodide exposure enhance neuro cell damage and  
11 supplication of glutathione prior to  
12 [unintelligible]. The authors conclude that  
13 oxidative stress and associated mitochondrial damage  
14 are critical components of neurotoxicity -- of  
15 methyl iodide.

16         With the above in mind it is worth noting that  
17 fetuses and infants have lower levels of glutathione  
18 in their tissue then young adults. Glutathione  
19 levels also decline in older people, and its general  
20 antioxidant is diminished in the very young and the  
21 aged. Lower baseline levels of glutathione would be  
22 anticipated to increase susceptibility to the  
23 neurotoxicant like methyl iodide whose mode of  
24 action depends at least on part on glutathione  
25 depletion.

1           For that reason alone, it can be predicted  
2 that the developing brain is more vulnerable to  
3 iodomethane neurotoxicity than the fully developed  
4 adult brain. Beyond that, however, impacts of  
5 oxidative stress differ in the developing brain.  
6 Because the unique developmental effect chronic  
7 heart disease in adults. Moreover the results of  
8 the impaired developed processes in the brain are  
9 particularly long lasting and often irreversible.

10           In closing, I would like to suggest that the  
11 California registration of methyl iodide offers us  
12 the rare opportunity to do things right from the  
13 beginning. To fully consider this science, wait  
14 until we are sure we won't be harming the health of  
15 people or the environment with the registration of  
16 one more pesticide in the state that already uses  
17 the most pesticide in the nation.

18           MS. KOBYLEWSKI: Thank you.

19           MS GILJE: Thank you very much for your  
20 time.

21           MS. KOBYLEWSKI: Ben Ebbink.

22           MR. EBBINK: Good afternoon. My name is  
23 Ben Ebbink, and I am a chief consultant for the  
24 Assembly Committee Labor and Employment. I am here  
25 today on behalf of Assemblyman Bill Monning, the

1 chair of the committee.

2 As some of you may know, on August 13th the  
3 Labor Committee held an informational hearing on the  
4 very important subject of methyl iodide and the  
5 potential impacts to worker health and safety. As  
6 Mr. Monning stated at the time, the informational  
7 hearing was not an effort to undercut or supplant  
8 this external peer review process. Rather it was  
9 hoped that our hearing added to this process and  
10 dialogue.

11 At the Labor Committee hearing testimony was  
12 presented about the toxic nature of methyl iodide  
13 and some of the potential health risks to workers  
14 and local communities. This testimony raised  
15 concerns for Mr. Monning and the other members of  
16 the committee. However, they acknowledged and  
17 respect that is precisely for this reason that this  
18 external peer review panel was convened. You are  
19 the scientific experts in this area and we respect  
20 and refer to your analysis and judgment.

21 However, one point that was made very clear  
22 during the hearing was that even the best  
23 precautions, safeguards and label instructions do  
24 not always translate to the appropriate safety  
25 levels in the field. As we have learned with the

1 application patterns of other pesticide fumigants,  
2 the reality is instructions are not always followed,  
3 protective gear is not always provided or used  
4 properly. Unfortunately, mistakes in every day  
5 occur to the detriment, to the workers and to the  
6 communities. It is Mr. Monning's hope that as you  
7 evaluate methyl iodide you balance those issues  
8 considering the various health risks based on the  
9 analysis of the real practice in the field. And I  
10 think Dr. Froines state that there is the real world  
11 and there is the world we would like to live in.

12 In order to provide you as much information  
13 from our informational hearing, I am submitting  
14 copies of our post-hearing report.

15 DR. FROINES: Great.

16 MR. EBBINK: The document summarizes  
17 testimony that was presented at our hearing and  
18 includes copies of documents that were submitted at  
19 the hearing by the various witnesses. As mentioned  
20 earlier, it was Mr. Monning's hope that this  
21 information will supplement the information you are  
22 receiving today and will assist you in this very  
23 important process.

24 Thank you for your time.

25 DR. FROINES: Thank you very much. Thank

1 you for your materials.

2 MS. KOBYLEWSKI: Dr. Husein Ajwa.

3 DR. AJWA: Thank you. My name is Husein  
4 Ajwa. I am on the faculty of U. C. Davis. My house  
5 is in Salinas, California. I am a soil chemist with  
6 over 20 years experience with fumigants and  
7 pesticides. And I have been involved in methyl  
8 bromide and methyl bromide alternatives for the last  
9 13 years. I have helped develop research protocols,  
10 conducted lab and field research on fumigants and  
11 pesticides, and I have served as a scientific  
12 advisor for DPR on various efficacy and technology  
13 regimes, on a water safety buffer zone and DOC and  
14 flux from fumigant and field. I have been working  
15 on iodomethane for lots of -- many years. I have  
16 done extensive research and efficacy on worker's  
17 exposure and basically on efficacy and the use of  
18 methyl iodide as a crop replacement for methyl  
19 bromide. The effective tool to our growers,  
20 iodomethane can be a crop replacement for ozone  
21 depleter as with methyl bromide. Also, iodomethane  
22 is used at much lower rate than other fumigants.  
23 That will leave us get some more toxic fumigants out  
24 of use.

25 Iodomethane is a non-ozone depleter, patented

1 by the Regents of the University of California. So  
2 it is our research product, basically, and we don't  
3 need as much water to do the job for soil  
4 disinfection as much as other fumigants on  
5 pesticide.

6 MS. KOBYLEWSKI: One minute.

7 DR. AJWA: Our research demonstrates and  
8 project showed clearly that iodomethane is safer and  
9 more environmentally safe fumigant than current  
10 fumigants, registered fumigants. Without  
11 iodomethane we will have to continue using other  
12 fumigants at higher rates.

13 I urge you to take a look at the critical use  
14 numerations that we submit every year to keep using  
15 methyl bromide because we do not have a viable  
16 alternative like methyl iodomethane.

17 Thank you very much.

18 DR. FROINES: Thank you.

19 DR. BLANC: Could I ask a point of process.  
20 That speakers who speak disclose potential conflicts  
21 of interest that they may have in terms of support  
22 or financial interest in the methyl iodomethane  
23 product. The speaker very clearly disclosed that  
24 the University of California, which has the patent  
25 interest, for example.

1 DR. FROINES: Let me ask the panel. How do  
2 you feel about Paul's recommendation? What is your  
3 view?

4 DR. SLOTKIN: We have to do that when we  
5 speak.

6 DR. HAMMOND: I am not sure we should. As  
7 a panel we should have done that. I think it is a  
8 good idea.

9 DR. HATTIS: I think more or less people  
10 have a point of view and those are more or less  
11 clear, I think. Sometimes they are.

12 DR. FROINES: I'm not hearing --

13 DR. BLANC: I think you're hearing a  
14 consensus that we should forego and leave it to the  
15 people's discretion should they wish to say  
16 something.

17 DR. FROINES: Is that because that is not  
18 what Ted said?

19 DR. SLOTKIN: I withdraw my recommendation.

20 MS. KOBYLEWSKI: Amber Wise.

21 MS. WISE: Hi. I would like to thank you  
22 guys for convening an external review panel to  
23 discuss the findings of the California Department of  
24 Pesticide Regulation's assessment of methyl iodide.

25 My name is Amber Wise. I have a Ph.D. in

1 chemistry from U.C. Berkeley and a postdoctoral  
2 fellow at the program for reproductive health and  
3 the environment at U.C. San Francisco. And I am  
4 here today to present -- well, I was going to  
5 present the reproductive development in  
6 neurotoxicity concerns. Due to time constraints, I  
7 think you have heard enough of health concerns. But  
8 I would also like to speak to you a bit as a chemist  
9 today.

10           Most chemists are unfamiliar with pesticide  
11 practices. And when I tell them that we spray a  
12 hundred pounds of methyl bromide on a soil at any  
13 one time and millions of pounds per year, they are  
14 absolutely shocked. When I tell them that methyl  
15 iodide is being proposed as the replacement for  
16 this, they are stunned. Since every chemists uses  
17 methyl iodide in their reactions because it is an  
18 extremely reactive alkylating agent, we use it only  
19 under extremely protective conditions. And I think  
20 Susan showed us why that someone only uses a syringe  
21 and sealed container to prevent exposure, because we  
22 know it's an efficient DNA methylator that will  
23 increase our likelihood of cancer. We also only use  
24 these precautions when we're working with very small  
25 amounts, of few drops, a few milliliters at a time.

1 USEPA has determined that it is safe to spray  
2 up to 175 pounds per acre of this compound. One of  
3 the toxicity endpoints of greatest concern is CDPR;  
4 this is the perturbation of thyroid hormone in the  
5 resultant fetuses. At UCSF a major focus of our  
6 group's research is how the environmental factors  
7 may affect thyroid hormone disruption and subsequent  
8 reproductive health developmental problems.

9 I am going to skip over most of the health  
10 effects that I had. I think you heard a lot of it  
11 today. What I would like to do is point out few of  
12 the CDPR's assessment points that I find important  
13 and that's what you're hear to assess. CDPR more  
14 appropriately uses directional wind patterns for any  
15 of their wind/air calculations and found that --

16 MS. KOBYLEWSKI: One minute.

17 MS. WISE: -- the estimated  
18 [unintelligible] for bystanders were 375 times  
19 higher than the determined safety limit per  
20 bystanders. They also recommended actually tenfold  
21 uncertainty factor, which we agree with.

22 I think I would like to bring you up the point  
23 of water, potential groundwater contamination.  
24 There is a reference paper of people who were in  
25 early studies of transport or methyl iodide and

1 methyl bromide. Again, in 1997 they found methyl  
2 iodide is more persistent in soil than methyl  
3 bromide and, therefore, they were concerned that  
4 this might lead to more groundwater contamination  
5 for methyl iodide.

6 The other two points that I don't think has  
7 gotten quite enough coverage today is -- I guess one  
8 point. There's been no discussion or safety factor  
9 considered for background chemical exposures. I  
10 know there is not a standard way to do this  
11 assessment.

12 MS. KOBYLEWSKI: Thank you.

13 MS. WISE: The CDPR is on an approved risk  
14 assessment, but it is still incomplete. I believe  
15 that even if we find more data, there is not going  
16 to be some number that tells us this is going to be  
17 a safe chemical.

18 Thank you.

19 MS. KOBYLEWSKI: Caroline Cox.

20 MS. COX: I am speaking this afternoon on  
21 behalf of the Center for Environmental Health. We  
22 are a nonprofit organization that has a lot of  
23 experience over the last decade of enforcing  
24 Californian's Safe Drinking Water and Toxics Enforce  
25 Act. That is the law that most people call Prop 65.

1 And I'm sure that you all are familiar with Prop 65  
2 warnings and have seen them around this state.

3 Many people are not familiar with the drinking  
4 water protection part of the law, which are actually  
5 strongly worded and/or pretty much absolute  
6 prohibition against discharging into sources of  
7 drinking water. Proposition 65 lists chemicals  
8 which would include methyl iodide. The risk  
9 assessment minimized groundwater contamination  
10 problems with methyl iodide, but I would really urge  
11 you to look clearly at the studies which indicate  
12 that there could be a serious problem with this.  
13 And I also urge you to recommend against the  
14 registration of methyl iodide, if its use seems  
15 likely to contradict or violate existing state laws,  
16 specifically the safe drinking water enforcement  
17 portion.

18 Thank you.

19 DR. FROINES: Thank you.

20 MS. KOBYLEWSKI: Dr. James Sims.

21 DR. SIMS: Heard my name before. I am not  
22 the first name on the patent of methyl iodide. My  
23 idea. My baby. I have been dealing with it since  
24 1993. I hope you guys would do the job you're  
25 suppose to. I assume that you will. I don't have

1 any other kind of thing.

2           The reason I came up here was just to show  
3 that I support this. I don't support -- I'm an  
4 organic chemist. I've done a lot of work with  
5 marine algae, showing that they make halogenated  
6 compounds. The ocean is a big place to make  
7 halogenated compounds in metabolism. Whether that  
8 plays any role in this or not.

9           But the synthetic part of my career I used  
10 methyl iodide since I was a sophomore in organic  
11 chemistry. We weighed it out in the open lab. We  
12 used pro pipettes to pipette into reaction flasks.  
13 The regard reagent which supposedly for a chemist --  
14 has anybody here ever used methyl iodide?

15           You use all this protection --

16           DR. FROINES: I found methyl iodide  
17 terrified to use, so I was always extremely careful  
18 and I always worried whether my hood had proper flow  
19 rates. So, yes, I have.

20           DR. SIMS: I have used it out in the open  
21 lab. Distilled it because it constantly degrades  
22 from UV exposure. It will turn dark and be  
23 contaminated with iodine. You to have redistill it  
24 every time you use it. Anyway, the reason I brought  
25 out methyl iodide or had the idea was that the ozone

1 depletion issue with methyl bromide. Methyl bromide  
2 was going away. Dupont didn't save it, and the  
3 California Strawberry Commission didn't save methyl  
4 bromide. They each thought they would.

5       To me it's, beside the ozone factor, it is a  
6 low boiling liquid which you can put in a bottle and  
7 you know where it is. When you're working with  
8 methyl bromide, it is a gas and you never really do  
9 know where it is because it doesn't have a smell.  
10 And it could be used in the same equipment which  
11 makes it easy for administering and use it, methyl  
12 iodide that is. And there is a short half life in  
13 water. Basically, it degrades in 20 to 40 days in  
14 water in the dark. In the light methyl iodide  
15 disappears almost immediately. In steam and exposed  
16 groundwater, it is going to disappear very quickly  
17 and there is data to back that up. And as you all  
18 know, it is a human interest.

19       There are studies by the public health service  
20 that I have read about that says the amount of  
21 iodine in our population is going down, down, down,  
22 and we're having public health problems because of  
23 that. Methyl iodide is part of the methyl iodine  
24 cycle in the world. It is generated in the ocean.  
25 Comes out and drops on land.

1 MS. KOBYLEWSKI: One minute.

2 DR. SIMS: I don't have any other  
3 particular thing, any points to make other than I  
4 think you guys will do the job you need to do, and  
5 hopefully methyl iodide will be registered.

6 Thank you.

7 DR. FROINES: I realize I am out of line  
8 here, but we have a question that we can't get an  
9 answer to. And even though you're not a  
10 toxicologist, you're a chemist and I believe all  
11 chemists are smart.

12 DR. SIMS: I agree with that.

13 DR. FROINES: You may know the answer. We  
14 know how methyl iodide kills nematodes. We think it  
15 is an interesting question because there is  
16 something mechanistic elements in this process. So  
17 if you have any insights, please share it with us.

18 DR. SIMS: It's an alkylating agent and it  
19 probably gums up some of the processes. I don't  
20 know any better than that. I would assume it is  
21 working as an alkaloid. There is some talk that I  
22 read in the literature that it might get metabolized  
23 in formaldehyde and formaldehyde may be a reactive  
24 agent. The talk about the carbon 14 being found in  
25 other places, other than standard places. I think I

1 read somewhere that that has been found in  
2 carbohydrates, to be able to put into carbohydrates.  
3 The only place it acts with a methylate,  
4 metabolized there.

5 DR. FROINES: Thank you very much.

6 DR. FROINES: Erik Johansen.

7 MR. JOHANSEN: Thank you. I am here this  
8 afternoon as a representative of Washington State  
9 Department of Agricultural. I realize that you have  
10 been tasked by the California Department of  
11 Pesticide Regulation, but Washington is the other  
12 state that is still looking at iodomethane. And the  
13 information that we got so far from EPA, especially  
14 from California Department of Pesticide Regulation,  
15 has been extremely helpful. I appreciate the work  
16 you have been doing the last several days and if  
17 there is anything our review could be of use to you,  
18 please let us know. Be happy to help.

19 We will probably be meeting to discuss this  
20 further this October, and I don't know when we're  
21 going to make a decision. We definitely will be in  
22 touch with Cal DPR.

23 DR. FROINES: Will we be able to get copy  
24 of the transcript of your meeting so we can see the  
25 nature of discussions?

1 MS. JOHANSEN: There is not going to be a  
2 hearing, per se. Right now all we are planning on  
3 doing is meeting with our department of health and  
4 our own internal staff to discuss their concerns  
5 about iodomethane. And I think you have seen the  
6 earlier draft that was sent to EPA, and EPA  
7 responded to health is getting ready to send us a  
8 flow-up response. Once we have that we have to  
9 decide what we are going to do. We are going to ask  
10 for more information from the registrant or make a  
11 decision.

12 DR. FROINES: Anything that you have in  
13 writing that we might benefit from, please send it  
14 along, and we will take advantage of it.

15 MR. JOHANSEN: Be happy to.

16 MS. KOBYLEWSKI: Shirley Batchman and Jim  
17 Cochran.

18 MR. COCHRAN: I can't believe I see smiles  
19 on everybody's faces. Everybody is paying attention  
20 after two days of this is pretty remarkable. Jim  
21 Cochran. I am a strawberry farmer among other  
22 things. I farm around 200 acres and 20 acres of  
23 strawberries. I've been in business for 26 years.  
24 And I farm organically now. I started out using  
25 methyl bromide and other chemicals for the first

1 couple of years and decided that I didn't want to  
2 expose myself or my place to the various chemicals  
3 that we had in our arsenal, and so I switched over  
4 to the organic methods. And in '89 I was co-PI on a  
5 study that was looking at use of the various  
6 chemicals, principally methyl bromide but others as  
7 well in a randomized study that look at organic  
8 methods and chemical methods side by side.

9           The interesting thing about this study was  
10 that in not only looked at the science, but it  
11 looked at the economics as well. And what we found  
12 was that the organic method was economically viable.  
13 So as a consequence, at that time I was a hundred  
14 percent of the organic strawberries grown in the  
15 state. Now I am 1 percent of the organic  
16 strawberries in California. There are, I think,  
17 about 1,700 acres grown without any sort soil  
18 fumigants or synthetic pesticides, and people are  
19 making money and doing quite well.

20           I think the economic argument needs to be  
21 insinuated in here to some degree. It is not  
22 actually on your mandate, but the fact that it is  
23 possible to farm successfully --

24           MS. KOBYLEWSKI: One minute.

25           MR. COCHRAN: -- I think it is without

1 using chemicals is really an important one. I think  
2 that what we have here is an opportunity to, if we  
3 dial back the clock to '72 or '73 or '74, and  
4 Detroit was fighting all sorts of things like  
5 seatbelts and air bags and mileage mandates and all  
6 sorts of things that they didn't want, if they had  
7 listened to the scientists and the people who were  
8 doing the work at the time, they might have avoided  
9 being at the situation now where Toyota came and ate  
10 their lunch. And they now need to be bailed out by  
11 the U.S. government. And so I think your role here  
12 is really important for the future of agriculture in  
13 general and not just specifically about this  
14 particular chemical. That is the real hope for the  
15 future.

16 Thank you.

17 DR. FROINES: I can't get a recommendation  
18 for seatbelts out of this committee.

19 MS. KOBYLEWSKI: The next people are: Dave  
20 Cox, Ryan Jacobsen, Chris Valadez, Gina Solomon,  
21 David Chatfield, Elizabeth Martin-Craig, Barbara  
22 LaFave, Robert Dolezal, Manuel C. Cunha, James  
23 Randall.

24 MR. VALADEZ: I am not Dave Cox, nor am I  
25 Ryan Jacobsen.

1 MS. KOBYLEWSKI: Are Dave Cox or Ryan  
2 Jacobsen here?

3 Go ahead.

4 MR. VALADEZ: I am Chris Valadez  
5 representing the California Grape and Tree Fruit  
6 League. We are a grower-shipper organization  
7 statewide, but most of our effective membership,  
8 particularly on the issue we are discussing today,  
9 are on the eastern side of San Joaquin Valley. And  
10 I bring that up and want to draw importance to that  
11 bring up the economic discussion is some of the  
12 economic difficulties that our tree fruit growers  
13 have faced and packing houses have faced, they're  
14 closing down. We've had a few close this past year  
15 and some before that. It is likely due to capital  
16 issues and some other issues that we are going to  
17 have some future closers. Therefore, what happens,  
18 orchard removal, pulling of trees. You're going to  
19 have ground there that I think in order to -- for  
20 future reinvestments, so there is going to be a need  
21 for continuance of soil fumigants.

22 But the phase out of methyl bromide, many of  
23 our folks are going to continue to look at what are  
24 some of the available products out there. Looking  
25 to one such as methyl iodide. I think the economics

1 and the reality for our folks we're talking about  
2 turning back the clock somewhat, so to speak, I  
3 think in some situations that definitely is a  
4 valuable discussion. However, in our particular  
5 example we see we have food needs, we have food  
6 costs and there is some service that we produce --  
7 you have a food reality and a food demand based on  
8 price. You don't have everybody really willing to  
9 pay six, seven, \$8.00 a pound for peaches. They  
10 want lower prices.

11 How do he continue to produce food at the  
12 price consumers want and maintain economic  
13 viability? So I think in the end when we talk about  
14 available arsenals and/or available toolbox, I think  
15 we look to maintain our economic viability and at  
16 look what is available in that toolbox. We look and  
17 we see a product such as methyl iodide and we look  
18 at some of the data that comes across come of which  
19 are analyzing in your process, used in 47 states,  
20 approved at the federal level. We look at the --

21 MS. KOBYLEWSKI: One minute.

22 MR. VALADEZ: -- issue of maintaining or  
23 trying to going through this competitive  
24 disadvantage of other farmers and growers that are  
25 using products such as methyl bromide or other

1 products that we don't have in our toolbox. Are we  
2 going to continue to compete on a playing field that  
3 is becoming increasingly uneven for us. I don't  
4 think that California agriculture has a goal from  
5 some of which our state, excluding with our state  
6 Department of Agriculture and with our legislative  
7 folks and our governor -- California agriculture is  
8 here to say. Our workers are here to stay. I think  
9 in order to continue to provide a benefit to the  
10 economic activity here in this state for our  
11 workers, I think we need to have that methyl iodide  
12 or other products available in our toolbox, and I  
13 ask for your consideration of that today.

14 Thank you.

15 MS. KOBYLEWSKI: Dr. Gina Solomon.

16 DR. SOLOMON: Dr. Froines, Members of the  
17 Panel, thank you for your patience with all of the  
18 presentations. I am Gina Solomon. I am a senior  
19 scientist with the Natural Resources Defense  
20 council, and assistant clinical professor of  
21 medicine at UCSF.

22 Back in 2006, NRDC took a position on methyl  
23 iodide. At that time it was in the context of the  
24 USEPA registration decision. We severely criticized  
25 many of the -- much of the scientific basis of EPA's

1 registration, but as a policy matter did not oppose  
2 the conditional time-limited registration of methyl  
3 iodide because it was seen as an important  
4 replacement for methyl bromide.

5           In the intervening years, a fair amount of  
6 additional information has come forward, and in the  
7 context of the California current decision and risk  
8 assessment, we went back and took a very careful  
9 look at the science and the new data and also the  
10 new analysis, and came to the conclusion that, in  
11 fact, methyl iodide was considerably significantly  
12 more toxic than methyl bromide, and in addition  
13 poses a serious threat to groundwater sources and  
14 drinking water, and as such have changed our  
15 position to oppose the registration of methyl  
16 iodide.

17           And I will be submitting fairly extensive  
18 technical comments on paper to DPR. And I was very,  
19 very happy to hear the discussions, because it  
20 really seems like this panel have hit on pretty much  
21 all of the issues that I had with the DPR risk  
22 assessment. Very glad to hear discussion about  
23 talking benchmark dose on approach, of using that  
24 tenfold uncertainty factor --

25           MS. KOBYLEWSKI: One minute.

1 DR. SOLOMON: -- to protect kids across the  
2 board. And this very serious concerns about the  
3 developmental toxicity levels below the so-called  
4 NOEL of 2ppm, which already I agree should no be  
5 considered NOEL. These and many name other issues  
6 are very important and things I hope that DPR does  
7 incorporate into the next draft.

8 Thank you all for your hard work on this  
9 issue.

10 DR. FROINES: Thank you.

11 MS. KOBYLEWSKI: David Chatfield.

12 MR. CHATFIELD: Good afternoon. My name is  
13 David Chatfield and I'm the executive director of  
14 California for Pesticide Reform.

15 Just a coalition in California of over 180  
16 organizations. I don't think there has been a  
17 single issue we have dealt with that has united our  
18 coalition quite like methyl iodide. I think it's  
19 safe to say I speak for the entire coalition of  
20 organizations that we would oppose the registration  
21 of methyl iodide.

22 We couldn't bring everybody here. In fact, we  
23 couldn't bring very many people who are affected by  
24 methyl iodide. You will be hearing from a number of  
25 those people this afternoon. And there are hundreds

1 more like them in Bakersfield and Ventura, Santa  
2 Barbara who would have come here if they could. To  
3 tell you about how they believe methyl iodide will  
4 affect their lives. I was thinking of the problem  
5 you've got as a scientific panel. So appreciate the  
6 fact that your willingness to listen to the public,  
7 which is not going to directly talk about, in most  
8 cases, science. I'm saying: How do you deal with  
9 that problem as a scientific panel? It is seems to  
10 me that you need -- the reason is that you are  
11 listening to these folks is because you need no  
12 figure out whether the science you're reviewing and  
13 the science package that says this is okay to use  
14 adequately addresses the fears of the folks who will  
15 be directly affected by that. Does it adequately  
16 address those problems which are fears of long-term  
17 and short-term health. That is the connection.

18         So I urge you very much to listen to this  
19 testimony in that context as it comes in. 'Cause it  
20 won't be entirely scientific. That is for sure.  
21 But it will be about human experience. And your job  
22 is to relate science to human experience. I believe  
23 that at some points there has got to be a time when  
24 presumed economic need -- we haven't heard very much  
25 about California agriculture changes all the time.

1 I don't know, I call it presumed economic need.  
2 There must come a time sometime when that is  
3 outweighed by the dangers to health of pesticides.  
4 And I believe this is such a moment. And I'm very  
5 glad to hear you're looking very, very carefully at  
6 the science of methyl iodide. And I do urge you to  
7 think about that as you receive the testimony of the  
8 people come after me.

9 DR. SLOTKIN: This morning we heard from  
10 people from Arysta that there were no reported  
11 incidence involving methyl iodide. And yet you're  
12 telling me that you got people who are going to talk  
13 about this.

14 MR. CHATFIELD: People who -- I don't have  
15 people. They have themselves.

16 DR. SLOTKIN: I would like to ask you: Are  
17 you aware of any people reporting these exposures to  
18 --

19 MR. CHATFIELD: The folks you are going to  
20 here from today are people who work in California  
21 where they haven't had that experience yet. In many  
22 case they have had the experience of working with  
23 methyl bromide and other. They're afraid.

24 DR. SLOTKIN: I wanted to get this clear.  
25 It sounded to me as though you had reports of people

1 with methyl iodide exposure.

2 MR. CHATFIELD: You have to go to Florida.

3 DR. FROINES: There is no use whatsoever of  
4 methyl iodide in California in agriculture. Doesn't  
5 mean there aren't chemistry labs and other places,  
6 but in term of agriculture pesticides there are none  
7 at this point.

8 DR. BLANC: Except for experimental fields.

9 DR. FROINES: Good point.

10 MR. CHATFIELD: I reckon the jury is out on  
11 that.

12 Thank you very much. Thanks for all this  
13 time.

14 MS. KOBYLEWSKI: Elizabeth Martin-Craig.

15 MS. MARTIN-CRAIG: My name is Elizabeth  
16 Matin-Craig. I am the northern California community  
17 organizer for Pesticide Watch.

18 I first have to thank you very much for being  
19 able to bring science into the this process. So  
20 often these processes are dominated by big money  
21 and big ads. Thank you for your time and bringing  
22 science into the process.

23 Since 1991 Pesticide Watch has worked with  
24 dozens of communities and hundreds of individuals  
25 that have been exposed to fumigant pesticide in

1 California. Fumigant pesticides are dangerous to  
2 human health and the environment and continue to  
3 have no place in California. Especially in the  
4 capacity to grow free fruits and vegetables. So  
5 speaking on behalf of community of that we've worked  
6 with since 1991, I urge you not to register methyl  
7 iodide.

8 Thank you.

9 DR. FROINES: Thank you.

10 MS. KOBYLEWSKI: Barbara LaFave.

11 MS. LAFAVE: Good afternoon, Chairman,  
12 Members on the Panel. My name is Barbara LaFave and  
13 I am a state legislative director of California  
14 Women For Agriculture.

15 And I'm not even going to try to pretend to be  
16 a chemist or toxicologist. But I'm here mostly to  
17 discuss the importance of agriculture to the State  
18 of California. And I just want to be clear, we  
19 support both organic growers and conventional  
20 growers, and we support the opportunity for all  
21 growers to have all proper tools available to them  
22 when that they need them. I don't have to tell you  
23 California is the number one agricultural state in  
24 the nation. It provides hundreds of thousands of  
25 jobs in the urban and rural areas.

1           As the ag community continues to have fewer  
2 and fewer fumigant alternatives available to them,  
3 the registration of methyl iodide becomes even more  
4 critical in order to continue to provide abundant  
5 food supply. Many of us here today actually work  
6 and live in the communities with which methyl iodide  
7 will be used. I'm one of them. I live just north  
8 of Sacramento. I do not have to remind those of you  
9 on the panel and those in the audience that  
10 California has the most stringent regulations and  
11 rules in the world as it relates to chemical use.  
12 And no doubt, if methyl bromide is registered, it  
13 will have very strict label requirements. You are  
14 all aware that where it is registered in the  
15 different states, and I won't go into that.

16           But we do believe that this is an economic  
17 issue for agriculture as a whole. Given the ongoing  
18 drought situation and increasing regulatory climate,  
19 urban encroachment, we must do everything to provide  
20 tools for our farmers to compete and for many of  
21 these farm workers to continue working in  
22 California. As I like to point out to our urban  
23 folks who take organize for granted, the next best  
24 crop to be planted in our field in the future is  
25 houses. And if we not careful, our food supply will

1 be outsourced to countries --

2 MS. KOBYLEWSKI: One minute.

3 MS. LAFAVE: -- where we will no authority  
4 over the chemical use and the safety of the food  
5 imported into our country, much less their lack of  
6 concern for worker safety.

7 I again thank you very much for your time and  
8 the opportunity to be here.

9 DR. FROINES: Thank you.

10 MS. KOBYLEWSKI: Robert Dolezal.

11 MR. DOLEZAL: Hi. I'm Robert Dolezal for  
12 the California Association of Nurseries.

13 Thank you for giving us this opportunity to  
14 speak. Our nurseries have been responsible  
15 providers of skilled farm worker jobs that are now  
16 threatened by the very process we see here today.  
17 An added step in delays and necessary and beneficial  
18 solution for soils that harbor pathogens and harmful  
19 organisms and kill our crops, kill our food supply  
20 and may prevent it from being there at all. This  
21 process delays the decision, decision of what to do  
22 about the risk assessment. It is unnecessary in  
23 light of the much more thorough peer review job of  
24 risk assessment that was conducted by USEPA as was  
25 reflected in 47 states registering this product.

1           These difficult delays will mean farm workers  
2 will lose their jobs, nurseries will move out of  
3 this state and the crop agriculture will lose the  
4 foundation of clean nematode and pesticide pest free  
5 plants that our nursery grows. The seed, the germ  
6 plasma, the starts, the tress, all the orchard crops  
7 think that this is a -- this process should be  
8 concluded by reputably reviewing the many defects in  
9 the studies of risk assessments performed here.

10           MS. KOBYLEWSKI: Manuel Cunha.

11           DR. FROINES: Excuse me. I think you just  
12 gave a talk that was directed towards the Department  
13 of Pesticide Regulation. Because this panel is a  
14 scientific panel which has nothing to do with  
15 delaying the decision on its use. And so I think we  
16 are not the appropriate group to speak to that  
17 issue.

18           MR. DOLEZAL: To the contrary, you have the  
19 responsibility to review the science.

20           DR. FROINES: We were chosen by the  
21 decision of the Director of the Department of  
22 Pesticide Regulation, and I think that is where the  
23 comment should be lodged, rather than a group of  
24 interested scientists who have spent the last two  
25 days trying to understand scientific issues

1 associated with this.

2 MR. DOLEZAL: This is an added step that  
3 has never been followed before in the State of  
4 California. For that, the pesticide Department of  
5 Pesticide Regulation has the responsibility. Your  
6 responsibility, Dr. Froines, and think panel is to  
7 conduct a thorough review of the assessment of the  
8 sciences involved here, particularly Arysta  
9 assessment science associated with how this product  
10 disperses when you model it or when you don't model  
11 it. As you know, the modeling is deficient.

12 MS. KOBYLEWSKI: Manuel Cunha.

13 MR. CUNHA: Good afternoon. Manual Cunha,  
14 president of the Nesii Farmers League.

15 I am not Japanese. I am Portuguese, if  
16 anybody -- I was born in a a place called  
17 Vacaville, directly west of here. It was a cow  
18 town. There aren't many cows left. In fact, our  
19 cow town -- our farm got taken away by Interstate  
20 80. They took it away in 1963 under eminent domain  
21 and we \$300. It was not a lot of the money for a  
22 dairy farm. But, again, I want to thank you for the  
23 opportunity for myself to be here representing many  
24 of the small farmers throughout California, as well  
25 as in Oregon, Washington and Arizona and in Provo,

1 Utah.

2           The Japanese-Americans and Asian-Americans  
3 that farm in the San Joaquin Valley and other groups  
4 in California are small farmers. Just for the  
5 number, and I appreciate these scientists. I was a  
6 chemistry major at Cal Poly San Luis Obispo. First  
7 I started to be a veterinarian major until I decided  
8 that I had to open up the livestock, and that become  
9 pretty difficult for me to do, surgery on cows. So  
10 then I went into chemistry. Okay. That is an  
11 absolute exciting field. Then I changed and became  
12 a teacher.

13           Today, here I am representing farmers and farm  
14 workers and families. But let's go to the issue of  
15 the small farms. Many of our small farmers need all  
16 the tools that they can to produce on an average  
17 acreage size. In California out of the 81,000  
18 farmers USDA report census in December of 2007 is  
19 57,000 farmers out of 81,000 are under 50 acres, are  
20 under 50 acres in the USDA census. Another shocking  
21 number for you folks.

22           I know that you don't have this as your  
23 driving responsibility, but economics of our total  
24 81 percent -- 81,000 farmers, 53 percent of them are  
25 in the net loss of \$46,000 per year. So if it

1 wasn't for the wife being the school teacher or  
2 nurse or whatever, they would not have the farm.  
3 They've got to have the tools to farm. We small  
4 farmers need that. Whatever you can do, if we have  
5 to do a better job of labeling on the container.  
6 Tremendous amount of work has been done on education  
7 with Arysta. More than I have seen on methyl  
8 bromide, even. All the training, extensive  
9 training. Making sure that that follows through.  
10 If we have to do those things, than I think we need  
11 to look at those and we will be part of that. My  
12 farmers need the trials to be complete. If not, we  
13 are going to lose those acreages to the urban  
14 development because those are ten acres, 15 acres,  
15 40 acres. They will be gobbled up by future  
16 developers. We are going to lose the greatest  
17 farmland that we have between here -- all this home  
18 sales.

19           So, again, I thank you for the time. I know  
20 you have had two hard days and hope you're able to  
21 at least get a little bit of maybe the bridge or  
22 railroad yard over here, historical site. But,  
23 again, thank you. We look forward for your working  
24 with us and trying to get us the tools we need to  
25 keep our farmers alive and our farm workers in the

1 state.

2 Thank you very much. Appreciate it.

3 MR. RANDALL: To the panel, thank you for  
4 this opportunity. I did have a letter on behalf of  
5 the African-American Farmers of California, and it  
6 is addressed to the Department. So I would like to  
7 give that you. Will Scott, the president was unable  
8 to be here because of another obligation with the  
9 farmers market in Oakland. He has great concerns.  
10 He is part of that niche or small farmers that is in  
11 California that needs these essential tools.

12 Myself, I am vice president of the Home  
13 Management Corp and president of Hall Management  
14 Group. We provide -- I provide labor or personnel  
15 to 26 different counties in California. So this is  
16 vital. This is important to me. We have been hurt  
17 by other issues that is going on. A lot of growers  
18 have sold their farmland, other developments have  
19 come in. But this is a great need for our  
20 organization, for longevity of our company because I  
21 employ over 20,000 people a year; that is full-time,  
22 part-time, seasonal. Any decisions that is made,  
23 especially with legislation, affects our people,  
24 affects their life style. I know this is not -- I  
25 heard you earlier, this panel is scientific. But

1 this panel's decision affect us economically.

2 I just want to be on record. I thank you for  
3 your time and opportunity to speak on behalf of the  
4 people that I employ throughout California.

5 Thank you.

6 MS. KOBYLEWSKI: I will call the next team  
7 if ten. Paula Placencia, Gail Bateson, Martha  
8 Guzman, Olyo Ortio, Horraccio Ramirez, Hilda Ramirez,  
9 Oscar Soriano, Romiro Mendoza, Noel Soriano, Romelio  
10 Jimenes Virgen.

11 And the first speaker is Paula Placencia.  
12 Paula will be speaking through a translator.

13 MS. PLACENCIA: My name is Paula Placencia.  
14 It is pleasure to be here today before you. I come  
15 from the area of Salinas. There is a person in  
16 Salinas, one of many, this person was taking up the  
17 tarp for the methyl bromide. Because this person  
18 didn't know his rights, it did not mention anything  
19 to the foreman about the fact that he was not  
20 feeling well. He has been affected. He suffers  
21 from headaches. He's losing his sight. Obviously,  
22 he lost his job. And all of this because of a  
23 chemical, because of a chemical that is now going to  
24 be replaced by another chemical that is even  
25 stronger.

1 I believe that California has enough pollution.  
2 The soil is so saturated by chemicals and  
3 pesticides. I believe that we don't need yet  
4 another one for more contamination so that our soil  
5 and our families suffer. Methyl iodide is a  
6 fumigant that I don't believe should be in  
7 California. Our children are suffering because of  
8 this type of fumigant. During the time of the  
9 chemical application is when our children are having  
10 a harder time with school.

11 MS. KOBYLEWSKI: One minute.

12 MS. PLACENCIA: There are no regulations.  
13 There are no announcements in the schools that  
14 fields located nearby are going to be fumigated.  
15 Please don't let our children suffer from any more  
16 contamination. California doesn't need the methyl  
17 iodide.

18 Thank you.

19 MS. KOBYLEWSKI: Gail Bateson.

20 MS. BATESON: I am Gail Bateson, the  
21 executive director of Work Safe.

22 We are a statewide organization that advocates  
23 worker protection, health and safety laws and taking  
24 care of individual workers. I appreciate the  
25 opportunity to appear before this panel. I am to

1 going to be submitting and summarizing here today  
2 comments that have been collaborate effort between  
3 Work Safe and a scientist name Kathleen Burns, who  
4 is a Ph.D. based in Lexington, Massachusetts.

5 I'm not going to try to go into the science  
6 too much and add a few personal comments because I  
7 think Dr. Froines said talk about the real world.  
8 I've been at Worksafe about month. Prior to that I  
9 worked in the occupational health branch of the  
10 California Department of Public Health. One of the  
11 programs I worked on was the occupational pesticide  
12 illness prevention program, otherwise known as  
13 OIPP. And just like DPR, it has jurisdiction to do  
14 investigations following acute pesticide illness and  
15 poisoning.

16 A couple points I want to deal with are  
17 exposure and susceptibility. By exposure of older  
18 children, particularly between the ages 12 to 17, to  
19 methyl iodide as a result of working in agriculture  
20 requires careful evaluation. First, it is important  
21 to point out there are older children working out in  
22 those fields. I personally spent time out in the  
23 field as part of the sweep that CAL OSHA and other  
24 agencies have done around breaking our laws to  
25 enforce the new CAL OSHA heat regulations. I've

1 gone out with the inspectors to see them pull teen  
2 after teen to find out whether they have a valid  
3 permit to work out there.

4       Teens are allowed to work out in the fields  
5 starting at age 12, but they need have a permit to  
6 do that. And the only restrictions that they are  
7 not allowed to apply for -- to use pesticides until  
8 they turn age 16. So they are allowed to be up and  
9 working where fields have to be sprayed and  
10 fumigated. So we're concerned about this. It is in  
11 every concern that adolescent children have elevated  
12 inhalation rate compared to adults.

13       This is a chart of accumulative exposure  
14 levels, notably higher than those of many other  
15 members of the population explained in EPA's  
16 exposure factors handbook. The inhalation of  
17 contaminated air is much higher than during manual  
18 labor. Dr. Burns goes on to express and provide  
19 some citations about concerns about dermal exposure.

20       Second, around susceptibility. Adolescent,  
21 then we go intense and rapid brain development and  
22 substantial hormonal changes. Any of us who are  
23 parents of teenagers can testify to that. But this  
24 is important because teens are highly susceptible to  
25 neurotoxic chemicals, and methyl iodide is a

1 neurotoxin. We are also concerned that methyl  
2 iodide is a hormone disrupter. And at this point  
3 there is insufficient evidence on which to base a  
4 claim that there is a safe level of exposure that  
5 can be determined for older children. And we cited  
6 several studies that describe this issue in greater  
7 depth.

8         The final point I want to make, even though  
9 you are not in the policy area, there is a lack of  
10 regulatory protection for older children who work  
11 out in the field. And the while the panel can't  
12 assess that, it is important to know the reality.  
13 As I mentioned before, the labor code does not  
14 prevent youths under the age of 16 from handling --  
15 from working out in the fields that have been  
16 sprayed or fumigated. If we look at overall the  
17 occupational pesticide illnesses that the health  
18 department has put together, about 16 percent of  
19 those are from drift. So that workers, if they are  
20 not in that particular field, the drift goes over.  
21 One of the other concerns around drift is that while  
22 employers are required to notify their own workers  
23 who are working within a quarter mile with certain  
24 pesticides, if the workers are employed by somebody  
25 else in an adjacent field, they are not notified of

1 drift. That is a real concern. There are loop  
2 holes and young workers really need to have the  
3 special protection.

4 Thank you.

5 DR. BLANC: Technical question. In terms  
6 of the age limit of 16 for working with pesticides,  
7 you alluded to. Assuming that you otherwise were  
8 permitted between the ages of 12 and 16 to be an  
9 agricultural fieldworker, do you know whether that  
10 regulation would apply only to pesticide application  
11 in its technical sense or would it also apply to  
12 preclude someone between the age of 12 and 16 being  
13 involved in hole punching or tarp adjustment  
14 --shoveling occurs during application, so I assume  
15 that would be an application. You may not know the  
16 answer.

17 MS. BATESON: I am thinking that -- I don't  
18 think that the labor code that I pulled out did not  
19 have that kind of specificity in it. They could not  
20 be involved in any exposure and application. That  
21 might be something we need to get clarification on.  
22 I understand both federal law and then it's been  
23 adopted in California as well.

24 DR. BLANC: Thank you.

25 MS. KOBYLEWSKI: Martha Guzman.

1 MS. GUZMAN: Martha Guzman, also with the  
2 California Rural League Assistance Foundation.  
3 Just to add to that question quickly. Hole punching  
4 is definitely a part of planting. So it is not  
5 something that would be considered a part of  
6 pre-fumigation application. It is something that  
7 hopefully you will hear from some of the workers  
8 about. You know, even if it is a week after  
9 fumigation has taken place, you punch a hole in  
10 there to put in a transplant, and you're breathing  
11 it. It is not gone. You know, it hasn't  
12 disintegrated fully into the soil or into the air,  
13 obviously. It's still there to be inhaled. So that  
14 is something I hope you look at closely because  
15 there is no protective equipment for that type of  
16 work performed.

17 We have had cases of workers that are tarp  
18 removers, that are part of the application process  
19 have long-term health impacts. This chemical is far  
20 worse. You've heard and you all know far better  
21 than any of us about health and long-term impacts  
22 that this has. Not only to workers, but to their  
23 families. It is just disgusting to me that the  
24 question is: Is there a job or is it the chemical?  
25 When are we going to get some leadership in the

1 state where we can have agriculture without making  
2 the choice of whether you are kid is going to  
3 develop some thyroid problem or whether you are  
4 going to have a miscarriage and can work in  
5 agriculture? This is ridiculous. Why are we even  
6 asking the question?

7 I just want to point out that today's paper  
8 has an article that talks drinking water in schools.  
9 DCP is still in drinking water in schools. EPA  
10 registered that chemical. You know, they registered  
11 it. Yesterday from the department said, "Well, if  
12 we find it in the groundwater after monitoring, we  
13 will mitigate it." We are still not mitigating DCP.  
14 Orosi has a disposable income of, like, 12 or 15  
15 percent is going to give their kids bottled water.

16 MS. KOBYLEWSKI: One minute.

17 MS. GUZMAN: This is insane that we would  
18 not have 100 percent certainty that this type of  
19 disrupter to so many parts of our health system, is  
20 not going to end up in our water. It doesn't take  
21 that much to disrupt your endocrine system. That is  
22 just your endocrine system. You know more details  
23 on the neurotoxicity parts of this.

24 It is really critical that you get to the  
25 bottom lines of these questions. Because nobody

1 else but you guys know how to ask these questions  
2 and nobody else is asking them. They are asking the  
3 question about whether or not I can -- this terrible  
4 thinking or not whether I can keep my job versus  
5 having this fumigant approved to California. That  
6 is not the question we should asking.

7 Thank you.

8 MS. KOBYLEWSKI: Speak, through a  
9 translator.

10 MR. ORTIO: My name is Horracio Ramirez  
11 First of all, good afternoon to everyone and thank  
12 you for listening to me. I am a farmer worker, and  
13 I'm always exposed to chemicals. I would like for  
14 you to put yourselves in my place and see all the  
15 things that we are exposed to. Because not all of  
16 us workers get the protection that we need. There  
17 are some places where workers are protected, but  
18 there are other places where workers are not  
19 protected at all. Like when we go plant the  
20 strawberries, that you to have punch the holes to  
21 put in the plants, there are chemical residues, and  
22 it comes out at the moment that you open the hole.  
23 And at that moment is when you get dizzy.

24 That's all. Thank you.

25 MS. KOBYLEWSKI: She'll be talking through

1 a translator.

2 MS. NOLASIO: I am here in place of  
3 another one of the my colleagues. My name is  
4 Alejandra Nolasio. I come from Salinas. I work in  
5 the strawberry fields. I have been working there  
6 for six years. Well, about a year and a half ago I  
7 was pregnant, and there was spraying near where we  
8 were. I felt light-headed and nauseous. I told my  
9 forman and all he said was it is nothing but soap,  
10 so you don't have anything to worry about. Imagine  
11 if that is what soap does to you, what is methyl  
12 iodide going to do to you?

13 The only thing that I have left from that is  
14 that right now I have something of a -- something in  
15 my eyes because of that work that I did on the  
16 strawberries. The companies always want to make  
17 money and the farm owners do also. I also know that  
18 the chemical companies do, too, and even more so  
19 when the chemicals are strong.

20 If this chemical is accepted, if this chemical  
21 is accepted, shortly after they are gone to want  
22 another one that is even stronger. Because  
23 according to them, this is going to be more work for  
24 more people because there is going to be more  
25 product. But people don't know that the more work

1 that this is, the more machinery and equipment that  
2 they are going to bring, and there is going to be  
3 less work. They are doing that already. Humans  
4 makes mistakes. Even if people are trained,  
5 mistakes always happen. Nevertheless, we are the  
6 ones that pay the consequences in terms of our  
7 health and medical bills.

8 I believe that all of us would rather be  
9 healthy than to have more money. We want you to  
10 think about it a lot. And thank you very much.

11 DR. FROINES: Thank you.

12 MS. KOBYLEWSKI: I am going to call the  
13 next ten. Deroteo Lopez, Julian Cruz, Alejandra  
14 Nolasio, Jose Agular, Francisco Cerritos, Maria  
15 Valdez, Myra Masive, Enrique Hernandez, Tom  
16 LaSaudro, Dirgillio Lopez, Santiago Vasquez, Teresa  
17 Espinoza, Manual Ramirez, Allejandro Alinjaosa,  
18 Yolanda Gasia.

19 MR. CRUZ: Good afternoon. My name is  
20 Julian Cruz. I come here to talk about my own  
21 experience. A while ago I worked in the same as  
22 illustration that was shown there, where I was  
23 covering the soil with the plastic. Our boss was  
24 sending us to -- well, we didn't know how many days  
25 were given for that plastic to be lifted. So he

1 would send us to lift the plastic with our hands.  
2 So when the time came to break plastic, you could  
3 right away feel the chemical substance. But because  
4 we needed the work and we had to do it, and because  
5 the chemical was so strong, I think, so my nose  
6 would start bleeding and my eyes would get very  
7 watery and very red all day long. And so I think if  
8 that substance is very strong, if it is that strong,  
9 what's it going to be like with the other one that  
10 is going to be approved? Because I am still today  
11 working on the strawberry, and we are exposed day by  
12 day, every day to the strawberries. Because even  
13 after time passes, the time that you are supposed to  
14 wait so that the risk of the product passes, there  
15 is always a residue.

16 MS. KOBYLEWSKI: One minute.

17 MR. CRUZ: That is all. Thank you.

18 DR. HATTIS: Have you ever had the  
19 experience of having personal protection, a gas mask  
20 or personal protective equipment given, either  
21 protective clothing or gas mask to wear?

22 DR. FROINES: Respirator.

23 MR. CRUZ: Today as another colleague said,  
24 somebody that spoke before, there is places today  
25 where they do give you protective equipment because

1 companies are afraid of being sued. There is  
2 farmers who just plant small amount of acres, a few  
3 acres, and they don't care about that. So they  
4 bring in the worker just like that.

5 DR. HATTIS: So it sometimes happens, about  
6 but half and half?

7 MR. CRUZ: It's optional. For example, if  
8 you want to protect your own hands, you have to  
9 bring your own gloves.

10 DR. BLANC: Has he ever or any of his  
11 friends been spent somewhere special to see how the  
12 respiratory fits?

13 MR. CRUZ: No. Unfortunately no. Because  
14 in one of the illustrations that you showed there,  
15 they say that there are no incidence, but  
16 unfortunately there are. It is just that because  
17 from fear, from not saying anything, it just stays  
18 like that.

19 DR. BLANC: That wasn't my question. My  
20 questions was: Has he or any of his friends ever  
21 been sent somewhere special where they check and  
22 make some fumes to see how the mask fits?

23 MR. CRUZ: At present they do. Some  
24 companies do send you, and they give you an  
25 orientation.

1 DR. BLANC: Thank you.

2 DR. FROINES: But others don't.

3 DR. BLANC: I wanted to know if anybody  
4 does.

5 MS. KOBYLEWSKI: Talking through a  
6 translator.

7 MR. AGULAR: Good afternoon. I am Jose  
8 Agular. My experience is that I work in the  
9 strawberry field. Now is the time to prepare the  
10 ground, and we have two farms that are next to each  
11 other. One here they plant and one and they follow  
12 and the other plant and the other. Right now they  
13 are applying methyl bromide. And we are harvesting  
14 here, there. And sometimes people start having  
15 headaches. The foreman would let the foreman know,  
16 but he says to us that it is not dangerous. Well,  
17 if that chemical is not dangerous, why is it that  
18 people have headaches. If they are going to bring  
19 in another that is more powerful, what can we expect  
20 from that. We hope that you will not accept that  
21 new chemical.

22 Thank you.

23 MS. KOBYLEWSKI: The next speaker will be  
24 through a translator.

25 MR. CERRITOS: Good afternoon. My name is

1 Francisco Cerritos, and I come from the area of  
2 Salinas. I just want to share with you a little  
3 about what I think. It makes me sad when there are  
4 people that are trying to pass chemicals and all  
5 they are thinking about is how to fill their bags  
6 with money, with the idea that there is going to be  
7 more production. Production is going to increase.  
8 Not that there is going to be more work for the farm  
9 workers.

10           What I am more concerned about is that my  
11 family and my community are going to be exposed to  
12 death. You should take more into account how our  
13 environment is and because there is so many  
14 chemicals. That is why there is so many new  
15 diseases that were unknown. There are companies,  
16 for example, like the one that where we come from.  
17 There they do respect the workers, for when the  
18 worker is applying the chemicals. But there is  
19 other coworkers that work for other companies as  
20 some of the other people mentioned. Because  
21 sometimes strawberries are not grown by the large  
22 companies. And some of the small farms that are in  
23 the hills, they apply whatever they want.

24           MS. KOBYLEWSKI: One minute.

25           MR. CERRITOS: The farmers know that there

1 isn't enough personnel to come and inspect. That is  
2 why I urge you not to approve it and to think more  
3 about the environment and your families. That is  
4 more important than the production and everything  
5 else.

6 Thank you very much.

7 MR. LA SANDRO: Hello. My name is Tom  
8 LaSandro. I am from the San Joaquin Valley. My  
9 family has been working for over 50 years in  
10 agriculture business, myself 27 years. I was raised  
11 in every way, shape and form of my life in the  
12 agriculture business. We depend on it. We all do.  
13 With every fiber of my being to survive financially.  
14 Give us the tools to produce hardy crops, longer  
15 harvest and keeping us employed longer, which in  
16 turn keeps my family fed. I love my job. I love my  
17 family. Please don't make me uproot my family to  
18 look for work in a different state. Each and  
19 everyone of my brothers and sisters before you have  
20 the similar story like mine with the same common  
21 denominator. Without work we do not eat.

22 Thank you.

23 MS. KOBYLEWSKI: The next speaker will be  
24 through a translator.

25 MR. HERNANDEZ: Good afternoon. My name is

1 Enrique Hernandez. I come here from Fresno,  
2 California. I am here in a show of support. There  
3 is a lot of unemployment where I live because there  
4 is not enough harvest. Because they are not  
5 allowing or approving fumigation. And I am here to  
6 support this because my family is devoted to the  
7 fields. I know that fumigant is dangerous because  
8 it is harm, but people need to work. Because all  
9 the people from where I am from, they do this and  
10 nothing else. And now since they've taken the water  
11 away, there is even less production. And it is not  
12 true that they put you to work right after they  
13 fumigate. Like a week or two weeks go by before  
14 they put you to work.

15 That is all. Thank you.

16 MS. KOBYLEWSKI: The next speaker will be  
17 talking through a translator.

18 MR. LOPEZ: My name is Dirgillio Lopez  
19 Luis. I am here from Fresno, California. I am here  
20 to support people who devote themselves to working  
21 in the field. We are cousins. If there is strike,  
22 we work and feed ourselves. If there is no product,  
23 we can't work. I work for three or four years in  
24 the field, but the ranchers always put up the sign  
25 when they are applying the chemicals. They don't

1 let us go in after they applied the chemicals until  
2 two or three days. They let us in eight or ten days  
3 later. That is a all I have to say.

4 MS. KOBYLEWSKI: The next speaker will be  
5 talking through a translator.

6 MR VASQUEZ: Hi. My name is Santiago. I  
7 am here from Fresno. I am here to support and have  
8 you approve the chemical. Without the chemicals  
9 there won't be good fruit. If you don't use it, if  
10 we don't use it, then the plants won't give good  
11 fruit and other ones that do, they will give the  
12 fruit. I always work here in the fields. And the  
13 ones who don't use the chemicals don't give the good  
14 fruit and the ones that do, do give good fruit.  
15 Without the chemicals there won't be much work.  
16 That is all.

17 DR. BLANC: I have a question. Has he ever  
18 used a mask, face mask?

19 MR. VASQUEZ: Yes.

20 DR. BLANC: With his beard?

21 MR. VASQUEZ: Yes.

22 DR. BLANC: Thanks.

23 DR. MELNICK: How often do they change out  
24 the cartridge?

25 MR. VASQUEZ: What is that?

1 DR. BLANC: The thing that goes like this  
2 and that.

3 MR. VASQUEZ: They give us protection when  
4 we work. I use a mask with a beard.

5 DR. HAMMOND: Can you just say, at the  
6 beginning of day when you use the mask, what do you  
7 -- do you do anything or just put it on?

8 MR. VASQUEZ: Yes.

9 DR. HAMMOND: At the end of the day, do you  
10 do anything special to the mask or just take it off?

11 MR. VASQUEZ: They give out masks and we  
12 just throw them away.

13 DR. FROINES: I think he's -- they are  
14 being given face masks, not cartridges.

15 DR. HAMMOND: Let me follow this through.  
16 Are these masks that go like this or like  
17 this?

18 MR. VASQUEZ: Like this.

19 DR. HAMMOND: They are white?

20 MR. VASQUEZ: Yes.

21 DR. HAMMOND: You just throw them away at  
22 the end of the day?

23 MR. VASQUEZ: Yes.

24 DR. BLANC: Is that when you're applying  
25 fumigant?

1 MR. VASQUEZ: Yes.

2 DR. BLANC: Thank you.

3 DR. LOECHLER: Are you being told that the  
4 masks help?

5 MR. VASQUEZ: Yes.

6 MS. KOBYLEWSKI: The next speaker will be  
7 talking through a translator:

8 MR. RAMIREZ: My name is Manuel Ramirez. I  
9 come from the Salinas area. I am a fieldworker,  
10 also. I work for a boss; 95 percent of us work for  
11 a boss. And my own experience is from what I know  
12 from the 17 years that I have been working in the  
13 fields. I am not [unintelligible] and it is my  
14 responsibility as father of my family, and the Bible  
15 says that if you don't work, you will not eat. But  
16 you have to understand one thing written in the  
17 Bible, that the science, while my respect to the  
18 scientists and doctors that are here, my experience  
19 six years ago in the soil fumigation, we are behind  
20 the machinery where they were fumigating the soil  
21 around 10:00 in the morning. It was very hot and  
22 chemicals were very strong. And one of my  
23 colleagues, well, the plastic part exploded and he  
24 was hit hard by the chemicals. We didn't have masks  
25 and goggles. Didn't help us. I ran away, but my

1 coworker, he was stuck there. He fell there and the  
2 fact he was taken for medical treatment. So there  
3 is no doubt that the chemicals are harmful.

4 And that three weeks ago, I'm still working in  
5 the fields, we harvested the fruit and strawberries.  
6 Among there were the rows, and amongst the plants,  
7 they were about this high. They were small.

8 The second panel, they were talking about rats  
9 and rabbits. I was harvesting the fruits and  
10 strawberries, and I put my hand in, harvest the  
11 strawberry, and there was a rabbit there and it was  
12 poisoned. In fact, I got scared. I was  
13 concentrating on harvesting the fruit when I turned  
14 around and put my hand and grabbed something that  
15 was soft, and it was a rabbit that was poisoned.

16 So I am against this new chemical being  
17 approved. I know that the chemicals that are there  
18 now are sufficient. This is my opinion. I can't  
19 say on my own that it should be approved. It is  
20 inevitable, but I am against it. And last of all, I  
21 am going to a doctor for an eye problem that I have.  
22 The doctor said it was due to chemical.

23 That is's all. Thank you.

24 DR. LOECHLER. One question.

25 DR. MELNICK: About the mask that you wear

1 have replacement cartridges?

2 MR. RAMIREZ: Yes, it did.

3 DR. MELNICK: How often are they replaced?

4 MR. RAMIREZ: I just went twice and I had  
5 to run because fear the chemical would do something  
6 to me. I can't say because they gave us new  
7 equipment, but it didn't help my coworker.

8 DR. LOECHLER: Did I understand that he  
9 said that the tarp exploded?

10 MR. RAMIREZ: Yes, it exploded.

11 DR. LOECHLER: Can you describe why it  
12 would have exploded or how it exploded?

13 MR. RAMIREZ: Sometimes with the air or  
14 sometimes the soil itself is very sandy and there is  
15 a little hole in the plastic; the air went in and it  
16 exploded.

17 DR. FROINES: Thank you.

18 MS. KOBYLEWSKI: The next speaker will be  
19 talking through a translator.

20 MS. ESQPINOZA: Good afternoon. My name is  
21 Teresa Espinoza. All my respect to you. Your work  
22 is good, but I want you to also think about us. We  
23 work in the fields, and there are many pregnant  
24 women, pregnant mothers. And doctors have said due  
25 to the very chemical there are a lot of

1 miscarriages. There three children in my family  
2 with asthma and with kidney problem due to the very  
3 fact we work in the fields while we were pregnant.  
4 So if you stop to think about women and mothers, we  
5 are struggling for our children, to give them the  
6 best of everything we can so they don't have to be  
7 in the same place that we are.

8 My respect to all of you, like I said, to all  
9 of you, to the doctors, to the scientists. I  
10 appreciate you, but please stop. I am against this  
11 chemical that you want to use again. Right now  
12 where we are working, I am coming from Salinas. We  
13 are not all given protection. We have to buy our  
14 own protection, like sleeves, the protection for our  
15 hands. And one of them, I am losing my eyesight due  
16 to the chemicals. If this one now is affecting us,  
17 what is to become of us? What is going to happen in  
18 the future with this new one, with the new chemical  
19 they want to apply? Love us, love us and love  
20 yourselves. We appreciate your lives. Appreciate  
21 ours. Very sad and very young to be losing my  
22 eyesight. I have been planting for two years. For  
23 two years now I have been moving the soil to  
24 transplant the soil, and I am losing my eyesight.  
25 My throat is very irritated now due to the

1 chemicals. The doctor told me that this is the  
2 reason, due to the chemicals. Even though we are  
3 given masks, it is very hot. It affects anyway  
4 because it all comes up.

5           What I want to tell you, take your time.  
6 Think about the children. The children God sent us.  
7 We should receive them and not do away with them.  
8 We have lots of rashes on our hands. I am against  
9 this.

10           DR. FROINES: Are the masks that she wears  
11 going back to the same --

12           DR. HAMMOND: Could you do me a favor and  
13 tell me which kind of mask you use? Point to the  
14 picture.

15           MS. ESPINOZA: None of those. They are  
16 just rags, pieces of cloth that we buy.

17           DR. HAMMOND: You say you buy your own or  
18 they gave you?

19           MS. ESPINOZA: They gave the men the masks,  
20 but not to us.

21           DR. FROINES: They did what?

22           DR. HAMMOND: They give the men the masks,  
23 but not the women.

24           Are you working in a different part of the  
25 field? How close to where the men are?

1 MS. ESPINOZA: We are together. Couples  
2 are together.

3 DR. HAMMOND: Husbands have a mask?

4 MS. ESPINOZA: Sometimes they give and  
5 sometimes they don't.

6 DR. HAMMOND: Can you say which of these  
7 masks that the men would get? Can you tell me?  
8 Maybe the picture -- maybe they are not good  
9 pictures. You can't tell?

10 MS. ESPINOZA: They are more transparent.

11 DR. FROINES: Maybe they're like doctor  
12 masks?

13 MS. ESPINOZA: Yes, like the doctors wear.  
14 Yes.

15 DR. HAMMOND: That's when you're doing --  
16 what things are you doing then?

17 MS. ESPINOZA: Picking strawberries.

18 DR. HAMMOND: Are you in the field -- do  
19 you know whether -- do you know what they mean as  
20 the fumigant?

21 MS. ESPINOZA: Yes.

22 DR. HAMMOND: Are you in the field then?

23 MS. ESPINOZA: Yes.

24 DR. HAMMOND: Do you have just a different  
25 kind of mask then?

1 MS. ESPINOZA: Just a piece of cloth that  
2 we use at work to cover our heads and cover our  
3 mouths.

4 Thank you all. Thank you to you all for  
5 listening to us.

6 MS. KOBYLEWSKI: The next speaker will be  
7 talking through a translator.

8 MR. DEROTEO: Good afternoon to all of you.  
9 My name is Doreteo Lopez. I am a fieldworker, and I  
10 am not in agreement with this chemical. I'm working  
11 strawberries for 11 years in the Salinas area. And  
12 the truth is many ranches don't care about the  
13 workers. What they care about is making money. And  
14 the truth is they don't take care of us. I have  
15 seen a lot of ranchers in the fields where I have  
16 gone to look for work in other places, and they put  
17 sulphur, and that sulphur makes the fruit ripen.  
18 And that is very harmful. Makes our eyes burn.  
19 That is why I am not in agreement.

20 And that is all. Thank you very much.

21 DR. FROINES: Thank you.

22 We're going to take a ten-minute break right  
23 now.

24 (Break taken.)

25 MS. KOBYLEWSKI: Everybody, can I have your

1 attention? I think a lot of people who wanted to  
2 speak had to leave. I'm not going to call the names  
3 by card. If you want to speak, please just form a  
4 single line behind the podium, and when you get up  
5 to the podium, please just state your name and you  
6 will still get three minutes to speak.

7 DR. FROINES: I don't believe it.

8 DR. BLANC: Just recognize that we have one  
9 more speaker, and then we are finished with the  
10 speaking session.

11 DR. FROINES: If somebody pops up we  
12 will.

13 MS. DIANDA: Hi. My name is Teresa Dianda.  
14 I am from Earlimart, California in the Central  
15 Valley, where we are surrounded by ag. It is a  
16 small town, a lot of Hispanic farmworkers. And I  
17 guess the point I want to make -- first, thank you  
18 all for being here and spending your afternoon and  
19 days and working so hard and all. But we had to get  
20 up so early to drive down here from Earlimart. Four  
21 hours to get here and four hours back. On Friday in  
22 the afternoon it will be more because of the traffic  
23 and rush hour.

24 So the point I want to make is that Earlimart  
25 in 1999 we had a big accident, and I was in that

1 accident. I live on the eastern southern most side  
2 of Earlimart. It was metam, so you know it was a  
3 fumigant. People begin getting pink eyes and  
4 vomiting and coughing. And they were unable to  
5 breathe and it was a really horrible accident, and a  
6 lot of things -- they took to maybe the physical  
7 ailment and things that came from that accident, but  
8 nobody hardly talks about the traumatization that it  
9 caused. It changed so many people's lives. Their  
10 house was changed so people that didn't have asthma  
11 had asthma the next day. But the traumatization and  
12 depression that I felt and my kids felt at the time  
13 was like the government was here to protect us,  
14 which is kind of what DPR is charged with. I read  
15 the mission statement, to protect human health and  
16 to protect ag, which is -- I don't -- contradictory  
17 in a bit in many ways sometimes. But our lives  
18 change drastically with that accident. I mean,  
19 eyesight, in a year people that could see normally  
20 perfectly, their eyesight was really, really  
21 affected. They couldn't see very well. One of my  
22 daughters, Tina, she had chronic migraine headaches  
23 so bad she would cry under her blanket and just be  
24 crying, "Mom, I want to die. Mom, I want to die.  
25 My head hurts so bad. She also had bloody noses.

1 And all of our immunity to fight illnesses was very  
2 much lessened. And so at one point all of us had  
3 throat infections and ear infections at the same  
4 time, which is something that doesn't happen. And  
5 just the misbehaving of children. It so strange.  
6 Why are the kids misbehaving? I would go to my  
7 kid's second grade class and all the kids were being  
8 punished for misbehaving. And I guess it was the  
9 nervous system being affected. I'm not saying this  
10 because I'm not a doctor or scientist. I imagine it  
11 was just all these kids just got really whopped by a  
12 strong event of metam sodium.

13 So that was a long time ago and then in 2002  
14 there was argon incident. I went by myself with a  
15 couple friends from the Center for Farming and the  
16 Environmental. Oh, by the way, I work for  
17 California for Pesticide Reform. I don't know if I  
18 said that. Now I work for them because I was trying  
19 to do so much to prevent any accidents in Earlimart  
20 happening. Like Earlimart happening again. They  
21 hired me after I learned a bunch of stuff. I kept  
22 talking up at conferences and going to meetings,  
23 really complaining about being exposed to pesticides  
24 all the time. Twenty-two million pounds of  
25 pesticides are applied around Earlimart and around

1 the Central Valley.

2           In 2002, when argon happened, we went up there  
3 and the news report said that only one person had  
4 been taken to the hospital. And we didn't believe  
5 -- I didn't believe that. And I kept telling county  
6 ag commissioner. He wouldn't go. He said what  
7 matters to me is how the accident happened and not  
8 how far it got, not how many people were sickened by  
9 that. That was crazy. So we went door to door.  
10 And the first time we got about 40 people that were  
11 affected. Their stories were identical to the  
12 Earlimart stories. It was metam sodium there, too,  
13 and argon. I think the distance from field to the  
14 houses, the front door of the houses, was even  
15 closer than the argon. So it was 40 people and the  
16 next time we went out, 91 people; and then after the  
17 DPR people got involved and the county ag  
18 commissioner somewhat got involved. Then it was 268  
19 people that were infected in argon from that drift.  
20 And by being affected, these people were just  
21 inundated with the smell, kids vomiting in the front  
22 yards, people coughing. One woman said she felt  
23 like she was going to die. She could not even  
24 breath. She said I thought the big bomb had  
25 attacked. She thought it was a terrorist attack.

1 That smell came; it was so bad. So that was 2002.

2 And then a little bit after that -- I am  
3 really honored to be able to go over and see this.  
4 It's scared to being exposed again. I have fear of  
5 being exposed since Earlimart. I'm really glad I  
6 went.

7 In 2003, there was two accidents. It was on a  
8 Friday and on a Saturday. And it was in the patch.  
9 Basically, it is the same story. That was  
10 chloropicrin. What happened was the fire department  
11 didn't pay attention to these people. They were  
12 complaining of the same thing, burning eyes and  
13 vomiting and coughing. And the fire department went  
14 out there and told them go back inside our house,  
15 open your windows and it will be fine. If it  
16 doesn't, call us back. These people had waited  
17 about an hour and a half for emergency crews to get  
18 out there. One of my friends was out there with his  
19 camcorder getting everybody. Don't worry; it is  
20 going to be fine. They'll take us out of here.  
21 Don't worry.

22 Lo and behold, they removed them out of there.  
23 This was accident number three in four years in an  
24 area of 60 miles.

25 MS. KOBYLEWSKI: One minute.

1 MS. DIANDA: So after that, since they  
2 didn't evacuate or do anything about it, on Friday  
3 they applied it again on Saturday, and then it  
4 drifted a different way, and it hit other people in  
5 another region, Rubin G. Glenn area. I think you  
6 are familiar with the accident. You're nodding your  
7 head.

8 DR. HATTIS: I would benefit greater by  
9 knowing something a bit more about how the accident  
10 happened. I am from Massachusetts.

11 MS. DIANDA: I think we have all those  
12 facts. I think all those are on record, how it  
13 happened. I think they were applied actually  
14 according to how they were supposed to, which comes  
15 my final point. The next day it went to more people  
16 and made more people sick and a lot of those people  
17 stayed in a toxic area all night. They didn't have  
18 any way of getting out of there. So a lot of them  
19 ended up with asthma and lot of them ended up with  
20 severe headaches, RSV sickness that kids get when  
21 they can't breathe right. They couldn't pay the  
22 bills. So what came out of that was a good thing,  
23 SB 291, was a bill how to improve pesticide  
24 emergency protocol.

25 DR. FROINES: Time.

1 MS. DIANDA: I am the last one so I can  
2 talk a little more. I want to talk about the cost,  
3 the cost on human health 'cause it really affects  
4 human health. It affects kid's health. I  
5 appreciate and respect everybody who works. You  
6 know, farmwork is the hardest work. My mom and my  
7 dad were farmers. I worked out in the field. I  
8 really respect anybody that works out there. But  
9 there needs to be a point where you take into  
10 account kids' health and adults' health and  
11 everybody's health. And so if you could just take  
12 that into consideration.

13 And I have a son with autism. I don't know if  
14 it comes from sulphur or not. He is 17 and he is  
15 living well. But just the things that it can cause  
16 are terrible. Miscarriages, cancer. And cancer is  
17 ugly to suffer with and see a loved one die of  
18 cancer. So please take all of those things into  
19 account. I guess that is all.

20 Thank you.

21 DR. FROINES: Now I want to thank everybody  
22 for spending their afternoon and attending. I think  
23 we have benefited very much from the testimony that  
24 we heard this afternoon. I think it gives people a  
25 sense of, as I say, the real world. And some of the

1 differences of opinion that exist because we  
2 certainly heard different points of view. And I  
3 think that is useful in terms of looking at these  
4 issues.

5           So we are done. So thank you all for coming,  
6 and we will see you some time if the future. We  
7 don't quite know when that is. We will see you for  
8 next meeting and continue on with the process.

9                           (External Peer Review Panel  
10                           workshop concluded at 4:10 p.m.)

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REPORTER'S CERTIFICATE

STATE OF CALIFORNIA                    )  
  )        ss.  
COUNTY OF SACRAMENTO                )

I, ESTHER F. SCHWARTZ, certify that I was the official Court Reporter for the proceedings named herein, and that as such reporter, I reported in verbatim shorthand writing those proceedings;

That I thereafter caused my shorthand writing to be reduced to printed format, and the pages numbered 2578 through 511 herein constitute a complete, true and correct record of the proceedings.

IN WITNESS WHEREOF, I have subscribed this certificate at Sacramento, California, on this 16th day of October, 2009.

\_\_\_\_\_  
ESTHER F. SCHWARTZ  
CSR NO. 1564