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STATE OF CALIFORNIA
AIR RESOURCES BOARD
SCIENTIFIC REVIEW PANEL
ON TOXIC AIR CONTAMINANTS

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THURSDAY, FEBRUARY 13, 2014
11:22 A.M.

JAMES F. PETERS, CSR, RPR
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A P P E A R A N C E S

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Paul D. Blanc, M.D.

Alan R. Buckpitt, Ph.D.

Stanton A. Glantz, Ph.D

S. Katharine Hammond, Ph.D.

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Dr. Andy Salmon, Senior Toxicologist, Division of Scientific Affairs

A P P E A R A N C E S C O N T I N U E D

REPRESENTING THE OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT:

Dr. David Siegel, Chief, Air, Community and Environmental Research Branch

Dr. Melanie Marty, Assistant Deputy Director, Division of Scientific Affairs

I N D E X

P R O C E E D I N G S

CHAIRPERSON KLEINMAN: I'd like to call this meeting of the Scientific Review Panel to order. And as you just heard, I just want to remind everybody that this call is being recorded. When you're making a statement, please speak into your telephone or the microphone and introduce yourself each time when you're speaking, so that the recorder can -- or the court recorder can pick up the names.

With that, I'd like to ask the Panel members to identify themselves and their institutional affiliation and their call location. And we'll will start with UC Berkeley.

PANEL MEMBER HAMMOND: This is Katharine Hammond at UC Berkeley Room 757, University Hall.

CHAIRPERSON KLEINMAN: Thank you.

UC Davis.

PANEL MEMBER BUCKPITT: This is Alan Buckpitt. It's 1101 Vet Med 3B.

PANEL MEMBER ANASTASIO: And this is Cort Anastasio in the same location.

CHAIRPERSON KLEINMAN: UCLA.

PANEL MEMBER ARAUJO: This is Jesús Araujo at UCLA from UCLA Division of Cardiology in Hollywood Room, Center for Health Sciences, Room number 16145.

PANEL MEMBER RITZ: This is Beate Ritz, professor of epidemiology from the Department of Epidemiology at Fielding School of Public Health, UCLA the same room CHS 16145.

CHAIRPERSON KLEINMAN: This is Michael Kleinman at University of California, Irvine in the Faculty Research Facility in Room 10. And I do have a member of the public here. David Rothbart, who's a professional engineer from the Sanitation Districts of Los Angeles County.

And now for the staff from California Environmental Protection Agency.

CAL/EPA DEPUTY SECRETARY SOLOMON: Wait, UCSF.

MR. MATHEWS: We haven't heard from UCSF.

CHAIRPERSON KLEINMAN: Oh, I'm sorry, UCSF.

PANEL MEMBER GLANTZ: Well, Paul and Stan are here.

MR. MATHEWS: Paul, are you there?

PANEL MEMBER BLANC: Yes.

PANEL MEMBER GLANTZ: And we're in room 366 in the UCSF Library.

CHAIRPERSON KLEINMAN: Okay. All right, California EPA.

DR. MARTY: Melanie Marty, OEHHA.

DR. SIEGEL: David Siegel, OEHHA

DR. ZEISE: Lauren Zeise, OEHHA.

DR. COLLINS: Jim Collins, OEHHA.

OEHHA CHIEF DEPUTY DIRECTOR HIRSCH: My name is Allan Hirsch with OEHHA.

MR. ALVARADO: Alvaro Alvarado with the Air Resources Board.

CAL/EPA DEPUTY SECRETARY SOLOMON: Gina Solomon with CalEPA.

MR. MATHEWS: Peter Mathews, Air Resources Board.

DR. DODGE: Daryn Dodge, OEHHA.

DR. SALMON: Andy Salmon, OEHHA.

PANEL LIAISON BEHRMANN: And Jim Behrmann with the ARB.

CHAIRPERSON KLEINMAN: Thank you very much. All right, the -- I'd like to welcome everybody to this telephone conference. This conference call meeting is follow up from our last meeting, which we held on November 1st in Sacramento. And at that meeting, the Panel reviewed the benzene document. And made several comments on benzene reference exposure levels. A major comment that had been made was that the factor used to account for variability across the human population should be increased. And there were a number of specific comments on the document.

So today, we're going to hear about the changes

to the reference exposure level document for benzene in response to the Panel comments provided at the November first meeting. Then we're going to allow the Panel to discuss and provide additional feedback to OEHHA relating to any changes in the document.

Again, everyone should speak directly into the phone, introduce yourself. And all of the materials for this meeting were provided to the SRP members and are available on the website to the public. And we are going to start with OEHHA describing the changes made to the document. And then we're going to have two lead discussants provide their comments. Then the rest of the Panel will have an opportunity to follow along with that.

So I would like to begin by inviting Dave Siegel, Chief of OEHHA's Air Community Epidemiology Environmental Research Branch to start the staff presentation.

DR. SIEGEL: Thank you, Dr. Kleinman. I just want to briefly go over the timeline for this document. The first draft -- public draft of the document was released in June 21st last year.

Oh, this is slide 2, I'm sorry.

Then we had two public workshops in Sacramento and Diamond Bar during the public comment period. The comment period ended on August 20th last year, and OEHHA took those comments and revised the document, responded to

the comments.

And on slide 3, we sent the new draft, the SRP draft, of the document to the Panel on October 9th last year. We had the meeting, the SRP meeting, on November 1st, where Panel members commented on the document, gave us a number of comments. And we went back and revised the document based on the comments received, and sent out a new draft on January 17th of this year. And today, we're here to describe the changes that we made to the document to the Panel.

And for that, I am turning it over to Dr. Jim Collins who is the author of the document.

DR. COLLINS: Jim Collins, OEHHA.

Slide 4, key change in the revised REL. As Dr. Kleinman said, the principal comment at the last meeting was the intraspecies Uncertainty Factor. And we did increase it. And as a result of increasing it, the chronic REL went down as well as the 8-hour REL. And we added text and tables to describe the data underlying the change in our rationale.

Slide 5, the chronic REL intraspecies UF. We initially used a value of 30, which is equivalent to the default, but we did not apportion it into toxicokinetic or toxicodynamic subfactors. The SRP asked OEHHA to reconsider the value of 30, either to increase it or to

strengthen the rationale for the use of the 30.

Slide 6. After much deliberation, OEHHA decided to use a UF_H of 60 based on, among other things, the Chen et al. study, which showed a 20-fold variability in chronic benzene toxicity in workers based on a three-gene interaction; other studies of variability in the production of benzene metabolites and enzyme content or activity consistent with about a 10-fold kinetic variability for the enzymes or metabolite study; and, because we still felt there were uncertainties remaining in the toxicokinetics and dynamics of benzene in infants and children compared to adults. Thus, we used a factor of 60 to account for both the toxicokinetic and toxicodynamic uncertainties among humans.

Slide 7 is the derivation of the REL pretty much the same thing that was shown last time the Lan et al. study of 250 Chinese shoe workers, who were exposed to benzene eight hours a day six days a week for an average of 6.1 years. The critical effect was decreased B cells. The LOAEL was 0.57 ppm. And we used the BMDS software and got a BMCL of half the standard deviation of 0.476 ppm with the Hill model. We then averaged it to continuous exposure of 0.204 ppm. We used a subchronic Uncertainty Factor of the square root of 10, a combined intraspecies Uncertainty Factor of 60 for a cumulative Uncertainty

Factor of 200.

And we ended up with a chronic REL half of what we previously obtained. And the current recommended REL is 0.001 ppm or 1 ppb or 3 micrograms per cubic meter.

Slide 9. Is that right?

Slide 9. In the document itself, if you have it in front of you, we expanded the discussion of the toxicokinetic studies in humans on pages 12 and 14. We expanded the discussion of genetic polymorphisms in enzymes involved in metabolism of benzene, and added three tables on that topic.

And we also expanded the discussion regarding variability in humans in the metabolism of benzene and its impact on chronic benzene poisoning in justifying and changing the Uncertainty Factor.

In slide 10 more changes related to the factor. We added Table 8.5, which lists some of the results of studies quantifying variability in the human toxicokinetics of benzene metabolism. The first part of the table shows three studies with large odds ratios, odd ratios as high as 20 for the interaction of three genes in affecting benzene metabolism.

We also listed several studies showing liver enzyme activity or content by age or ethnicity, including NQ activity among Chinese compared to the Caucasian

population and variation in a epoxide hydrolase with age. And we also included some metabolite studies from Rappaport in Table 5.

In slide 11, we added a text and table describing results of Rappaport on benzene metabolism in Chinese subjects at low benzene levels. That should be Table 8.6 not Table 8.5. We added additional text on pages 54 and 55 clarifying the limitations of the Bois study, which showed theoretically a 20-fold range in benzene metabolism was only based on three subjects.

And we listed some of the limitations of metabolism studies and the difficulty of translating these directly into the value for the Uncertainty Factor. And then finally, we presented our rationale for a factor of 60, and for not dividing it into subfactors.

Slide 12 on page 55 to 56 is basically a summary statement that basically we decided was prudent to increase the fact -- the Uncertainty Factor for humans to 60, which is twice the default. And we did not, as again, divide it.

The third chapter -- slide 13 has to do with the acute REL. We did not change the acute REL value, and we discussed it, at least limited. We did not feel that the Chen et al. study, which showed a 20-fold increase in benzene poisoning among people with three null genes, that

that necessarily could be applied to the acute REL, which was just an occasional high dose or high concentration exposure for an hour. We felt in both slides 13 and 14 that we didn't need to increase the Uncertainty Factor for the acute REL based on that study.

On slide 15 there are a variety of changes listed that we made based on the November 1st comments. We clarified -- and we'll go into detail, clarifications on the ambient measurements of benzene, reorganization and additions to the metabolism section, addition to the chronic benzene toxicity session -- section, addition to some information on the acute toxicity to children, and discussion of benzene as a TAC that disproportionately impacts infants and children.

Slide 16, benzene in the ambient air of the Bay Area and the south coast. On pages five and six, we added a footnote to Table 3.1 that the values in the table are 24-hour integrated samples. We added some information about what the MATES III study was. We included maximum values in Table 3.2. We highlighted in Table 3.3 the paired stations that were used in the MATES study, and we mentioned preliminary results for MATES IV.

On slide 17, metabolism. On page eight of the diagram, we used a more recent diagram of metabolism by Rappaport. It was suggested by the Panel. On pages nine

and ten, we added clarifying statements on possible metabolites responsible for benzene toxicity. On page ten, we mentioned obesity as a possible risk factor. And on pages 11 to 14, we subdivided the animal and human information and we did some additional -- we added a study by Kim and we summarized the studies.

On page 18, we added a new Section 4.3, which discusses the interaction of benzene and Table 1 -- I mean, benzene and ethanol, including induction of CYP2E1 by ethanol based, on the concern of a Panel member.

Table -- slide 19. On page 17, we added a recent 2013 report by D'Andrea and Reddy on the health effects of benzene exposure among children following a long-term flaring incident at a refinery in Texas. Benzene, of course, was only one of several chemicals released. The study indicated adverse effects on the nervous system and liver, but there were no exposure concentrations to give us an idea of how much benzene they were exposed to.

Slide 20. On page 20, we added a paragraph on myeloproliferative disorders and myelodysplastic syndrome, which are caused by benzene, and how they are considered currently to be cancer endpoints, not non-cancer endpoints.

On pages 24 and 25, we added these three tables. One was a table on NQO1 null diplotypes in various ethnic

groups. We added a table on GST genotypes, how they vary among ethnic groups. And we put the entire table of Chen on the interaction of three genes in affecting benzene metabolism.

On page 27, we mentioned that HIV also affects CD4 plus T-cells, which benzene does with chronic exposure.

In slide 21, developmental and reproductive toxicity, we added a footnote on page 39 about the difference between early and late nucleated red cells, which are a key cell in the study -- a key study for the acute REL. And we added a page on page 41 -- study on page 41, a study by Zhu et al., on increased sensitivity to hydroquinone in immature me at that time hematopoietic cells in mice. The study was also brought to our attention by one of the Panel members.

On Slide 22, TAC impacting children, we added on pages 57 and 58, information to strengthen the argument that benzene should be added to the list of TACs that may disproportionately impact infants and children, and a conclusion that the Panel agreed to in its previous meeting on November 1st.

So that's a rapid go-through. I guess the next thing is back to the Chairman.

CHAIRPERSON KLEINMAN: All right. Thank you very

much. At this point, I would like to ask our two lead discussants to comment. And start with Dr. Anastasio.

PANEL MEMBER ANASTASIO: Sure. Sounds good. I'm just going to go through in order of the document, so not order of importance.

First, I'd like to -- oh, sorry, Cort Anastasio, UC Davis. I'd like to start by saying I thought the draft was much improved.

My first comment is on page 6 on Table 3.3. I thought it would be helpful, if possible, to also include maximum values there as the other tables did.

On page 8, the Rappaport diagram is a nice improvement, but the nomenclature between the diagram and some of the text is slightly different for the muconaldehyde and the muconic acid. He's using e,e, whereas the text uses trans, trans or t,t. So that should be made uniform.

On page 17. So this is the new study with the flaring at the refinery in Texas.

DR. COLLINS: Yes.

PANEL MEMBER ANASTASIO: I thought this was valuable in the sense of showing that this complex mixture of refinery emissions causes toxicity. But I thought the document was a little too specific attributing it to benzene. I mean, it was good that you pointed out that

only a few percent -- what, three percent of the total emissions was benzene. But, you know, within the text, it keeps talking about benzene-exposed children, and making it, I think, too much emphasis on benzene as a mechanism or the toxicant in that exposure.

I think the value of that study again is that it shows that this petroleum mixture caused toxicity. Benzene is probably responsible for some of the toxicity, but, you know, the other 97 percent of the mass is probably responsible for some, if not most, of the toxicity. So I think that needs to be parsed out a little more carefully.

DR. COLLINS: This is Jim Collins. Could you just strike out benzene and benzene-exposed in the three places, that would -- and basically exposed children, whatever. There's lots of stuff.

DR. MARTY: Jim, we'll fix it.

DR. ZEISE: Yeah, we'll fix it.

DR. COLLINS: Okay. Sorry, excuse me.

PANEL MEMBER ANASTASIO: Oh, no, no. Sorry. Are we -- I'm happy with the back and forth. Are we not supposed to have a back and forth?

CHAIRPERSON KLEINMAN: We haven't discussed it, but I think it's, you know, good to pick up on these points as they come up, so let's continue in that mode.

PANEL MEMBER ANASTASIO: Okay.

PANEL MEMBER GLANTZ: Well, so I mean Jim had made a suggestion of just slightly editing it by taking out the term benzene-exposed. Would that deal with your problem?

PANEL MEMBER BLANC: By taking out the word "benzene" and leaving "exposed".

PANEL MEMBER GLANTZ: Okay, yeah. So instead of "benzene-exposed", it would just say "exposed".

PANEL MEMBER ANASTASIO: Yeah. I would add a little -- I think that's a good change. I would also add a little bit more about the other emissions. You know, within that 97 percent, there must be some that are also toxic with the same endpoints, including a little bit more about the other toxicants that were released.

PANEL MEMBER GLANTZ: This is Stan. I really don't think that's worth it, at this point. I mean, we do want to get the document out, and it's a small point. And I don't really think it adds anything. It's obvious. They say it's only three percent. And taking the word benzene out I think is enough. We do want to try to bring the document to closure.

PANEL MEMBER ANASTASIO: Oh, I agree with that. And I'm not saying I would want to look at it again, but that's just my opinion.

CHAIRPERSON KLEINMAN: Okay. Thank you very much, Cort. Do you want to continue?

PANEL MEMBER ANASTASIO: Yes. The next comment is on page 57.

DR. MARTY: Hey, Cort, this is --

PANEL MEMBER ANASTASIO: I'm sorry?

DR. MARTY: Cort, this is Melanie. We'll go back to that Reddy study and see what else they say about which chemicals were released. It won't take that long.

DR. ZEISE: Yes, we can do that.

PANEL MEMBER ANASTASIO: Thank you.

Fifty-seven. So this is benzene as a TAC, especially affects infants and children. You know, this REL is all non-cancer effects of benzene. Whereas, this section it seemed to be focused on cancer. And I don't know how that works if within the -- is there a cancer REL for benzene that's a separate document and does that information go there as well, or how does that work?

DR. MARTY: This is Melanie. Yes, you're right that it does discuss a lot about the carcinogenicity. We do have a separate dose response analysis for benzene that was done sometime ago, went through the Panel, et cetera. So you're right that this is the noncancer dose response assessment for benzene. This section has been added for a couple of reasons, so it's not just that we're concerned

about the cancer effects, but also some of the noncancer.

We're also using this mechanism to update our list of toxic air contaminants that may disproportionately impact infants and children.

So we just took the opportunity to put in everything, all of the arguments, including the cancer arguments in this section. So that's why -- and I realize it's a little bit confusing.

PANEL MEMBER ANASTASIO: Yeah, maybe just a sentence -- explanatory sentence at the beginning of that section would help --

DR. MARTY: Okay.

PANEL MEMBER ANASTASIO: -- explain that. And then my last comment was on the next page. So again, the same issue. And I felt that here that the Zhu study about the higher hydroquinone sensitivity of the immature mouse cells compared to the adult mouse cells might be additional evidence the disproportionate impact on children and infants for noncancer endpoints.

DR. MARTY: Okay. So we'll go ahead and add in the Zhu to that. And I think we're ready for the next lead.

CHAIRPERSON KLEINMAN: Dr. Hammond.

PANEL MEMBER HAMMOND: Yes. This is Kathy Hammond, and thank you. I agree with those comments.

In general, I wanted to say that I really was struck with this interhuman variability and how well that's documented now. I think that's great, and I don't know if there's anything else that has so much data or is as well documented, but I thought that was a major improvement.

And in terms of the exposures that were listed starting on page five of the document, I just had some comments to make. I hate to keep asking for these things, but I do think that we need to have measures of dispersion, so at least there should be a standard deviation in Table 3.1 not just the average. I always like to have geometric mean and standard deviations as well, but these are just, you know, you can do them if you want. It's not a do or die thing.

I appreciated very much having the 24-hour integrated information. And I just thought we would just, you know, kind of comment, and notice here that there are 24 stations, and 14 of them have 24-hour values that are above a half of the 8-hour standard -- of the recommended value, so that, you know, we are talking about some pretty serious things there. And actually four of them were -- four of the stations had at least one value above the recommended standard.

And by contrast, in Table 3.2, all the stations

had a maximum value that exceeded the proposed value in each of the two years listed. I also wanted in Table 3.3 to have the maximum value included in that. And I appreciate that these tables had the standard deviation.

So anyhow, to me, these were good tables, interesting, and of concern. So -- and really, I think that the document is much improved. And so these are really my comments. That's it.

CHAIRPERSON KLEINMAN: All right. Thank you, Dr. Hammond.

I'd like to ask the other Panel members now to make any additional comments. We'll start with Dr. Araujo.

PANEL MEMBER ARAUJO: Hi. Yeah, Jesús A. Araujo, UCLA. I think that the document is really much, much improved, and that they were very responsive to the suggestions and the critiques that we had in the first review. And especially I like the new information that is more explicit now about the gene-gene interactions, and as Kathy alluded, also in relation with the different susceptibility of the individuals.

One thing that I have -- I don't have entirely clear in that section though when they're examining the effects of different genes. And, for example, in page 24, and so they're looking at the CYP2E1 activity, and the

NQO1, and they're looking at different variants of the CYP2E1 that results in the slow metabolism of the chlorzoxazone in Table 6.2 for rapid metabolism of that. And, I mean, basically also there is an interaction with the null variant for the NQO1.

So at the end, they're bringing all this information to support the importance of one of the metabolites in the benzene, and which is the benzoquinone, and supporting that the benzoquinone is one of the active and toxic compounds.

So when I go back, and if I can understand, how is it all this information relates really to the toxicity of the benzene. So I go to page eight and Figure 4.1, and they print the table from Rappaport in 2010, and I do see how the benzoquinone is metabolized by the NQO1 to the hydroquinone. What I don't see is whether the benzoquinone is derived entirely from the hydroquinone by the action of MPO, in this case, or in the case of the 1,2-benzoquinone from the catechol or if there is another way of the generation of it?

So, in other words, if there is no MPO present, will there be any generation of the benzoquinone directly from the benzene, do we know?

DR. COLLINS: I don't know offhand.

DR. MARTY: I don't think we know.

PANEL MEMBER BUCKPITT: Jesús, could you repeat that question, please.

PANEL MEMBER ARAUJO: Yeah. Maybe, let me phrase it in another way.

After this table, the benzoquinones are derived from the action of the MPO on the -- I assume the MPO refers to myeloperoxidase?

DR. COLLINS: Yes.

PANEL MEMBER ARAUJO: That acts upon the hydroquinone on the left side of the table to the bottom, or the figure, or acts on the catechol, right?

DR. SIEGEL: Yes.

PANEL MEMBER ARAUJO: So acting upon the hydroquinone it leads to the benzoquinone. And acting upon the catechol, it leads to the 1,2-benzoquinone also. So in the presence of the NQO1, this benzoquinone goes back to the hydroquinone. And the data that you're showing now from the human studies is that is the NQO1 sufficient, there is actually more toxicity?

So in other words, is really the 1,4-benzoquinone, you know, more active and more toxic than the hydroquinone?

DR. COLLINS: Yes.

PANEL MEMBER ARAUJO: And is this 1,4 and 1,2-benzoquinone only generated from the hydroquinones and

through these paths or are there other ways of generation of it? If there is no MPO, or there is no --

DR. COLLINS: This is Jim Collins. We're not aware of other pathways to do that.

PANEL MEMBER BUCKPITT: This is Al Buckpitt. I agree. I think that would be the primary pathway. The one thing that is not in this metabolism scheme, of course, as you cycle those hydroquinones to the quinones, you're going to generate reactive oxygen species to super oxide. And that may be part of what's driving the toxicity of benzene and other aromatics as well.

DR. COLLINS: Yes, we allude -- this is Jim Collins. We allude to the super oxide and the ROS, but we don't go into it in much detail.

PANEL MEMBER BUCKPITT: And I agree. I think you've taken the right approach to that.

PANEL MEMBER ARAUJO: Okay. In that case, yeah, I don't really have any additional comments. I think that it's clear.

So aside from that, I don't really have any other comments. I think that I've -- again, the document looks much improved, and very good address of the points that we had at the previous meeting.

CHAIRPERSON KLEINMAN: All right. Thank you.

Dr. Blanc.

PANEL MEMBER BLANC: Hi. Yeah, I want to echo the other comments that have been made, that I think the revision is quite responsive to the comments that were made at the last meeting. And it was good to see them extensively laid out. And I feel far more comfortable with this process this way than sort of saying -- the other option we were considering last time was, well, you decide and then whatever you decide is okay. So this good to see this spelled out, because I think the arguments are convincing.

I just have a question for clarification. I don't know if you have to change anything or not, but if you go to table 6.3 on page 24, which, in general, was very useful is one of the gene prevalence tables. The data in the paragraph above, which talks about the Hmong studies, which I wasn't quite sure -- it was the 198 Hmong, which is more than any of the persons genotyped in the table below.

The table below now no longer reflects the single study. It's more than one study. It's -- is it Ross and Kelsey?

DR. COLLINS: Jim Collins. Ross, 2005, refers to like three studies that he summarizes together to get his list.

PANEL MEMBER BLANC: Okay. Is there any reason

why Table 6.3 isn't from a single study that you can't add a row for the Hmong data that you just said above there?

DR. COLLINS: Yeah, I actually -- when looking through my papers last night, there's actually table in one of Ross's papers that has it in. So I will go back and see whether or -- yeah.

PANEL MEMBER BLANC: Oh, good. And the other thing that sort of relates to this, I think it's excellent, as you said, hey, you know, by the way there's a lot of Hmong in California. But later on it kind of gets lost when you're only talking about the Chinese, but actually that's the surrogate for a lot of other Californians too. It's not just that there are a lot of Chinese Americans in California, there are a lot of Korean Americans and Japanese Americans and Native Americans and Vietnamese, which you don't have any data for, but I imagine all these other groups have a high prevalence of the polymorphisms in question --

DR. COLLINS: Null.

PANEL MEMBER BLANC: -- of the null is probably true for them too. So it wouldn't be an extensive revision, but I think, at some point, you know, just an added phrase that, you know, Chinese and other groups that are likely to be this way, or that the Chinese -- we're talking about the Chinese, but they're just a marker for a

whole series of at-risk -- probable at-risk populations or something like that.

I don't think it's a major thing, but it did kind of strike me as I read it, that I thought you were -- the argument was strong, but you actually were under-selling it a little bit.

DR. MARTY: We will add.

PANEL MEMBER BLANC: And then one just point of clarification for you guys when you go -- going forward in other documents. I guess what you guys use is EndNote or something like that to generate your references?

DR. COLLINS: Many of us do.

PANEL MEMBER BLANC: Yeah. So what that does is it doesn't actually allow you to highlight which are the newly added references when you look at this reference list. I know there are a lot of -- you did a lot of work. There's a lot of newly added references. It doesn't show up. So that's just a heads up for future things. You should try to -- you know, because it strengthens your argument. Hey, you know, there's all these new references here too.

PANEL MEMBER GLANTZ: Well, they could just go through after they create the reference list, and just highlight them.

PANEL MEMBER BLANC: Yeah. Well, I'm not saying

you have to do that for some revision, but I'm just saying in future time just take note of it that it just didn't show up here.

DR. ZEISE: That's a good suggestion. Thank you.

PANEL MEMBER BLANC: That's it.

CHAIRPERSON KLEINMAN: Thank you.

Dr. Buckpitt, any further comments?

PANEL MEMBER BUCKPITT: Mike, I sent a comment to you and it's a very minor thing on page 10. The way that reads is probably not practical. It says, "The rapid phase..." -- and this is down at the bottom under the "Toxicokinetic Studies:". The rapid phase has an elimination half life of 0.7 hours...", and that's the alpha. And the elimination half-life is 13.1.

And then the subsequent sentence says, "The long elimination half-lie for benzene is due to the formation of catechol...". Well, it doesn't make sense with what's up in the previous sentence. I'd just remove that. Anything that eliminates benzene is going to shorten the half-life.

So the long elimination half-life isn't due to the formation of catechol, quinone, and the hydroquinone. Those would actually shorten the half-life, okay?

DR. MARTY: Yeah, we'll -- Allan, we'll go and look -- go back and look at that study. I actually read

that this morning and thought this doesn't make sense. So I think maybe what we're talking about is elimination half-life of benzene and its metabolites, rather than just --

PANEL MEMBER BUCKPITT: Actually, I did look at that, Melanie, and they were just measuring benzene disappearance --

DR. MARTY: Okay.

PANEL MEMBER BUCKPITT: -- I think in the air. But again, I don't think they could really tell whether that was due to metabolism to the catechol and quinones.

DR. MARTY: Okay. We need go look -- go back and fix it.

PANEL MEMBER BUCKPITT: But if you'd go back and do that, I think it will make it clearer. It just didn't make any sense to me.

And I'd like to echo the other comments from the Committee. You really did a nice job putting this back together.

DR. SIEGEL: Thank you.

CHAIRPERSON KLEINMAN: Thank you very much.

Dr. Glantz.

PANEL MEMBER GLANTZ: Well, I also agree that the revision was very responsive to the comments. My only suggestion that nobody else has picked up is on page 24 at

the top line, you should delete the hyphen in CYP2E1. A highly substantive suggestion.

(Laughter.)

PANEL MEMBER GLANTZ: I've never seen it done that way.

DR. MARTY: You know, I think that's a deletion mark for the space.

PANEL MEMBER GLANTZ: But other than that, I was fine. I think you did a nice job.

DR. ZEISE: And we think the edit is showing a deletion mark for the space.

PANEL MEMBER GLANTZ: Oh, okay.

CHAIRPERSON KLEINMAN: Thank you.

Dr. Ritz.

PANEL MEMBER RITZ: Yeah. So again, I reiterate what others have said. It's a much improved document. I really enjoy reading G times E, but I have some wording and clarification issues with that new paragraph, but let's start from where I started having some marks.

So one is on page 21, under "Chronic Toxicity to Adult Humans". In the first paragraph, in the middle, it says, "...this bias could be highly significant". I would try to reword that to, "...this bias could be very strong", because highly -- we don't talk about biases as being statistically significant. We're talking about

biases in terms of being strong or weak. So that's just a wording.

In the next paragraph, the Ward study -- or actually the Rinsky study. It says, "The study design was case-control and estimated benzene exposures with a job exposure matrix...". And that really confused me, because the outcome was a number of white blood cells and red blood cells. And that's not a case definition. That's a continuous outcome.

So what Rinsky et al. did there is actually come up with a threshold to define what a low count is. And it's just a confusing way of stating it, because, you know, in a case control study, you need cases and they have to be defined. It can't just be a continuous outcome. If you could just add maybe the outcome measure there, that makes it better -- easier to understand what they're actually talking about.

DR. MARTY: Okay.

PANEL MEMBER RITZ: Okay. Then the -- all of the added genetic information I would suggest to actually write a one or two sentence introduction on what you're talking about, because it kind of jumps into -- oh, and now there's G times E. The first batch of G times E is metabolism related, and you should make that very clear that all of these enzymes and their genetic variants have

to do with metabolism of benzene.

And then the next batch actually has to do with the outcome assessment. And it's kind of unrelated or its's almost -- somebody who doesn't know the genetics or these enzymes would start being kind of confused, so you need a little bit of a sentence or a heading or something that actually tells you what these next set of genes are all about, the TP53, RAD51, et cetera, that they're all messing with what kind of outcome measure you're actually going to be doing, right?

DR. MARTY: Yes.

PANEL MEMBER RITZ: So you want to just distinguish that. It's also hard for somebody who doesn't know these genes and variants or enzymes, you have to be a little more careful about SNPs and functional genes. So you might want to just say that there are genes for -- there are enzymes for which the gene has a known functionality, the null variance that clearly -- there is no gene, so there is no enzyme, and list those. And then distinguish them from functional or non-functional or not knowing SNPs, because all of this is otherwise seen as the same kind of evidence. While I think people would agree that when we know functionality, it's a stronger argument than when we just have the SNPs that could go either way.

And it's especially strong for NQO1, where you

have a null genotype and then you have the 609C2T mutation, that you kind of tell the reader what the difference is between the two. And it's also for -- you know, the GSTs are null, so that's all kind of clear.

When you have these large odds ratios, I would get rid of the second digit behind the colon, especially when you have like two subjects in one of the groups. There's one phrase where it jumps and it's awkward. On page 28, there's this awkward jump from the first to the second paragraph. It seems like that second paragraph belongs somewhere else.

DR. COLLINS: This is Jim Collins. In response to criticism?

DR. MARTY: Yes.

DR. COLLINS: Okay. Thank you.

PANEL MEMBER RITZ: And then on page 30, you've got that first paragraph, "Other recent studies have not found effects of benzene on blood cell counts at such low levels". And that such, I was -- it was hard for me to know where that comes from, because the table above and the study above had low and high levels. You might just want to re-word that.

DR. MARTY: Okay.

PANEL MEMBER RITZ: And then I also didn't understand really what these ppm's in Table 6.8 now refer

to, given your critique of the study, that you don't know how long these workers have been exposed. So are these averages for anything between four and nine years, or do we know at all, or is that at time of blood draw? I mean, if there's anything you can say. It's really confusing to think, hmm, we have these very precise ppm measures, but we don't really know what they refer to -- what length of time they refer to.

I also noted that in these comparisons, you sometimes refer to showed no significant differences. I have trouble with this no significant. I don't know how that was evaluated. Was it P values? Did they -- you know, when you look at Table 6.8 for the red blood cells, less than 1 ppm is 10.8, but that confidence interval is huge, 1.4 to 83 almost. And then it just jumps down to 5.13 and the confidence interval is 0.66 to 40.

I don't think there's a difference between those two values, given the confidence intervals, honestly. No epidemiologist should be thinking there's a difference between these two values.

So I just would be more careful with interpreting any of this according to P values, when clearly the confidence intervals are so wide that these point estimates don't really tell you there's a difference. And the confidence intervals, including or excluding, also

doesn't tell you much when they're that wide. So that's just wording.

And then one last addition. You do talk about chronic toxicity in infants and children. And I know you don't want to talk about cancers too much, but you are, on page 31, talking about the cancers and saying that the benzene exposures were all measured as proxies. Actually, we just published one study in January where we're using the CARB monitoring stations for benzene, and we're using all of California's leukemia data for children under age five and show a 50 percent increase in risk with benzene. That's just come out. I don't know whether you want to add it or not. Is there a cutoff for those papers to be included?

DR. MARTY: We will add it.

DR. COLLINS: Today.

DR. MARTY: Today is the cutoff.

(Laughter.)

DR. COLLINS: Today is the cutoff.

PANEL MEMBER RITZ: I mean, that would -- you would add one sentence, that there is now a study that actually, you know, has looked at actual benzene levels. But I know this is not focused on cancer, so it's probably not as important.

DR. MARTY: It's important to the argument at the

end.

PANEL MEMBER RITZ: Yeah. You want me to send you the paper? It's in press.

DR. MARTY: Sure.

PANEL MEMBER RITZ: Okay. And then on page 32, my last note, it refers to that Texas study, Whitworth et al. 2008 as an ecologic analysis using the exposure value from EPA modeled at the Census Tract. I'm not sure that that's really an ecologic analysis. You're using the same kind of model in other papers, and it's not referred to as ecologic. I think most of the measures we have here are ecologic exposure measures, because nobody took personal monitoring devices, except for the Slama paper, the EDEN cohort, for fetal development.

But everybody else used some kind of spatial modeling or monitors that reflect exposure at home. And the monitors are two, three miles away from the home, so they're all kind of measured at an ecologic level, but that doesn't make it an ecologic analysis in epidemiology. So just be careful with that wording. It's an individual level analysis with an ecologic measure of exposure.

That's pretty much all I had.

CHAIRPERSON KLEINMAN: Thank you very much.

This is Mike Kleinman. And I agree with the comments that were made. I think the report and the staff

has done a great job in being responsive to the previous comments.

I wanted to follow up on something that Kathy Hammond brought up, and that was on the table of measured amounts showing that the maximal were exceeded at almost all of the measuring sites at some point for the maximally exceeded proposed REL. And I think it would -- if the date are available, it would be useful to have an indication of how often is the REL exceeded at these cites, since the means and standard deviations would indicate that we would approach it at -- you know, to some extent. Now, I'm wondering whether the number of exceedances is more than, you know, say a 95 percent confidence limit.

So that would be something to consider adding to that table, if the data were available, but I don't think it changes anything in terms of our evaluation of the health basis for setting the REL.

So with that, I would like to just ask if there are any additional comments or suggestions having -- you know, everybody having had a chance to listen to everyone's comments, do you have any specific changes or points that you think need to be discussed further?

Okay. Hearing none, I think everybody was reasonably specific, and I think the give and take

indicates that the staff has really been keeping track of the comments that have been made, and I haven't heard any instances where there was a problem with responding to any of the comments presented today.

Does anybody have anything specific?

PANEL MEMBER BLANC: I'd just like to make a motion that we accept the document presuming that the very minor points that were raised are addressed in the final text.

PANEL MEMBER GLANTZ: I'll second that.

CHAIRPERSON KLEINMAN: So there's a --

PANEL MEMBER GLANTZ: I'll second it.

CHAIRPERSON KLEINMAN: Okay. Who is that?

PANEL MEMBER GLANTZ: Stan.

PANEL MEMBER BLANC: And it was Paul Blanc making the motion. Stan Glantz seconding it.

CHAIRPERSON KLEINMAN: Okay. Let me just call for a vote then. I'll call for any discussion of that. Anybody want to discuss the motion?

If not, I'll call the question.

Dr. Hammond?

PANEL MEMBER HAMMOND: Yes, I agree with adopting the report.

CHAIRPERSON KLEINMAN: Okay. Dr. Anastasio?

PANEL MEMBER ANASTASIO: I vote yes.

CHAIRPERSON KLEINMAN: Dr. Buckpitt?
PANEL MEMBER BUCKPITT: I vote yes.
CHAIRPERSON KLEINMAN: Dr. Ritz?
PANEL MEMBER RITZ: I vote yes.
CHAIRPERSON KLEINMAN: Dr. Araujo?
PANEL MEMBER ARAUJO: I vote yes.
CHAIRPERSON KLEINMAN: Dr. Blanc?
PANEL MEMBER BLANC: Yes.
CHAIRPERSON KLEINMAN: Dr. Glantz?
PANEL MEMBER GLANTZ: Yes.
CHAIRPERSON KLEINMAN: And very good, the motion is adopted.

So just to finalize this, you know, the State law required that OEHHA seeks the advice and recommended changes from the Panel before they finalize the document. I think with the discussion today, the Panel has fulfilled the statutory obligation, and the Panel has made some minor clarification suggestions, which the staff has agreed to investigate. And I believe that will conclude our review of the benzene REL.

So the next item of business that I'd like to bring up is I'd like to ask Daryn Dodge, the Acting Chief of Air, Epidemiology and Risk Assessment, to brief the panel on items of --

PANEL MEMBER GLANTZ: Before we do that -- this

is Stan -- the way we've just -- since the Chair is new, the way we've handled this in the past when we have a situation like this is after OEHHA makes the changes, the Chair -- it's reviewed by the Chair one last time on behalf of the Panel to certify that the changes are all acceptable. So I just wanted to mention that, just so we have -- we don't leave it to OEHHA to decide that they've done what we've asked them. The Chair can act on behalf of the Panel.

CHAIRPERSON KLEINMAN: I'll be happy to do that.

PANEL MEMBER GLANTZ: Okay.

CHAIRPERSON KLEINMAN: Very good. So at this point, I'd like to ask Daryn Dodge to tell us about what items are coming up for 2014.

DR. DODGE: Okay. This is Daryn Dodge. The last slide, slide 24, is the staff update of items likely coming to the Panel in 2014, this year. All these documents are in various stages of internal review. But the one most likely to come up next is the Hot Spots Guidance Manual for Preparation of Health Risk Assessments.

We also have reference exposure level documents, or summaries for carbonyl sulfide, toluene diisocyanate, methylene diphenyl diisocyanate, and cancer potency value summaries for arsenic and isoprene.

Now of these chemicals, the most likely ones to come up next would be carbonyl sulfide and the cancer potency document for arsenic.

CHAIRPERSON KLEINMAN: Thank you. What I'd like to do is ask for volunteers to act as leads on the three items that are most likely coming up in the near future. On the guidance manual?

PANEL MEMBER GLANTZ: This is Stan. I think I already told OEHHA I would be willing to be a lead on that.

CHAIRPERSON KLEINMAN: Very good.

PANEL MEMBER GLANTZ: Didn't I, Melanie? Didn't I already tell you that?

DR. MARTY: Yes, you did.

PANEL MEMBER GLANTZ: Yes, because you're so charming.

(Laughter.)

CHAIRPERSON KLEINMAN: That's good. If anyone else wants to volunteer to work with Stan as a co-lead, that would be fine, if anyone would like to do that. Again, you don't have to decide now, you can let me know at some later time.

The next chemical is carbonyl sulfide. Now, carbonyl sulfide is compounded similar to H2S, in some ways hydrogen sulfide. And acute exposures result in

neurotoxic outcomes, also as a respiratory effects.

This will --

PANEL MEMBER BLANC: What's it used for?

CHAIRPERSON KLEINMAN: It's used, among other things, in rubber and fumigation for --

DR. COLLINS: This is Jim Collins. It's also an emission from refineries. There's one refinery in the south coast that puts out 7,000 pounds a year. And it's been proposed -- it's been suggested it be a fumigant that might replace methyl bromide and/or phosphine.

PANEL MEMBER BLANC: I'd be happy to be one of the two people.

CHAIRPERSON KLEINMAN: Who was that?

PANEL MEMBER BLANC: Paul Blanc.

CHAIRPERSON KLEINMAN: Beate, given that it's a neurotoxin, would you be able to act as the second lead?

PANEL MEMBER RITZ: Yeah, I can do that.

CHAIRPERSON KLEINMAN: Great.

PANEL MEMBER RITZ: Then you don't have a toxicologist?

PANEL MEMBER BLANC: I'm a trained toxicologist.

PANEL MEMBER RITZ: Oh, you are. Sorry.

CHAIRPERSON KLEINMAN: Okay. Great. And on arsenic for the cancer potency value, anyone want to volunteer on that?

PANEL MEMBER ARAUJO: I can do arsenic.

CHAIRPERSON KLEINMAN: Very good. That's Dr. Araujo.

PANEL MEMBER ARAUJO: Yes.

PANEL MEMBER RITZ: And this is for the potency of cancer, right?

DR. MARTY: Yes.

PANEL MEMBER RITZ: Well, I imagine there's a lot of human studies.

DR. MARTY: Yes.

DR. COLLINS: Yes, the basis for the potency.

PANEL MEMBER ARAUJO: It's a good idea that I do it in that case.

PANEL MEMBER RITZ: Fine. I can be the second, but it depends on how much work carbonyl sulfide is.

DR. SIEGEL: We don't think carbonyl sulfide will be very difficult. It's not a very big document.

CHAIRPERSON KLEINMAN: I did a quick literature search, and there isn't a tremendous amount of information out there, so.

Okay. Then I believe -- and then as the other documents come closer to fruition, we'll be able to be informed about that and start to select leads for the other compounds.

DR. MARTY: Mike, this is --

CHAIRPERSON KLEINMAN: So --

DR. MARTY: Mike, excuse me, this is Melanie. We weren't sure if Jesús is still going to be the primary on arsenic or is now Beate the primary?

PANEL MEMBER RITZ: I'll leave to Jesús, because I'm already the secondary on carbonyl sulfide. So we can switch if it's needed.

DR. MARTY: No. We were just confused on our end. No, I think it's good. You can be secondary on two things, but it's hard to be primary on arsenic and secondary on something else. Okay. Thank you.

CHAIRPERSON KLEINMAN: All right. Are there any other comments from the Panel regarding, you know, any future concerns?

PANEL MEMBER ANASTASIO: This is Cort Anastasio. I have -- I wanted to ask a few questions about the guidance, the scientific review document that Jim sent out.

CHAIRPERSON KLEINMAN: Yes.

PANEL MEMBER ANASTASIO: So I'm still a little confused. The first question was how many members constitute a quorum for our Panel?

PANEL LIAISON BEHRMANN: For the Panel, would be -- this is Jim Behrmann. For the panel, it would be five.

PANEL MEMBER ANASTASIO: Five, okay. And then this distinction between discussion, deliberation, and sharing questions is a little vague to me, but I guess that's the way it needs to be. I mean it seems a difficult thing to parse out.

PANEL LIAISON BEHRMANN: Well, what we would offer perhaps is maybe at the next meeting, we can have our legal counsel there to have a further discussion. That way, I'm not acting in my unofficial capacity as an attorney.

(Laughter.)

PANEL MEMBER ANASTASIO: Okay. Yeah, that's probably a different pay grade.

(Laughter.)

CAL/EPA DEPUTY SECRETARY SOLOMON: Yeah, we'll put that on the agenda.

PANEL MEMBER ANASTASIO: But can I ask this. It's okay for Panel members to -- so, for example, for Kathy and I this time, I wasn't clear if we were able to contact each other about the document as leads. That's okay, as long as you're not having a discussion of the kind outcomes, but you can talk about issues.

PANEL LIAISON BEHRMANN: It's my belief that yes, you can have a discussion between the two of you. Where we get into an issue is if you are then, in turn,

contacting other members of the Panel trying to lead to a concurrence among all of you, then that would be clearly deliberating.

PANEL MEMBER ANASTASIO: I see.

PANEL LIAISON BEHRMANN: But if it's simply the two of you discussing, I don't see a problem with that.

And especially when you'd be -- at least some of your final discussion you'd be having that at the meeting. You'd be reviewing what your comments are.

PANEL MEMBER ANASTASIO: Okay. Yeah.

PANEL MEMBER GLANTZ: Yeah, this the Stan. You know, in the past, I mean, it's not been at all unusual for OEHHA to meet with the two leads, you know, to be talking about the report.

PANEL LIAISON BEHRMANN: And that's fine.

PANEL MEMBER ANASTASIO: Oh, okay.

PANEL MEMBER GLANTZ: But I mean we're not a quorum of the Committee. We're sort of working to prepare the report with OEHHA for the Committee.

PANEL MEMBER HAMMOND: Okay. Yeah, I guess, I had thought I could talk to OEHHA, but not to other members of the Panel. That was -- so I guess that is something that needs some clarification.

PANEL MEMBER GLANTZ: I think that -- this is Stan again. I think the issue -- there's sort of two

different points. I mean, the whole reason we set up the lead system was so that the Panel could be working with OEHHA to kind of knock the rough edges off the document before it came to the full Panel for formal consideration.

So, I mean, as I said, I've had many meetings where they'll have the two leads and OEHHA get together to talk about things. And there are times that I've reached out to other members and asked them about stuff. I think it becomes more of an issue once the document -- the draft document has actually been submitted, and the Panel, as a whole is considering it.

CAL/EPA DEPUTY SECRETARY SOLOMON: This is Gina. Just a couple of comments from the Agency perspective. One point that's important is that, you know, the Panel -- the lead reviewers are not -- the intent is for them not to be collaborating with OEHHA in any way on the preparation of the document, but rather to be seeking clarification from OEHHA, so that they can better provide the independent peer review at the meeting.

So the role of the lead reviewers isn't -- you know, isn't to help OEHHA not, you know, exactly, you know, knock rough edges off the document or anything along those lines, but rather, you know, if there is, you know, a meeting between the lead reviewers and OEHHA staff, that it's designed to allow the lead reviewers to ask OEHHA

staff any questions that they might have, get clarification, so that then they really can provide, you know, very high quality peer review at the meeting.

And similarly, you know, with regard to the question about whether reviewers can talk to each other, it is absolutely fine, for example, two lead reviewers to discuss the reports and their -- you know, their thoughts and reactions. But it starts to become a problem very quickly if those conversations propagate out through the Panel. So it's not okay for -- you know, for example, the two lead reviewers to talk and then one of them to go talk to a colleague, and then other to go talk, you know, about the same thing, because that becomes what is known as a rolling meeting, and that's a problem.

So just sort of keep it as, you know, if you're preparing your review and have questions or clarification, that's fine. And then you can -- the lead reviewers, if they do talk, can summarize the gist of that conversation in the public meeting once the meeting happens.

PANEL MEMBER ANASTASIO: Thank you, Gina.

I have one question for OEHHA. Who at OEHHA should we talk to for a given document? Is that the preparers, the technical reviewers? Do you guys have a preference?

DR. ZEISE: I think right now what we do is ask

you to go through the -- to go through Daryn Dodge, the Acting Chief of the section.

PANEL MEMBER ANASTASIO: Okay.

CHAIRPERSON KLEINMAN: Are there any other comments or questions?

DR. MARTY: Just Melanie. I'd like to thank the Panel for all their input.

CHAIRPERSON KLEINMAN: On behalf of the Panel, I accept that.

(Laughter.)

PANEL MEMBER GLANTZ: So this is Stan. And Paul and I are here fighting off really bad colds, so we'd like to move this --

CHAIRPERSON KLEINMAN: I'd like to ask for a motion to adjourn then.

PANEL MEMBER GLANTZ: I so move.

PANEL MEMBER BLANC: I second.

CHAIRPERSON KLEINMAN: Okay. All in favor?

(Ayes.)

CHAIRPERSON KLEINMAN: Opposed?

Hearing none, I declare this meeting adjourned.

(Thereupon the California Air Resources Board, Scientific Review Panel adjourned at 12:33 p.m.)

C E R T I F I C A T E O F R E P O R T E R

I, JAMES F. PETERS, a Certified Shorthand Reporter of the State of California, and Registered Professional Reporter, do hereby certify:

That I am a disinterested person herein; that the foregoing California Air Resources Board, Scientific Review Panel meeting was reported in shorthand by me, James F. Peters, a Certified Shorthand Reporter of the State of California;

That the said proceedings was taken before me, in shorthand writing, and was thereafter transcribed, under my direction, by computer-assisted transcription.

I further certify that I am not of counsel or attorney for any of the parties to said hearing nor in any way interested in the outcome of said hearing.

IN WITNESS WHEREOF, I have hereunto set my hand this 27th day of February, 2014.

JAMES F. PETERS, CSR, RPR
Certified Shorthand Reporter
License No. 10063