

MEETING
OF THE
SCIENTIFIC REVIEW PANEL ON TOXIC AIR CONTAMINANTS
CALIFORNIA AIR RESOURCES BOARD

MILBERRY CONFERENCE CENTER
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UNIVERSITY OF CALIFORNIA, SAN FRANCISCO
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Reported by:
James Ramos

PETERS SHORTHAND REPORTING CORPORATION (916) 362-2345

APPEARANCES

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Dr. Paul D. Blanc
Dr. Anthony Fucaloro
Dr. Stanton Glantz
Dr. Hanspeter Witschi

REPRESENTING THE CALIFORNIA AIR RESOURCES BOARD

Mr. Jim Behrmann
Mr. Peter Mathews
Dr. Shankar Prasad
California Air Resources Board

REPRESENTING THE OFFICE OF ENVIRONMENTAL HAZARD ASSESSMENT

Dr. George V. Alexeeff
Deputy Director for Scientific Affairs

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Dr. James Collins
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Dr. Judy Polakoff
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PROCEEDINGS

CHAIRPERSON FROINES: We will officially call to order the Scientific Review Panel meeting for June 15th, 2001. We shall be discussing at the outset the issues surrounding SB 25, and why don't we proceed. And the first speaker, I believe, will be Dr. Melanie Marty.

SUPERVISING TOXICOLOGIST MARTY: Good morning. What we wanted to do was first go through some material on chemicals that came up at the last Panel Meeting as a concern to some of the Panel Members. Questions arose as to what information we found on those chemicals and why did we not think they rose to the top.

So, we can do that fairly quickly. We have four or so slides on each one, or I can just say it, however you want.

CHAIRPERSON FROINES: Show us the slides.

SUPERVISING TOXICOLOGIST MARTY: All right.

The first chemical is arsenic. Arsenic is a concern because of the toxicity information on arsenic. The epidemiology data are weak but suggestive of a possible differential toxicity between children and adults, and I'll go over that in just a second.

The airborne exposures are pretty low. The ambient air concentration ratio to our chronic REL is .03 or .04 or so. And our chronic REL also has an uncertain factor

1 of 1,000, so there's a certain amount of buffer room in that
2 number.

3 There's some indication of differential effects in
4 human studies on birth weight, fetal mortality, and
5 congenital malformations, and this information comes from a
6 series of studies done in Sweden near a smelter. The
7 problem with the studies is that there's also huge exposures
8 to lead. There's some exposures to cadmium and to mercury
9 and there were no individual exposure measurements, -- but
10 there basically is no exposure measurement.

11 There's also evidence of higher lung cancer in
12 young men, and these are people aged 30 to 39 in a community
13 in Chile that's exposed to concentrations of arsenic in
14 drinking water that were pretty high, upwards of 600
15 micrograms per liter as kids and then the concentrations
16 have dropped off because they've installed treatment. But,
17 at any rate -- I think I have a slide on that actually.

18 And then there's some information from two
19 studies, one in Thailand and Mexico, that there might be
20 effects on intellectual development, but the problem with
21 those studies again is they're fairly weak studies and lots
22 of compounding, but it's a very intriguing finding and it
23 could be that arsenic is impacting I.Q.

24 Then finally there's evidence of teratogenicity in
25 animals, intraperitoneal exposure is relatively high. I'm

1 not sure of the relevance, at this point, to airborne
2 exposures.

3 In terms of emissions to the air, in the air
4 toxics hot spots program database for, I believe, it's
5 reporting year 1999, there were about 11,000 pounds of
6 arsenic emitted by facilities reporting to the program. And
7 then the ambient air concentration was a little over a
8 nanogram per cubic meter.

9 If you assume a certain breathing rate per day,
10 you're getting about .02 micrograms per day from the ambient
11 air. The major sources really are food and drinking water.
12 And drinking water, the Department of Health Services
13 estimates the average intake is about ten micrograms per day
14 and for food, especially seafood, we're looking more at 50
15 micrograms per day. So the total exposure from -- airborne
16 exposures are a pretty small part of the total.

17 Here's a little bit of information on the Ronnskar
18 Smelter Studies and the effects they found. Significant
19 reductions in birth weight; birth weight with parity, which
20 is unusual, because usually the babies get bigger instead of
21 smaller; significant increase in congenital malformations in
22 one study, but the data were a little bit inconsistent;
23 significant increases in, if you combined the spontaneous
24 abortions and stillbirths, the study author called that
25 perinatal mortality and found a significant increase, as you

1 get closer in to the facility.

2 So the communities that were closest to the
3 facilities experienced these effects.

4 As I said earlier, no exposure assessment, and it
5 was confounded by pretty high exposures to lead and other
6 metals, so it's suggestive evidence, but it's on the weaker
7 side.

8 And then here's Alan Smith's paper, looking at
9 arsenic induced lung cancer, and this is exposures via
10 drinking water in Chile, in a community in Chile, and you
11 can see that the SMR for men age 30 to 39 is pretty large,
12 11.7 and the confidence interval goes from 6 to about 20.
13 And the author himself expressed that he was concerned that
14 the reason this age group had such higher lung cancer risk
15 is because when they were younger than ten years old they
16 were being exposed up to 570 micrograms per liter in their
17 drinking water.

18 Because this is a fairly high exposure via
19 drinking water it has somewhat limited relevance to what
20 we're looking at now, although arsenic is definitely a lung
21 carcinogen by the inhalation route, so it's actually pretty
22 interesting.

23 And then here's a little bit of information on the
24 two children's intelligence studies that I mentioned
25 earlier. So they had actually in the one study in Mexico,

1 which is Calderon, et al, they looked at arsenic in the
2 urine and found decreased verbal I.Q. with increasing
3 arsenic in the urine. And they also found with increasing
4 lead in blood they got decreases in certain of Wreshler
5 Intelligence Scale Sequential Processing Tests.

6 And then the study in Thailand looked -- actually
7 it was exposures in drinking water. It was surface water
8 contaminated by a mine and they looked at hair
9 concentrations and found that if the hair concentration was
10 higher, the I.Q. of the child was lower and that arsenic
11 exposure could explain about 14 percent of the variance in
12 I.Q.

13 So there's definitely reason to be concerned about
14 arsenic, but we just feel at this point that the airborne
15 exposures are such a small part of the total that it doesn't
16 really make it to the top five.

17 PANEL MEMBER BLANC: Two questions, just to
18 generalize from this. One would be from a policy point of
19 view what would be the proportion of air to total body
20 burden that would make you --

21 SUPERVISING TOXICOLOGIST MARTY: Be more worried?

22 PANEL MEMBER BLANC: Be more worried or wouldn't
23 raise the bar? In other words, I don't think any of us
24 would require that it be 51 percent, you know, that you'd
25 have to show that the majority of the exposure were from

1 air, but would it be ten percent or five percent?

2 DEPUTY DIRECTOR ALEXEEFF: Well, it would probably
3 be a health effect, but pretty much 20 percent would
4 definitely be an issue. In other programs where we're
5 looking at relative source contributions, when things are at
6 20 percent, it would be a concern. But it's not a bright
7 line, but 20 percent definitely.

8 PANEL MEMBER BLANC: So there would probably be
9 the sense that something less than one percent wouldn't come
10 in, something 20 percent or more would definitely hit the
11 bar. And if you were talking about something that was
12 between five and 20 it would be a subject of some
13 discussion, is that a reasonable --

14 SUPERVISING TOXICOLOGIST MARTY: Yeah, a fair
15 analysis.

16 PANEL MEMBER BLANC: My second question or more of
17 a comment would be that one of the suggestions that I made
18 to Dr. Froines and I think is reflected in this draft
19 material is that there are really two different issues that
20 we're looking at here.

21 One is the evidence that a substance has a
22 differential effect in children. And I think to evaluate
23 the scientific evidence in that regard we can adapt, roughly
24 speaking, Bradford-Hill criteria so that we can look at the
25 strength of the association, the reproducibility of it, the

1 biological plausibility, in particular which has been an
2 issue with some of the associations.

3 And then there's a second sort of column of issues
4 which has to do with how much exposure is there and how much
5 public health policy implications is there to the exposure.
6 In other words an exposure may, in fact, be present, but if
7 there's no conceivable public policy that's going to
8 intervene or, looking the other way, if there's already a
9 lot of policies in that regard, it may not rise to the
10 threshold on that account.

11 So coming at arsenic from that point of view, the
12 issue with arsenic is not that the biological evidence isn't
13 plausible and it's not that there -- there could be more
14 studies, but there's certainly been enough to raise a fair
15 amount of concern, but, in fact, exposure, the current
16 exposure levels do not appear to meet the threshold for
17 public action. Is that a fair summary?

18 SUPERVISING TOXICOLOGIST MARTY: The current
19 airborne exposures, yes, I think that's a fair summary.

20 PANEL MEMBER BLANC: And do you think that that's
21 an approach that we can take as we go through these other
22 presentations that you make and some of the written
23 documents as well?

24 SUPERVISING TOXICOLOGIST MARTY: Yes.

25 CHAIRPERSON FROINES: I think that this approach

1 that Paul has just enunciated I certainly agree with and I
2 want to make sure that the rest of the Panel feels
3 comfortable with what he's suggested. So, Peter, Stan,
4 Tony?

5 PANEL MEMBER GLANTZ: I generally agree. My only
6 slight quibble is, I think that a couple of the things that
7 you said near the end sort of really spill over into
8 regulatory questions, which really we shouldn't be
9 addressing, I don't think. We should just be looking at the
10 scientific evidence about is there evidence that there's
11 particular reason for concern for these compounds regarding
12 children. And maybe I misunderstood what you were saying
13 near the end, but --

14 CHAIRPERSON FROINES: I think I would subdivide
15 Paul's comments and he can disagree. One is the question of
16 is there sufficient exposure in California to consider it a
17 matter of concern, and I don't think that falls into a risk
18 management.

19 I think the question of whether or not if
20 something is already under a control strategy plan, maybe
21 crossing the line to some extent and I would certainly
22 weight that much less so that we don't into an area that is
23 really out of our statutory definition.

24 Do you agree with that?

25 PANEL MEMBER BLANC: Yeah, I was just thinking,

1 for example, an example might be, you know, we actually
2 can't look at pesticides, but let's suppose we were allowed
3 to look at pesticides and we had data on DDT and its
4 metabolites in terms of their preferential effects on
5 children. I mean there's still a lot of residual DDT in
6 breakdown products in the environment, but it's already a
7 banned substance.

8 You know, what would be the utility of raising it
9 up to -- so in that point of view, I think what I'm getting
10 at isn't a regulatory issue so much as taking into account,
11 not only what the current exposure levels are, but what the
12 trajectory of those exposure levels is going to be absent
13 any further intervention.

14 And I think that that, for example, that's quite
15 important when we come to manganese where the current
16 exposure levels are very low.

17 CHAIRPERSON FROINES: We certainly are within this
18 law or rather OEHHA is precluded within the law to address
19 pesticides. That doesn't mean that in our findings we can't
20 make a comment about our views on the fact that pesticides
21 are excluded. So we can come back to that at a later time.

22 Melanie, -- and so I think the bottom line, based
23 on this discussion is that given the level of evidence and
24 the level of exposure that you would recommend that arsenic
25 be considered as a quote, "Tier 2" compound, and if that's

1 the case then we should move on to the next --

2 SUPERVISING TOXICOLOGIST MARTY: That's the case.
3 I would not put it in Tier 1 at this point.

4 The next chemical is carbon disulfide. Carbon
5 disulfide, there is some evidence of increased sensitivity
6 to acutely lethal exposures and also evidence of lower
7 metabolic rates in new born animals.

8 There is some evidence, although it might be
9 called equivocal of teratogenicity in rodents. It's
10 definitely a neurotoxin in adults, as seen by occupational
11 exposures. Neurotoxicity is an endpoint which we identified
12 as important to children but we have little to no evidence
13 of differential effects for neurotoxicity.

14 And finally there's really a fairly low potential
15 for exposure in California. We have no ambient
16 concentration data for carbon disulfide, but in the air
17 toxics hot spots program database, California industry has
18 reported about 1800 pounds of CS2 emissions in 1999.

19 Interestingly enough when you look at the federal
20 database, the toxics release inventory database, from three
21 refineries we actually got more than they reported to the
22 Air Resources Board, so we're going to have to look into
23 that.

24 The Air Board looked for us at the ATEDS database
25 to see where the CS2 was coming from. It's primarily a

1 fugitive emission from refineries and it looks like the
2 sulphur recovery units would be where it was coming from
3 mostly.

4 In terms of toxicity studies for acute exposures,
5 one-day old rats were about three times more susceptible
6 than 20 to 40-day old rats to lethality. This was an LD 50
7 study.

8 Also in a metabolism study by Synderwine and
9 Hunter 40-day old rats metabolized more CS₂ to carbon
10 dioxide and expired less CS₂ via inhalation than one-day old
11 rats.

12 And in terms of developmental toxicity, rabbits
13 inhaling CS₂ at gestation days six through eight showed
14 developmental toxicity at 600 ppm, with little maternal
15 toxicity at that dose level. So that is an evidence of some
16 differential sensitivity.

17 There were also other developmental toxicity
18 studies. Some of them found -- they found different types
19 of effects at different doses, including reduced fetal body
20 weight and post-natal growth.

21 The transient delays in development were found at
22 3 ppm in rats in the Tabacova and Balavaeva study, but
23 nobody else seemed to report that.

24 Also in the two-generation rat study there were
25 terata reported at 3 ppm, but only in the second generation

1 and not in the first generation, even though they were also
2 being exposed.

3 And basically growth retardation perinatal
4 mortality embryo and fetal survival were the types of things
5 that were decreased by quite different ranges of exposures.

6 Some of the studies found terata at high
7 concentrations and some of them did not.

8 And there's just a few studies that kind of
9 indicate that maybe there's a differential sensitivity, but
10 maybe not, so in other words, the concentrations that
11 resulted in significant, either fetal toxicity or, in this
12 case, unossified sternebrae, which is basically a
13 developmental delay, also were very toxic to the mothers,
14 and that's just the point of this slide.

15 So, we do when we look at developmental toxicity,
16 we do consider whether the doses were maternally toxic or
17 not and you tend to weight it less if the doses were
18 maternally toxic. But overall I think you could say that
19 there's developmental toxicity associated with carbon
20 disulfide.

21 And this again, the issue that's keeping it out of
22 Tier 1 is exposure. We just don't think there's a lot of
23 exposure to CS₂. We would encourage the Air Board to look
24 into the TRI database and figure out why those facilities
25 reported different amounts to the feds than they did to the

1 states. And it would be very nice if we could get some
2 actual measurements of CS2 near facilities, but at this
3 point I don't think we can put it into Tier 1.

4 CHAIRPERSON FROINES: But I also heard you
5 suggesting that the data was somewhat equivocal?

6 SUPERVISING TOXICOLOGIST MARTY: The reason I said
7 that is because in certain studies that used fairly high
8 concentrations they found no developmental effects. In
9 other studies with quite lower concentrations, they did find
10 developmental effects. They weren't necessarily looking at
11 the same endpoints, so that's a little bit of an issue.

12 But there's so many studies that did find
13 something, either reduced growth delays, reduced body
14 weight, developmental delays, that I think you can say
15 that's pretty strongly suggestive evidence that CS2 can
16 cause developmental toxicity. So it's still an important
17 chemical.

18 PANEL MEMBER BLANC: And also I think it's one of
19 the few chemicals we've had presented to us where there
20 really are convincing metabolic data that newborns
21 metabolize the toxin more slowly and therefore the half life
22 is longer. Would you agree with that?

23 SUPERVISING TOXICOLOGIST MARTY: Yes.

24 PANEL MEMBER BLANC: And one of the end points
25 that is also an issue with this toxin is that in addition to

1 neurotoxicology is athrosclorosis, and I think that would
2 also be a concern if you had children exposed, starting at a
3 young age, in terms of -- in the same generic sense, you
4 know, that cancer has been an issue to your --

5 SUPERVISING TOXICOLOGIST MARTY: Okay. I should
6 have mentioned that, Stan, I'm sorry, within occupational
7 studies, athrosclorosis has been associated with CS2.

8 PANEL MEMBER GLANTZ: I don't like nagging, but
9 cancer is not -- I think there's evidence of environmental
10 causes of heart disease too, which has gotten very little
11 attention from anybody.

12 PANEL MEMBER BLANC: Do you know whether there's
13 any information or has there ever been any monitoring near
14 geothermal sites, because that would be another area where
15 you would expect natural release of this potentially.
16 Volanic sources are one of the few natural sources.

17 SUPERVISING TOXICOLOGIST MARTY: We can check that
18 out, ask the ARB if they have ever looked at that.
19 Certainly there is reduced sulphur monitoring near
20 geothermal sites, but I don't know if they've ever --

21 PANEL MEMBER FUCALORO: Do you have any idea what
22 the lifetime of CS2 in the atmosphere is?

23 SUPERVISING TOXICOLOGIST MARTY: I don't.

24 CHAIRPERSON FROINES: I bet it's longer than we
25 think, but that's a guess.

1 PANEL MEMBER FUCALORO: I don't know what I think,
2 so I'm --

3 (Laughter.)

4 CHAIRPERSON FROINES: Melanie, I think that it
5 would be -- I don't know, have there been any studies, as
6 far as you know, of measurement of carbon disulfide in and
7 around petroleum refineries, because we certainly have a lot
8 of sulphur to worry about within that environment.

9 SUPERVISING TOXICOLOGIST MARTY: I'm not aware of
10 any, I hate to say I'm not aware of any, but if anybody is
11 here from the Air Board who could answer that question?

12 CHAIRPERSON FROINES: Is there?

13 DR. PRASAD: It's been correctly planned and in
14 the Bay Area there'll be two sites which we'll be monitoring
15 for carbon disulfide starting in the next couple of months.

16 Shankar Prasad from the Air Resources Board.

17 CHAIRPERSON FROINES: I think this is an important
18 point. I think that there is sufficient evidence to
19 indicate, as we've known for quite some time about the
20 toxicity of carbon disulfide, so if there is a potential for
21 hot spots I think it's worth documenting. And so we'll
22 presumably proceed with this is a Tier 2 chemical from the
23 standpoint of SB 25, but I think it's a general issue as a
24 toxic air contaminant that it is something for which we
25 should have additional information on potential exposures in

1 this state. Especially because it really does affect so
2 many different end points, toxicologically.

3 PANEL MEMBER BLANC: Well, also, John, wasn't it
4 one of the minor breakdown products of metam sodium?

5 SUPERVISING TOXICOLOGIST MARTY: Yes.

6 PANEL MEMBER BLANC: And that is the -- well,
7 we'll come back to this one-page summary issue, but I think
8 it's --

9 CHAIRPERSON FROINES: And my impression from that,
10 Paul just said of the minor breakdown products of metam
11 sodium, but I suspect that we really don't know, either as a
12 biotransformation product or as an atmospheric breakdown
13 product to what degree carbon disulfide is produced relative
14 to some of the other compounds.

15 Let's go ahead. We're going to have to avoid
16 these kind -- I just fell into talking about a subject area
17 that I'm interested in and I know Paul is interested in it
18 too, and we're going to have to be careful today to avoid
19 some of that so we stay on some sort of reasonable
20 timeframe.

21 SUPERVISING TOXICOLOGIST MARTY: Another chemical
22 that came up at the last meeting was chlorine, which, as
23 everyone is aware, causes irritation of the respiratory
24 tract, eyes and skin. As such you may suspect that chlorine
25 might exacerbate asthma, and OEHHA has identified asthma as

1 a disease that disproportionately impacts children, thus
2 children may be more susceptible to chlorine in the air.

3 A study by D'Alessandro, et al, of which Dr. Blanc
4 was one of the authors, did demonstrate in adults who were
5 hyper-responsive that they showed a greater loss in FEV1 and
6 a greater increase in airway resistance than five normal
7 adults at 1 ppm. And I'm recalling correctly that at .4 ppm
8 the difference was not significant between the hyper-
9 responsives and the "normals".

10 We did try to look at any information from
11 accidental releases, because chlorine is probably number 2,
12 if I'm recollecting correctly, in terms of accidental
13 releases, ammonia being the first.

14 There was one study that we found on accidental
15 release of chlorine vapor at a swimming pool in Italy and
16 there were quite a number of kids and adults present who
17 experienced respiratory symptoms and it seemed that about
18 the same percentage of adults and kids experienced
19 respiratory symptoms, for whatever that's worth. And that
20 more adults reported persistent respiratory symptoms after
21 the accident than kids. A little bit of information, I
22 don't know if you can do too much with that.

23 The emissions information that we have from ARB's
24 ATEDS database in '99 indicated considerable emissions
25 statewide of chlorine, 245,000 pounds or so. In talking

1 with the chemists at ARB, chlorine levels are not routinely
2 monitored in California because it's too reactive and they
3 can't measure it.

4 They do measure chloride in particulate, so on
5 their website chloride as particulate is reported, but it's
6 not chlorine gas, it's largely soluble salts of chlorine --
7 chloride salts.

8 PANEL MEMBER GLANTZ: And the emissions are due to
9 what, primarily?

10 SUPERVISING TOXICOLOGIST MARTY: That I would have
11 to ask ARB to respond to. I don't know myself.

12 PANEL MEMBER GLANTZ: Hypochloride?

13 SUPERVISING TOXICOLOGIST MARTY: No, this is
14 actually chlorine gas emissions.

15 PANEL MEMBER GLANTZ: Yeah, but hypochloride will
16 go to chlorine.

17 SUPERVISING TOXICOLOGIST MARTY: Oh, hypochloride,
18 I'm sorry.

19 PANEL MEMBER BLANC: The issues include that we do
20 actually have hypochloride manufacturing here but also
21 chlorine is a major intermediate in chlorinated hydrocarbon
22 manufacturing. So I'm assuming it's largely in the
23 petrochemical industry one way or the other. It's not
24 refining, per se, it's synthesis.

25 I don't know if we have primary chlorine gas

1 manufacturing facilities in California or not, to tell you
2 the truth.

3 CHAIRPERSON FROINES: We really don't have much
4 petrochemical manufacturing either.

5 PANEL MEMBER BLANC: Well, this would be, you
6 know, breaking it down, you know, -- and that I don't know.
7 But the reason why this number underestimates is that what
8 it won't include is the small point source releases,
9 including water purification plants, swimming pools and then
10 household releases. But the issue in your chlorine
11 presentation, as opposed to the last two, is not that you
12 don't think the exposure potential is widespread, it's
13 really more that the database for preferential child effect
14 is very weak.

15 SUPERVISING TOXICOLOGIST MARTY: It's limited.

16 PANEL MEMBER BLANC: And it would rest entirely on
17 two suppositions. One is that one could reproduce the
18 findings that people with airway hyper-responsiveness are
19 preferentially responsive. And the extrapolation that was
20 made earlier that, ergo, children, because they have smaller
21 airways and have more asthma would bear the brunt of this.

22 So, to the extent that you would show that people
23 there with hyper-responsiveness respond preferentially, if
24 that were established then, based on the criteria you've set
25 forth in the document, you'd actually be forced to put it in

1 tier 1, because you've already stated that that's going to
2 weight heavily. But I don't think that the database
3 supports that preferential response in airway hyperactivity.

4 SUPERVISING TOXICOLOGIST MARTY: Yeah, I would
5 agree to an extent, but I also don't think that there's
6 widespread exposure to chlorine, except what is not
7 widespread. Accidental releases are really the issue.

8 PANEL MEMBER BLANC: Yeah, but they're a common
9 enough event, I think, that we're talking about something of
10 concern, particularly these little small point source
11 exposure --

12 PANEL MEMBER FUCALORO: They're not accidental,
13 they're just released. I mean if you want to call it
14 accidental, but they are --

15 PANEL MEMBER BLANC: Unintended.

16 CHAIRPERSON FROINES: But I also think, I don't
17 entirely agree with Melanie. I think that what you say may
18 be true, but it also, given the level of this discussion, it
19 suggests that we don't know either.

20 SUPERVISING TOXICOLOGIST MARTY: The water
21 treatment plants, the big ones are already in the hot spots
22 database, but there's probably little ones all over the
23 place that aren't. That's another problem.

24 CHAIRPERSON FROINES: There's a lot of swimming
25 pools in southern California. But I think that the other

1 point to make is I think Paul is trying to press on your
2 defining as clearly as possible the basis of your decision-
3 making.

4 SUPERVISING TOXICOLOGIST MARTY: Yes.

5 The next chemical that came up at the last meeting
6 is manganese. Manganese is definitely neurotoxic and animal
7 studies are out there which show that rat pups, rodent pups
8 are more sensitive than adults. However, the ambient air
9 concentrations and the potential exposures appear to be
10 relatively low.

11 The other issue is that manganese is actually an
12 essential nutrient and that exposure from your diet is about
13 four orders of magnitude higher than from typical ambient
14 air, which we'll get to in a second.

15 Manganese is an essential nutrient. It's needed
16 for lipid-synthesis and oxidative phosphorylation. There's
17 actually an adequate daily intake set for kids, one to
18 three, of 1.2 milligrams per day. And then for men and
19 women between about two milligrams per day.

20 There have been a couple of studies, looking at
21 kids who were hyperactive and/or learning disabled, both, I
22 guess. And they looked at elevated hair manganese levels as
23 -- or they looked at hair manganese levels as an indicator
24 of exposure to manganese. And in this study the children
25 who were learning disabled had higher levels than normal

1 kids and the difference was significant at $p < 0.05$.

2 However, no correction was made for compounding
3 for other exposures, including lead.

4 There have been some indications that pediatric
5 patients on Total Parenteral Nutrition are more likely to
6 show neurotoxic effects of manganese than are adult
7 patients, so there's three or so citations looking at that
8 issue.

9 In terms of the experimental animal studies, young
10 rats showed neuronal degeneration in the cerebral cortex and
11 cerebellar cortex after only 30 days of oral administration
12 of 50 micrograms of manganese per day -- manganese chloride
13 per day.

14 Adult rats required a longer time period to show
15 the same amount of neuronal degeneration, so that right
16 there is an indication that young animals are going to be
17 more sensitive to manganese neurotoxicity.

18 There's some information, and I don't know what to
19 do with this, but that manganese homeostasis is suspended
20 during pregnancy and lactation from farm and lab animals.
21 We're not sure if that is the case with people. And the
22 suspension of the homeostasis allows for higher levels of
23 manganese in fetal and neonatal blood and tissues.

24 So if you're being exposed young to elevated
25 manganese, you're in trouble from a homeostasis standpoint.

1 Studies have been done looking at rat pups exposed
2 as neonates to manganese and it's showing that there's
3 elevated brain levels of manganese relative to the control,
4 so it's getting into the brain.

5 PANEL MEMBER FUCALORO: And this is all manganese
6 2, right, manganese plus 2?

7 SUPERVISING TOXICOLOGIST MARTY: Yes, that's
8 right.

9 And adults only showed increased brain levels, but
10 the exposures had to be higher, so that again is an
11 indication that pups are going to be more sensitive to
12 impacts on the brain.

13 And also there have been some neurobehavioral
14 studies, looking at hyperactivity in rat pups at post natal
15 day 21 who were exposed as neonates to manganese in their
16 drinking water. They also, Pappas, et al, '97, observed
17 basically the same thing, increased locomotion and rearing
18 in an open field in rat pups whose mothers were exposed
19 during gestation. So this is gestational exposure resulting
20 in hyperactivity in the offspring.

21 And then also, following oral exposure as
22 neonates, at post natal day 21, the animals exhibited
23 increased acoustic startle response, which is along the same
24 lines as a hyperactive animal would -- you would see in a
25 hyperactive animal.

1 In terms of actually looking at histological
2 evidence, cortical thinning has been observed in rat pups
3 who were exposed -- whose mothers were exposed, so this was
4 in uter exposure looking at the rat pups post natal day 32
5 and observing cortical thinning in the brain. There was
6 some comment by the author that it may result -- maybe it
7 was not just manganese, but have something to do with the
8 nutritional status of the animals. And then Dorman, et al
9 in 2000 did not see this neuroanatomical effect.

10 And then again some evidence of lack of manganese
11 homeostasis in newborns. Newborn mice are unable to excrete
12 manganese very well relative to an adult mouse and maintain
13 manganese blood levels relative to an adult mouse for the
14 first 17 days or so of life in this study. And then,
15 interestingly enough, manganese in the hair of human infants
16 has increased significantly during the first six weeks after
17 birth if you're formula-fed, but not breast-fed. And
18 formula has a lot more manganese, especially formula made
19 with soy, than breast milk.

20 In terms of exposure considerations the ambient
21 levels of manganese are fairly low as measured by ARB's
22 monitoring network. There are some significant hotspots
23 emissions, 105,000 pounds per year statewide in '98.

24 The daily exposure from the diet for a child
25 should be about a milligram per day. If you use the ambient

1 concentration measurements, then from ambient air they would
2 be getting about 210 nanograms. Now that's average ambient,
3 it's not next to a hotspot, so we still have some concerns
4 about the hotspots emissions and unfortunately we don't have
5 information on what concentrations are found near source.

6 PANEL MEMBER FUCALORO: And what are the sources
7 for emissions of manganese?

8 SUPERVISING TOXICOLOGIST MARTY: This is a good
9 question, if anyone from the Air Board can help me out on
10 it?

11 PANEL MEMBER FUCALORO: I guess what I'm asking is
12 all these data, toxicological studies are based on manganese
13 plus two.

14 SUPERVISING TOXICOLOGIST MARTY: Uh-huh, so what
15 is --

16 PANEL MEMBER FUCALORO: It's quite possible it's a
17 higher oxidation state of manganese --

18 SUPERVISING TOXICOLOGIST MARTY: Okay, I can tell
19 you that the --

20 PANEL MEMBER FUCALORO: It's particulate matter I
21 assume?

22 SUPERVISING TOXICOLOGIST MARTY: Yes.

23 I can tell you that manganese is reported as total
24 manganese and it's not speciated.

25 PANEL MEMBER FUCALORO: I understand, depending on

1 how they do the analysis. I mean, they can do total
2 manganese. But, you know, chromium, I mean not to say that
3 this is as bad as chromium, but chromium 3 or chromium 2 is
4 not very toxic, but it's chromium 6 which is the hexavalent
5 state of chromium, which is, and that's the one that's of
6 great concern.

7 And so I'm just wondering if there's similar --
8 I'm not a toxicologist, I'm just wondering if there's a
9 similar situation with manganese?

10 PANEL MEMBER BLANC: I don't think the
11 toxicological data has suggested that. And some of these
12 experiments are done with manganese chloride and some are
13 done with manganese oxide and, in fact, organo-manganese
14 compounds seem to have quite a bit of neurotoxicity as well.

15 There is another issue in terms of the relative
16 contribution of inhalation versus ingestion and that is that
17 the -- well, there's two issues. One is that the absorption
18 from the respiratory tract is probably far greater than from
19 ingestion. There's a lot of data that suggests that and, in
20 fact, the most severe intoxications are from inhalational
21 exposures in occupational groups.

22 And the second issue is that there's even some
23 more recent intriguing data that suggests that it may not be
24 the respiratory fraction, but that in animal models
25 manganese deposited in the nasal tract is actually taken up

1 directly by neurons in the olfactory tract and transported
2 directly to deep brain centers and that may be, in fact, the
3 route of exposure that matters the most for some of the
4 basal gangliar effects of manganese.

5 So the issue here that people get more from their
6 diet and that you have a minimum dietary requirement may not
7 be applicable as the science changes. It's a very unusual
8 scientific issue, but nonetheless, I think it's one that
9 should be recognized.

10 So, in summary, in terms of manganese, my
11 impression from your presentation would be that the
12 preferential effects on children is as strong for this
13 substance as for any that we've looked at virtually, absent
14 maybe lead.

15 SUPERVISING TOXICOLOGIST MARTY: Lead or mercury I
16 think really are up there. But, yes, definitely --

17 PANEL MEMBER BLANC: Okay. And therefore the sole
18 issue is the levels of exposure, either current or
19 projected. And that being said, I think that both in our
20 findings and certainly in your final report, I think there
21 needs to be some very strong wording that if this is to
22 remain -- that this may be tentatively put in tier 2 to
23 start with, but here are the areas in which there's, you
24 know, very high levels of concern if either the hotspots
25 data reveal a population at risk within discreet

1 geographical areas or if there is introduction into
2 California under any circumstances of new sources of either
3 inorganic or organic manganese exposure.

4 CHAIRPERSON FROINES: I'd like to follow up on
5 that.

6 As I sat and listened to the presentation, my
7 sense is that up to now we've talked about there being two
8 tiers, and when you originally presented your information
9 you had the 11 compounds, five in the first tier, six in the
10 second. I would almost argue that one should further
11 subdivide the approach and actually have three tiers,
12 because I think that manganese is considerably different
13 than arsenic, for example.

14 I think arsenic is a very very important compound
15 from the standpoint of carcinogenesis, for example, but in
16 terms of children's effects it doesn't rise to the same
17 level. Whereas manganese, the evidence seems quite strong
18 relatively speaking, and in that regard when we have -- you
19 know, this law forces us to make decisions based on very
20 limited evidence and that's one of the difficulties and the
21 frustrations about the SB 25 process, that the evidence is
22 so limited. And where we do find sufficient evidence, then
23 it seems to me we have to highlight it and take it quite
24 seriously.

25 And in that respect I would argue that we put

1 manganese in its own tier and there may be other things that
2 join it, but at least at this point that it doesn't just get
3 lumped with vinyl chloride and glycol ethers and
4 noncoplanar, PCBs and arsenic and chlorine.

5 PANEL MEMBER GLANTZ: Can I argue against that?

6 CHAIRPERSON FROINES: Let me just finish my point.

7 The 105,000 pounds per year is obviously -- I
8 agree, by the way, with Paul, I think that the weakness of
9 the argument that you make here is that the toxico-kinetics
10 are not effectively taken into consideration and that's
11 unfortunate, but let's let it go for the time being. We all
12 recognize that there may be a significant difference between
13 the uptake through the gut and through the lung or through
14 the nasal passages as Paul suggested.

15 But I always have problems when we look at these
16 ambient air exposures for California because I live in Los
17 Angeles and we have more of everything.

18 (Laughter.)

19 CHAIRPERSON FROINES: And in that regard, to
20 compare Mt. Shasta with, you know, Pico Rivera, just is
21 not -- it's apples and oranges. And so when you tell me
22 about hotspots and manganese I worry that there are places
23 where the concentrations are considerably higher.

24 So this is actually a substance that I think
25 requires real follow-up activity. And it needs to be

1 pinpointed, especially given its potential for long-term use
2 on a national basis as well, and I don't think we should
3 take that up here.

4 But I think that we need to further clarify the
5 exposure question and I personally -- Stan's about to occur
6 argument notwithstanding, would put some emphasis on
7 manganese in at least our findings.

8 PANEL MEMBER GLANTZ: Well, I don't have any
9 problem with putting emphasis on manganese in the findings
10 and including the points that you two have made. But I
11 think, you know, if you go back to what you said earlier
12 about SB 25 has required us to move this process quite
13 quickly and not dig into certain things with the level of
14 compulsiveness that we've sometimes done, I think to then
15 start subdividing the second tier even further is just
16 silly, and I think it will open us up to further criticism.

17 I mean I think -- and remember we have a couple of
18 more tiers we're not even talking about which are those long
19 lists in the back of the document. And so I really think we
20 should do as we have in the draft findings that I worked
21 with Melanie on, which are, before we discuss, I need to
22 explain how they're structured. They're a little unusual.

23 But I think we should say these are the five and
24 these are the remaining, however many there are, and it
25 doesn't have to be six, it can be however many there are,

1 with why we think that they're important. And I think that
2 to include the kind of statements that you've been making in
3 the findings is totally reasonable and appropriate.

4 But I just think to start subdividing that list
5 further is just silly and I think that the points have been
6 made, I think they're on the record. This is going to be an
7 ongoing process and I think they'll be taken into account,
8 and if they're not then at that point we can jump up and
9 down. But I think that it's just not appropriate to start
10 subdividing that and prioritizing within the -- you know,
11 we've got the top five. We've got the bottom whole bunch of
12 them and then we're talking about this intermediate list
13 which isn't going to be that long a list anyway. And I just
14 think if we put manganese in there and we say the things
15 that have been said that that's adequate.

16 PANEL MEMBER FUCALORO: But you know, I'm agnostic
17 on this issue, but I think Paul's mission has been that we
18 mention the reason why a particular substance that makes for
19 tier 1 and they fall into two categories, possible
20 categories, differential toxicity and the other is exposure.
21 And I think that would be sufficient that we make sure that
22 those two points are made for every substance that doesn't
23 make the list.

24 PANEL MEMBER GLANTZ: I don't have a problem with
25 that.

1 CHAIRPERSON FROINES: Okay, why don't we come back
2 to this. I probably was premature. I think that we should
3 take this up when we talk about our findings. This is one
4 where I think there should be some level of emphasis that's
5 different than some other compounds, but I think we can take
6 that up when we get there. And we'll ask Stan to make an
7 argument that's different than this is silly. We'll go for
8 the more substantive argument at that point.

9 (Laughter.)

10 PANEL MEMBER GLANTZ: I thought that was a pretty
11 substantive argument.

12 CHAIRPERSON FROINES: Go ahead, Melanie.

13 SUPERVISING TOXICOLOGIST MARTY: Okay.

14 The fifth chemical that came up at the last
15 meeting is methyl bromide. There is some -- and I have to
16 say that this is a pesticide, but that regulation of methyl
17 bromide as an emission from a stack from fumigation chambers
18 has been allowed by a court decision.

19 There is some evidence of teratogenicity in
20 reduced birth weight in rabbits and rats. Methyl bromide is
21 a neurotoxicant. It's been observed many times in
22 occupational settings and following accidental exposures and
23 also in animal studies. And neurotoxicity is an end point
24 of concern for infants and children. But exposures to
25 methyl bromide are not widespread from stationary sources.

1 Methyl bromide is neurotoxic. Occupational
2 exposures have resulted in neurotoxicity. I just put a few
3 of the references that are available in the literature going
4 all the way back to the 1940s.

5 Animal studies have repeatedly demonstrated
6 neurotoxicity. Rodents seem to be somewhat resistant, but
7 dogs are a sensitive species. And the symptoms noted in
8 both people and also in animals include headache, nausea in
9 people and vomiting, tremors, convulsions and other signs of
10 neurotoxicity in animal studies.

11 In terms of differential effects, we don't have
12 any studies of neurotoxicity in neonates or young animals
13 versus older animals, but neurotoxicity is definitely an end
14 point of concern for kids because their nervous system is
15 developing all the way throughout lessens. Developmental
16 effects have been noted in rabbits at 80 parts per million,
17 including gall bladder agenesis which was repeated in
18 another study by Breslin. It's very unusual to not see any
19 historical controls to any extent.

20 Also reduced birthweight and fused sternebrae are
21 noted in the rabbit study by Breslin.

22 Rats have also showed reduced body weight gain in
23 pups exposed during lactation, so this is a young animal
24 exposure showing an effect. And exposure of rats in utero
25 has resulted in decreased brain weight and this was noted at

1 30 parts per million by Norris, et al and also in the width
2 of the cerebral cortex, which was noted at a higher dose in
3 two other studies.

4 So I think the upshot is we know it's a
5 neurotoxicant. We can see effects when exposure occurs in
6 utero. We are somewhat constrained in looking at exposure
7 issues since we only can consider basically fumigation
8 chambers and exposures from fumigation chambers. We know
9 there's some emissions from fumigation chambers in ports
10 where they're using methyl bromide to fumigate fruit that
11 comes in from out of the country.

12 CHAIRPERSON FROINES: With hexane, 2, 5-hexadione
13 you argued that there was no evidence of differential
14 toxicity and that the distal axonopathy that occurs does not
15 appear to be an age related or a children related
16 phenomenon. But here you just have made the statement that
17 said it's neurotoxic and children have developing brains,
18 therefore there's a matter of concern.

19 And there's a little contradiction between -- on
20 the one hand you're saying that because of developing
21 brains, neurotoxicity is a potential problem and in one case
22 you say methyl bromide, that that may be an issue and with
23 hexane you say it isn't an issue. So I think that one has
24 to be careful in discussing the evidence to suggest that is
25 there a mechanistic basis that the developing brain may be

1 more particularly susceptible to the mechanism of a
2 particular neurotoxicity. And that's just a general
3 comment.

4 PANEL MEMBER BLANC: Well, I think the big -- you
5 know from a theoretical point of view, because this is an
6 issue of central nervous system, toxicity versus peripheral
7 nervous toxicity, that may be the rationale. But, in
8 general, if you had to characterize, leaving aside the fact
9 that the exposure levels appear to be quite low from the
10 source that you can look at, that the database on a
11 differential effect is modest, at best.

12 SUPERVISING TOXICOLOGIST MARTY: Yes.

13 PANEL MEMBER BLANC: And certainly there is no
14 evidence in the key end point which would originally raise
15 the material concern, which is neurotoxicity specifically.
16 So I think that needs to be clear in your summary and I
17 think we'll make it clear in ours if this is one of the ones
18 which is weak on two fronts.

19 Up until now you've actually presented a series of
20 chemicals which have one thing missing, but are strong on
21 one or the other, whereas this is actually weak on both.

22 PANEL MEMBER FUCALORO: Are you suggesting a third
23 tier?

24 (Laughter.)

25 PANEL MEMBER BLANC: No, no, I'm just saying that

1 of all the ones we've heard so far, I mean I think there are
2 others in tier 2 which are also weak on both counts. But,
3 again, I'm just saying that this -- again, I'm trying to
4 return to the systematic way of looking at the data you're
5 presenting.

6 The only other way in which one could indirectly
7 look at methyl bromide I think would be by trying to look at
8 the literature on differential effects of bromine -- not
9 bromine gas, but bromide, I should say. And I wonder
10 whether or not you did that, because, you know, one of the
11 thoughts about methyl bromide is that it just happens to be
12 a very effective delivery mechanism for bromide to the CNS.

13 SUPERVISING TOXICOLOGIST MARTY: I don't know how
14 much staff looked at that. I do know that there was one
15 paper that we pulled that indicated, and I'm remembering it
16 was an in vitro system that methyl bromide was more potent
17 than the equivalent amount of bromide ion in the system, and
18 it was an in vitro nervous tissue --

19 PANEL MEMBER BLANC: Right, but I was asking a
20 different question, which would be just bromide exposure of
21 neonates versus adults, a propos, bromism, and that would be
22 a very indirect argument, but I think it wouldn't be
23 irrelevant to the issue. And I only bring it up because
24 your data are otherwise so -- there aren't any other data
25 that suggest any differential neurotoxicity.

1 I'm actually not aware of -- you know, bromism,
2 historically, was an illness in adults, not in children, for
3 medication sources.

4 CHAIRPERSON FROINES: Just two quick questions.

5 One, just a rejoinder to Paul, CPDA is not simply
6 peripheral it's also central. So I think one can't just
7 simply think of hexane as a peripheral, even though that's
8 obviously the manifestation that's most commonly reported.

9 Secondly, the -- are you familiar with any studies
10 of DNA methylation with respect to methyl bromide?

11 SUPERVISING TOXICOLOGIST MARTY: Only the genotox
12 studies that have been done and it's a bazaar compound
13 because it's genotoxic in several assays, but nobody can
14 seem to get tumors in the carcinogenicity studies.

15 PANEL MEMBER FUCALORO: Go ahead.

16 SUPERVISING TOXICOLOGIST MARTY: The sixth
17 chemical that came up is methylene chloride and the reason
18 for concern is that methylene chloride is metabolized to
19 carbon monoxide and that people poisoned by methylene
20 chloride have elevated levels of carboxyhemoglobin.

21 We also have concerns about hotspots releases and
22 therefore potential near source exposures. However, if you
23 look at the ambient data, which has been collected by the
24 Air Resources Board monitoring network, the measurements are
25 very low. Our chronic reference exposure level is based on

1 the formation of carboxyhemoglobin in adults and if you
2 ratio the ambient air concentrations to our chronic REL, the
3 ratio is .005. So we're pretty far away from our chronic
4 REL, which does have an uncertainty factor built into it for
5 intra-human variability.

6 CHAIRPERSON FROINES: Are there hotspots
7 measurements?

8 SUPERVISING TOXICOLOGIST MARTY: There are lots of
9 emissions. It's in the millions of pounds per year, but we
10 don't have any indications of what the concentrations are
11 around most of those facilities. We have a few facilities
12 that reported some years back and did a risk assessment, and
13 methylene chloride did not drive -- it was a cancer risk at
14 the time that was being looked at, so it's kind of hard to
15 tell by looking at those numbers.

16 CHAIRPERSON FROINES: I think looking at methylene
17 chloride from the standpoint of ambient concentrations may
18 not be the best way to do it, because, you know, there's a
19 million furniture refinishing shops that use methylene
20 chloride as a paint removal aid, stripper. So that probably
21 the problem is more localized, rather than general.

22 Go ahead, sorry.

23 SUPERVISING TOXICOLOGIST MARTY: We know that
24 lowered birth weights in humans have been associated with
25 prenatal exposure to carbon monoxide.

1 We also know from an animal study that prenatal
2 exposure to methylene chloride can cause slower behavioral
3 adaptation in pups at ten and fifteen days and altered
4 activity levels in adulthood. So that's a prenatal exposure
5 that produces an effect. It's fairly high concentrations.

6 We also looked at a study, Anders and Sunram, that
7 evaluated the carboxyhemoglobin levels in the animals at
8 maternal exposures to 500 or so ppm of methylene chloride
9 and they got equivalent carboxyhemoglobin levels in the rat
10 fetuses if the moms were exposed to 22 ppm or to 500 -- 22
11 ppm carbon monoxide or to 500 ppm methylene chloride.

12 This slide basically just says that in a picture
13 that the equivalent carboxyhemoglobin levels are produced in
14 the fetal tissues with 22 ppm CO and 500 ppm methylene
15 chloride on the left. And then on the right are measured --
16 I think these are maximum measured levels of both CO and
17 methylene chloride in 1999 and you can see that the max for
18 CO was 23 ppm and the max for methylene chloride was 4.8
19 parts per billion.

20 So in terms of the overall contribution to
21 carboxyhemoglobin that we might get from ambient levels of
22 methylene chloride, it's pretty tiny compared to carbon
23 monoxide.

24 CARB has one analysis where they looked at the
25 statewide median in maximal monitored ambient methylene

1 chloride levels and I think this is 2000 data, it was .5 was
2 the median and 4.8 was the maximum measurement. So the
3 median levels are actually quite a bit lower than that one
4 max, another order of magnitude down.

5 But we do have information that 8,000 tons or so
6 were released in 2000 and that's actually up from the data
7 that we had in '98.

8 PANEL MEMBER BLANC: That's not the hotspots -- or
9 that is the hotspots data?

10 SUPERVISING TOXICOLOGIST MARTY: Yes, it's the
11 hotspots data. It's tons per year.

12 And then, of course, the chronic REL, just for
13 reference is 100 ppb. So compared to ambient we're pretty
14 -- we're down there.

15 CHAIRPERSON FROINES: Do you know what the levels
16 were in Mates II?

17 SUPERVISING TOXICOLOGIST MARTY: In the Mates II
18 study, I don't remember. It was probably -- I don't
19 remember.

20 Okay. So in summary, the rat studies show it
21 takes, you know, in a rat about 20 times or more methylene
22 chloride to get the same amount of carboxyhemoglobin as
23 carbon monoxide.

24 We do know that carboxyhemoglobin is a serious
25 problem for humans in terms of impacts on growth rate in

1 utero and, therefore, birth rate. The highest measured
2 ambient was 4.8 ppb and we certainly consider methylene
3 chloride to be an occupational hazard. I'm not sure I would
4 have put accidental release.

5 The issues are it's definitely an occupational
6 hazard and we have concerns about hotspots exposures, but we
7 don't have data measuring concentrations near source. So
8 it's something that we think the Air Board should look at.

9 PANEL MEMBER BLANC: So again here to summarize,
10 the issue is completely based on exposure, because there's
11 absolutely no question that methylene chloride is
12 metabolized to CO and that there's a preferential childhood
13 effect of CO. So here the entire argument rests on exposure
14 potential, is that a safe summary.

15 SUPERVISING TOXICOLOGIST MARTY: I think that's a
16 safe summary. In fact, if you look at the data it appears
17 you need pretty high concentrations of methylene chloride to
18 get significant carboxyhemoglobin. And there are reports of
19 women who were pregnant poisoned by methylene chloride.

20 PANEL MEMBER BLANC: Well, and the other issue is
21 that there's another nuance to that, which is that, as with
22 arsenic where the issue is there are other sources of
23 exposure, here we're talking about an additional source of
24 carbon monoxide exposure and we know that people have an
25 ambient source of exposure to carbon monoxide, independent

1 of methylene chloride --

2 PANEL MEMBER GLANTZ: You mean carbon monoxide --

3 PANEL MEMBER BLANC: Carbon monoxide, I'm sorry.

4 So the issue is the contribution -- the potential additive
5 contribution of methylene chloride to a child or neonate or
6 fetal exposure that might be occurring through inhalation of
7 carbon monoxide to begin with, with superimposed methylene
8 chloride?

9 SUPERVISING TOXICOLOGIST MARTY: Yes.

10 PANEL MEMBER BLANC: I think that's important to
11 put in context.

12 CHAIRPERSON FROINES: There's an important issue.
13 I suspect you're familiar with the epidemiologic work that
14 Beate Ritz has done in the last few years, specifically on
15 reproductive outcomes of one low birth weight, low term and
16 what have you relative to carbon monoxide levels?

17 SUPERVISING TOXICOLOGIST MARTY: Yes, we actually
18 cited one of her papers.

19 CHAIRPERSON FROINES: Yeah, I saw that. And it
20 seems to me that one should actually look at her work
21 because it is so current and then ask the question, given
22 what we know about methylene chloride, would it demonstrably
23 increase the risk associated with the risk that she's
24 demonstrated at extremely low levels of carbon monoxide?
25 It's really a follow-up to what Paul is saying, because her

1 work clearly -- I won't say that, that gets you into
2 trouble.

3 Her work suggests that -- and my feeling is that
4 her work indicates an association with carbon monoxide,
5 however, that association may be a surrogate for something
6 else. As we know, that with air pollution lots of times
7 what we measure turns out to be a surrogate for some other
8 toxic exposure. But on the assumption that the carbon
9 monoxide relationship that she's identified has meaning then
10 it would make sense to look and see what the increased risk
11 would be associated with an additional exposure associated
12 with carbon monoxide.

13 PANEL MEMBER BLANC: So really we have two issues
14 that are sort of emerging thematically. One is that there
15 appears to be, and I think our findings will have to reflect
16 this, there appears to be a series of priorities for the Air
17 Resources Board to obtain data that relates particularly to
18 hotspot emissions and correlating hotspot emissions that are
19 reported in pounds with neighborhood air concentrations as
20 opposed to ambient California air levels statewide.

21 And the second issue, in certain cases are
22 situations in which perhaps sophisticated risk modeling in-
23 house that your group could do, be it the contribution of a
24 certain percentage of methylene chloride superimposed on
25 ambient carbon monoxide or similarly a small amount of

1 ambient manganese or arsenic, and I guess we'll come up
2 again with mercury.

3 Because the third arm of it is not something
4 that's within your control, which is, you know, people need
5 to go out and do, you know, differential epidemiologic or
6 toxicologic studies in newborns or infants versus adult
7 animals or humans. I mean that's not something that we can
8 do anything about. But these other two areas are areas in
9 which there can be some movement, I think.

10 SUPERVISING TOXICOLOGIST MARTY: Yes, we can do
11 that. I'm not speaking for ARB.

12 That summarizes the six chemicals that came up at
13 the last meeting. I should add that Dr. Froines asked us to
14 make sure we'd gotten everything that was available on a few
15 other chemicals and we did do that.

16 CHAIRPERSON FROINES: Dr. Froines actually
17 emphasized very strongly the issue of naphthalene and so
18 that was one that I feel pretty strongly about. But in a
19 telephone conversation with Melanie she suggested that the
20 methemoglobin issue was not a major issue, but that was a
21 question.

22 SUPERVISING TOXICOLOGIST MARTY: It was a
23 question. We did look at the literature on methemoglobin
24 anemia in kids who had basically eaten mothballs and it
25 looked like the concentrations of naphthalene these kids

1 were exposed to were in the gram per kilogram range. So
2 pretty high concentrations.

3 We also actually -- Dr. Witschi brought up Charlie
4 Plopper's work, the work that's going on in Plopper's lab,
5 looking at naphthalene and Clara cell toxicity. And this is
6 in post-natal and also adult mouse, showing that the Clara
7 cell toxicity is elevated in the younger mice relative to
8 the older mice.

9 So that's a very interesting area that's emerging.
10 These concentrations are milligram per kilogram body weight
11 range, so 20 all the way to 100, I think, were their doses.
12 So these are, relatively speaking, high doses. And, Andy,
13 was it -- it's inter-peritoneal?

14 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

15 SALMON: Well, in general terms, I think this is IP.

16 CHAIRPERSON FROINES: You're going too fast. Go
17 back to the -- can you help us understand this?

18 SUPERVISING TOXICOLOGIST MARTY: Okay, we're
19 trying to get the staff person that just put these together.
20 He stepped out for a second. But what Plopper's lab is
21 doing is looking at naphthalene toxicity in the lung and
22 looking at the difference between young animals and older
23 animals. So, John, maybe you can explain --

24 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

25 SALMON: Dr. Budroe is going to take over at this point.

1 DR. BUDROE: Yes, basically you can see that
2 there's a substantial difference in -- actually that should
3 be ciliated respiratory epithelial toxicity in both seven
4 and fourteen day old mice as compared to adults. And that's
5 especially interesting because adult mice have roughly two
6 and a half times the metabolic activation capability of
7 seven and fourteen day old mice.

8 So the effective dose --

9 CHAIRPERSON FROINES: You mean p450 in those Clara
10 cell?

11 DR. BUDROE: Correct. So the effective dose to
12 the adult mice is actually greater than the effective dose
13 to the neonatal mice. The toxicity you see in those
14 ciliated epithelium cells is greater in the seven and
15 fourteen-day-olds than in the adults.

16 The other interesting thing is, good point, that
17 that's an IP dose. So what you're seeing isn't a contact
18 effect, that's actually a systemic effect that's being
19 expressed as lung toxicity.

20 CHAIRPERSON FROINES: Well, in that sense you know
21 that work -- they're arguing that the p450 metabolism to the
22 naphthaquinones is occurring in the liver and not in the
23 lung, and so that this is a little more complicated than
24 you're describing.

25 DR. BUDROE: Okay, the second slide is looking at

1 Clara cell toxicity in the different parts of the
2 respiratory tract architecture. And you see a little bit of
3 toxicity in the 14 day old mice compared to the lobar
4 bronchi in the seven day old mice.

5 And what's actually happening is, you can see a
6 change in compartmentalization. You're seeing enormous
7 decrease in the nonciliated Clara cells and an increase in
8 the compartment evacuated cells, which are essentially
9 damaged cells.

10 So you're seeing this effect in a number of
11 different respiratory epithelium cell types and in the
12 number of different locations in the respiratory tract.

13 CHAIRPERSON FROINES: Following Paul's suggested
14 approach, this meets both your criteria.

15 PANEL MEMBER BLANC: I guess you'd say that
16 because this is one study there's not an extensive database
17 for differential toxicity. Although I will say -- well,
18 first let me ask a clarification. We've gone through a
19 series of presentations previous to this, substances that
20 your intent is to recommend for tier 2. We're going to come
21 back later to how the documentation is going to be handled
22 for the tier 2 substances so that it's consistent.

23 But leaving that aside, the naphthalene that
24 you're just discussing now, could you clarify for me, is
25 that something which you're also planning to propose as tier

1 2 or your argument here is why -- is this a clarification as
2 why it's not coming to tier 2?

3 SUPERVISING TOXICOLOGIST MARTY: We've actually
4 include naphthalene under PAHs, which is being recommended
5 for tier 1.

6 PANEL MEMBER BLANC: Okay, so this will fall into
7 the tier 1 argument.

8 The other issue I would say, and why it may be
9 helpful to have this two column evaluation, the issue as to
10 what the dose is that induces methemoglobin in the case
11 reports of an ingestion of naphthalene is not the issue.
12 That's an issue in terms of whether exposure levels would
13 ever get up to that area.

14 SUPERVISING TOXICOLOGIST MARTY: Correct.

15 PANEL MEMBER BLANC: So your issue in pediatric
16 sensitivity is, in fact, if you gave an adult versus a child
17 the same amount in milligrams per kilogram, whatever that
18 dose is, would children develop more methemoglobin anemia
19 and that's the data you have to look at, based on the
20 function of methemoglobin reductase. I'm assuming that
21 infants would be more sensitive.

22 So from a pure biological point of view you have,
23 I believe, put emphasis really parallel to the carbon
24 monoxide issue in fetal hemoglobin binding of carbon
25 monoxide. It's really not a biological issue. Then you're

1 completely pushed in to the exposure issue and is there
2 significant exposure at levels where this would be a health
3 effect that would matter.

4 SUPERVISING TOXICOLOGIST MARTY: Yes, that's --

5 PANEL MEMBER BLANC: I just want to clarify that,
6 unless I've misunderstood.

7 CHAIRPERSON FROINES: Roger Atkinson measured
8 levels of naphthalene in Glendora at 3600 nanograms per
9 cubic meter, which isn't by any means trivial, and so how
10 does that exposure finding relate to what Paul is asking?

11 SUPERVISING TOXICOLOGIST MARTY: I'm not sure I
12 can relate it directly to the methemoglobin anemia issue
13 other than to say that from the case reports these kids had
14 really high exposures. We don't have information on what
15 would happen if adults had gotten the same type of exposure.
16 You know, at the microgram per cubic meter amount, you'd
17 have to inhale an awful lot to get up to the gram per
18 kilogram range.

19 We did -- if you'll recall our chronic REL, our
20 chronic REL is about at the levels that Dr. Atkinson is
21 measuring, so we did have some alarm bells go off then. And
22 that's actually based on, if I'm recollecting correctly,
23 respiratory epithelial damage end point.

24 CHAIRPERSON FROINES: I don't remember and I
25 suspect nobody else does, but did you use Charlie Plopper's

1 work in your REL determination?

2 SUPERVISING TOXICOLOGIST MARTY: No.

3 CHAIRPERSON FROINES: So that you probably have to
4 go back and relook at that number.

5 SUPERVISING TOXICOLOGIST MARTY: Yes.

6 CHAIRPERSON FROINES: Because it may be -- your
7 number may be too high relative to the current information
8 available to us.

9 SUPERVISING TOXICOLOGIST MARTY: Yes, we need to
10 go back and look at that.

11 PANEL MEMBER WITSCHI: I have a couple of
12 comments. First of all, that's not all there is to the
13 naphthalene story. There actually are data out there in
14 which they did inhalation. So if you're worried about high
15 milligrams per kilo, I don't know the numbers offhand, but
16 you might want to look up the inhalation study stated.

17 The other thing is there's really much much more
18 to the naphthalene story than just this one single paper.

19 SUPERVISING TOXICOLOGIST MARTY: We realize that.

20 PANEL MEMBER WITSCHI: Because if you look up the
21 whole body of work that has been done over the last ten
22 years then you have a tremendous lot of information in
23 difference species at different ages.

24 SUPERVISING TOXICOLOGIST MARTY: Yes, we're aware
25 of that.

1 CHAIRPERSON FROINES: This issue is very
2 troubling. I agree one hundred percent with Peter. And,
3 you know, it is one of the central elements of our research
4 in Southern California, because we identified fairly
5 significant quantities of naphthaquinones in the air in
6 Southern California. And that has a great deal of
7 significance with respect to the production of reactive
8 oxygen species. And so that we think naphthalene is an
9 extremely important compound from the standpoint of
10 respiratory effects in children.

11 You know, sometimes it's good to be a lumper and
12 sometimes it's good to be a splitter. And in this case to
13 lump naphthalene at 3600 nanograms per cubic meter with
14 Benzo[a]pyrene at half a nanogram per cubic meter is really
15 a complete misunderstanding of a different significance.

16 I mean Benzo[a]pyrene is an important carcinogen,
17 yes, that's fine, but the reactive oxygen chemistry
18 associated with naphthalene is so important that, for us to
19 just kind of lump it with all the other PAHs, including
20 things like pyrene which obviously have very low toxicity is
21 really problematic I think.

22 I don't think we can just put naphthalene in there
23 with PAHs and kind of then forget it. It's just too
24 important at this point. And our work is showing it and
25 Charlie Plopper's work is showing it, you know, and so I

1 think we need an alternative approach to this one.

2 DR. ALEXEEFF: George Alexeeff. We haven't quite
3 gotten to the point of maybe discussing, you know, how
4 chemicals should be expressed on Tier 1 and Tier 2. But I
5 think our point was we felt there was enough evidence for
6 this and other PAHs to be in Tier 1. Okay, that was our
7 bottom line point.

8 Specifically what we were thinking, again we
9 haven't gotten to this part, but just to address your point,
10 under polycyclic aromatic hydrocarbons we felt we should
11 specifically list those for which there were data and this
12 is one of those that are listed. And then that might
13 address your concern.

14 So it's not hidden, it's specifically listed as a
15 chemical.

16 CHAIRPERSON FROINES: So that within the document
17 on PAHs that we may have some sections that address specific
18 toxicities associated with particular chemicals?

19 DR. ALEXEEFF: Yes.

20 PANEL MEMBER BLANC: That being said, that's
21 reassuring, that we're going to come back to the draft
22 potential structure of our findings, but since that draft, I
23 assume, represented a collaboration trying to emphasize the
24 key points, you know, naphthalene under five, naphthalene
25 doesn't receive any mention whatsoever separately, nor are

1 the airway effects of it mentioned. So the findings vis-a-
2 vis polycyclic aromatic hydrocarbons are driven largely by
3 the carcinogenicity issue and then --

4 SUPERVISING TOXICOLOGIST MARTY: That's why it's a
5 draft.

6 PANEL MEMBER BLANC: I know, I'm just saying,
7 let's bear this in mind, because --

8 SUPERVISING TOXICOLOGIST MARTY: Yes.

9 PANEL MEMBER BLANC: -- I support the statement
10 you made, but let's make sure that we're consistent.

11 PANEL MEMBER WITSCHI: And I think it's getting
12 messy. You know, we are dealing with categories on one
13 side, and we are dealing with something else on the other
14 side. Why don't we just deal with PAHs, with metals, with
15 organic chlorine compounds, with all those kind of things.
16 It's really getting messy.

17 CHAIRPERSON FROINES: Believe me, if we could do
18 that we would have done it two meetings ago, because some of
19 us are big believers in aldehydes.

20 (Laughter.)

21 CHAIRPERSON FROINES: And we would have had an
22 aldehyde category, but I've asked that question of George
23 and Melanie 25 times and been told and spanked each time,
24 saying, no, we can't deal with classes of compounds, with
25 the exception of PAHs, because --

1 PANEL MEMBER WITSCHI: What's the reason for that
2 one?

3 CHAIRPERSON FROINES: -- because of the fact that
4 we can only list compounds that are listed as TACs and PAHs
5 are listed as TACs and aldehydes as a class are not.

6 PANEL MEMBER WITSCHI: I'm sorry, this might be
7 before my time, when did they list PAHs as TACs? I do not
8 recall having done PAHs.

9 CHAIRPERSON FROINES: That's a good point.

10 DR. ALEXEEFF: We presented a document to the
11 panel in '93 on Benzo[a]pyrene and a number of polycyclic
12 aromatic hydrocarbons. At that same time another law
13 required us to adopt all of the hazardous air pollutants
14 that UCPA listed as TACs and that's how really all the other
15 PAHs came in. And then so in the actual listing that the
16 Air Resources Board did they listed it under the great group
17 of POM, polycyclic organic matter.

18 CHAIRPERSON FROINES: We had POM under the HAPs,
19 the Clean Air Act amendments in 1990 and then we had BAP,
20 but we listed maybe 20 other compounds in terms of their
21 relative potency, so in a sense we -- you're right though,
22 you're absolutely right. It was BAP that we formally
23 listed.

24 Let's take a ten-minute break.

25 PANEL MEMBER GLANTZ: Before we do that, are we

1 going to go on and start talking about the findings next?

2 CHAIRPERSON FROINES: No, we're going to go on and
3 talk about the documents -- I believe we're going to talk
4 about the revised documents that they've submitted to us.

5 PANEL MEMBER GLANTZ: Okay, can I just say one
6 word about the findings, what we handed out, just so people
7 are looking at it. Just so the people on the Panel know
8 kind of what we had in mind when I worked with OEHHA on the
9 findings.

10 It was clear that the final list of five hadn't
11 yet been determined. So the way the findings are
12 structured is finding number one, which you notice has a big
13 blank, is what are the five. And then the next ones are
14 little summaries, which can be edited and amended by the
15 Panel, of the seven compounds that it seemed to us were
16 likely to be the ones from which the top five emerged.

17 So what we would do in addition to any editing of
18 those is two of those will be removed from the top five.
19 Then what is now finding number eight is a place to say
20 basically that there's several other toxic air contaminants
21 that are of particular concern, which are what we've been
22 calling Tier 2 and then we've done little similar summaries
23 for those. And if someone wanted to move one of those up to
24 Tier 1 I guess we could.

25 And then I also included, in light of the

1 extensive discussion at the Panel, just a comment about
2 environmental tobacco smoke.

3 The one thing that isn't in here that we had
4 discussed that we'd all forgotten about was a comment about
5 pesticides and if we want to add that somebody will have to
6 draft something up. So just when you're looking at it, just
7 -- because this is a little different than the way they
8 usually come back.

9 CHAIRPERSON FROINES: So the plan right now -- let
10 me just, before we take a break, the plan right now,
11 Melanie, is one, you're going to go over the chemicals that
12 you have supplied, revised documents. Then we're going to
13 have a comment or two, I think from Colleen on some legal
14 issues with respect to the determination of how we proceed,
15 given that these documents have not been sent out for public
16 comment and issues of deciding what are five or what are ten
17 or whatever. And then we'll go and discuss the findings.

18 And the other thing that I want to mention here is
19 that since there are only five Members of the Panel, the
20 administrative matters on the agenda I think we'll defer to
21 a future meeting when the number of panelists are -- when we
22 have a more full panel. I'd rather discuss administrative
23 issues when we have everybody here.

24 So that's the plan for the day unless you have a
25 major disagreement or the Panel does.

1 PANEL MEMBER BLANC: The only thing I'd add to
2 that is that in the context of that first discussion to get
3 a sense from you of your plan for dealing with the written
4 documentation for the other Tier 2 chemicals for which there
5 is not yet written documentation. So that I'm quite
6 concerned that we have a consistent and symmetric approach
7 in terms of the written documentation.

8 SUPERVISING TOXICOLOGIST MARTY: Okay.

9 CHAIRPERSON FROINES: Let's just break for about
10 ten minutes.

11 (Thereupon a recess was taken.)

12 DR. ALEXEEFF: What we thought we would do next
13 is, there are some -- after presentations and some
14 discussion and some feedback from the Panel there are some
15 chemicals that were on our group of 11 that we feel really
16 are not likely to make this list of five, so we thought we
17 would just try to take those off the table now just to let
18 you know which ones we don't think are necessarily in play
19 anymore, just to help focus our discussion.

20 They would remain in Tier 2 and we'll explain
21 briefly why.

22 CHAIRPERSON FROINES: And we'll come back later to
23 the issue of comment period and review and all of that.

24 DR. ALEXEEFF: Right, we'll come back later for
25 that.

1 And then we also need to make a presentation in
2 dioxin, which will be a little bit lengthy because the Panel
3 hasn't heard the dioxin information.

4 CHAIRPERSON FROINES: And following Paul's --
5 what's that word, paradigm, it can be hopefully focused so
6 it doesn't have to be too long.

7 DR. ALEXEEFF: Well, there just is a lot of data.

8 CHAIRPERSON FROINES: The Panel will clearly be
9 interested in the second half of the paradigm on exposure.

10 SUPERVISING TOXICOLOGIST MARTY: For the proposed
11 Tier 2 TACs that we think just won't make the top five, I'd
12 just like to just briefly go over which chemicals those are
13 and why we don't think they'll make the top five, and I
14 don't have overheads for this.

15 But vinyl chloride we think has sufficient data
16 based on the studies done looking at increased potency in
17 neonatal animals. But there's not enough exposure to shout
18 about, so it's one of those where, if there were, for
19 example, if there was a great big facility that was emitting
20 lots of vinyl chloride then we would definitely be
21 concerned, so we encourage continued tracking of vinyl
22 chloride emissions in the state, but at this point we think
23 it should stay in Tier 2.

24 Noncoplanar PCBs are another group that we think
25 has actually fairly strong evidence for differential

1 effects, but there are virtually -- well, I wouldn't say no,
2 but there are extremely limited airborne emissions and most
3 of the PCBs are out there in the environment in reservoirs,
4 and I don't mean water reservoirs, just from past production
5 and past distribution throughout the environment, and there
6 really isn't very much being emitted anymore directly.

7 Glycol ethers, we think have fairly strong
8 evidence, based on teratogenicity in animal models, but it's
9 unclear how much exposure there actually is. There are
10 significant hotspots emissions. These compounds are not
11 monitored routinely in the air, so we have very limited
12 information, in fact none, on what would be an ambient
13 concentration on any of the glycol ethers.

14 CHAIRPERSON FROINES: Melanie, on that issue, I
15 sent you a note a long time ago which was, you probably
16 didn't worry too much about, because of other priorities,
17 but I actually think it would be useful to go to the
18 semiconductor association and actually ask them how much of
19 those glycol ethers are still being used. Because clearly
20 in the seventies and early eighties the semiconductor
21 industry got rid of those compounds and it would be
22 interesting to find out to what degree do they think there
23 is still some use, because I think that would probably be
24 the area that would drive it. And I don't know how much
25 there is still used in printing and inks and other kind of

1 solvent mixtures and that's obviously information that's
2 harder to get a handle on.

3 The other comment I was just going to make on
4 glycol ethers is again in the occupational health world
5 there are numerous case studies of neurobehavioral effects
6 in 1973 when acetone was replaced with glycol ethers because
7 of the energy crisis, which is an interesting question given
8 our current energy crisis.

9 So that the neurobehavioral effects seem to me to
10 be still a very very important end point for glycol ethers.

11 SUPERVISING TOXICOLOGIST MARTY: Finally in Tier 2
12 we had mercury, which is a developmental neurotoxin in
13 humans. The evidence is extremely strong. I don't think
14 there's any debate about that.

15 The issue in California is fairly low airborne
16 exposures. There are significant exposures via food,
17 primarily fish, so that too, I think it's an important issue
18 in California, but it's more from a waterborne perspective,
19 much more, than an airborne perspective.

20 We do have some emissions in mercury from
21 incineration sources, including municipal waste
22 incinerators, which seem to be the largest sources of
23 mercury emissions, at least in the Air Board's database, and
24 also some emissions from hospital waste incineration.

25 Mercury still is definitely a cause for concern.

1 If there were more emissions it would be a slam dunk to be
2 in Tier 1.

3 PANEL MEMBER BLANC: Can you quantify then -- I
4 know you had a slide that you had prepared for the last
5 presentation. Can you quantify the amount of mercury in
6 those hotspot emissions?

7 SUPERVISING TOXICOLOGIST MARTY: Yeah, the total
8 is about 10,000 pounds statewide. Some of the bigger
9 sources, I know we have a slide -- okay.

10 Okay, we have a large resource recovery facility
11 in Long Beach. It's 688 pounds per year emitted and also
12 the commerce, municipal waste incinerator is about 108
13 pounds per year. And then we have a fairly large medical
14 waste incinerator in Sacramento with about 45 pounds per
15 year.

16 PANEL MEMBER BLANC: And are those the only
17 incinerators in the state of California?

18 SUPERVISING TOXICOLOGIST MARTY: No, there's
19 probably six or seven municipal waste incinerators and
20 there's a few, in fact, there's a medical waste incinerator
21 in Oakland, and I'm sure there's a few other medical waste
22 incinerators.

23 When the dioxin airborne toxic control measure was
24 developed, because medical waste was the source of dioxins,
25 it sort of changed the paradigm of how medical waste was

1 incinerated and went from numerous incinerators that were
2 poorly run, usually on site, to very large state of the art
3 facilities. So the total number is much smaller than it
4 used to be.

5 PANEL MEMBER BLANC: So the data that you have
6 here don't imply that there is no emissions from the others,
7 it's just that the others haven't been measured?

8 SUPERVISING TOXICOLOGIST MARTY: The others have
9 lower emissions. What we did was pick out a few of the
10 highest emitters.

11 CHAIRPERSON FROINES: And then the total -- and so
12 what's the total pounds per year in all of them?

13 SUPERVISING TOXICOLOGIST MARTY: The total from
14 the air toxics emissions database for 1998 was 10,000
15 pounds, all sources, not just incinerators.

16 PANEL MEMBER BLANC: And what's the total amount
17 of that from incinerators?

18 SUPERVISING TOXICOLOGIST MARTY: We have to add it
19 up. We have a list of a large number of incinerators, and
20 these include crematoria, because you're -- so I could have
21 somebody add that up and then we could give you the answer.

22 PANEL MEMBER BLANC: Well, I guess the reason why
23 I'm asking you the question is I want to get a sense similar
24 to the question with arsenic. Although it's slightly more
25 complex because -- in fact the original source of most of

1 the mercury that gets into the food chain through the fish
2 or a source of a lot of it could have been airborne
3 deposition, so it's airborne exposure that then eventually
4 gets to us through dietary exposure, although it's
5 historical, I suppose.

6 SUPERVISING TOXICOLOGIST MARTY: Yes. In
7 California actually probably the largest contribution has
8 been gold mining, because, we, you know, a hundred years ago
9 took the mercury out and used it in the gold mines, so it's
10 gotten virtually everywhere in the valley and even in
11 estuaries. And so with uptake by fish that's then the
12 source of human exposure.

13 PANEL MEMBER BLANC: So what percentage, you know,
14 we had talked earlier about the percentage of an airborne
15 source to the total that would be of concern. Have you got
16 some sense of what 10,000 pounds of mercury is
17 proportionally in terms of the exposure per capita to
18 mercury per year?

19 SUPERVISING TOXICOLOGIST MARTY: Well, we have
20 actually, the concentrations measured in ambient air are
21 around one to four nanograms per cubic meter, on that right-
22 hand column and those represent maximums for specific
23 monitoring network sites.

24 What did I just say? Did I not say mercury?
25 Whatever I said I meant mercury.

1 So it's about 30, say 30 nanograms per day at the
2 peak concentrations that have been measured.

3 We did an analysis under Proposition 65 looking at
4 consumption in kids of mercury from tuna and George is
5 trying to remember the number. It's quite a bit larger than
6 20 nanograms per day or 30 or even 40.

7 PANEL MEMBER BLANC: It would have to be -- I mean
8 if it was only ten times larger, if it was 200 nanograms per
9 day, for example, that would mean that you were getting ten
10 percent, so then that would be something worse. It was a
11 thousand times larger then it would mean that it's .1
12 percent and then we would sort of fall below our -- I mean
13 isn't this something that needs to be written out and
14 spelled out explicitly in your written documentation?

15 SUPERVISING TOXICOLOGIST MARTY: Yes, we will do
16 that.

17 PANEL MEMBER BLANC: But it is your impression
18 that we're talking about some trivial percentage?

19 SUPERVISING TOXICOLOGIST MARTY: Yes.

20 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
21 SALMON: A lot of the dietary mercury from within California
22 it appears to -- I can't give you an exact number on this,
23 but it appears to ask to be coming by emissions which are
24 directly into water rather than directly into air in the
25 first place. And I believe I'm correct in saying, for

1 instance, that a major source of the contamination of Clear
2 Lake, for instance, which is known to have a mercury
3 problem, is a result of mine outflow.

4 So that, we need a quantitative figure on that.
5 But I think I'm correct in saying that that's the major
6 source, not emissions to air in the first place.

7 PANEL MEMBER BLANC: I mean I was asking an even
8 more simplistic question. I was just assuming that none of
9 the air then got into the fish, I was just assuming that
10 whatever --

11 SUPERVISING TOXICOLOGIST MARTY: Oh, no, there's a
12 certain cycling of mercury that happens that way. Yes, you
13 definitely get deposition.

14 PANEL MEMBER BLANC: I know, but I mean even if
15 you take that out of the equation, just in terms of people's
16 exposure via inhalation as opposed to their dietary
17 exposure, is it less than one percent?

18 SUPERVISING TOXICOLOGIST MARTY: I am remembering
19 it's less than one percent, but we can actually figure that
20 out at lunchtime and get back to you on that.

21 PANEL MEMBER GLANTZ: While you're eating your
22 tuna sandwich.

23 (Laughter.)

24 CHAIRPERSON FROINES: This is always the danger,
25 of course, in allowing yourself to get caught taking up

1 rules of thumb, because then they come back on you at some
2 point and you have to live with them.

3 But did you do a ratio of your REL for mercury
4 with your airborne concentrations?

5 SUPERVISING TOXICOLOGIST MARTY: Yes.

6 DR. ALEXEEFF: Looking at the doses that we
7 normally see, we'll double check this, but it looks like
8 it's about a 100 to 300 fold ratio between the oral
9 consumption, primarily through seafood versus inhalation.
10 But we can double check that here today -- well, certainly
11 in the document, we can put that in there.

12 PANEL MEMBER FUCALORO: There is no ratio of
13 ambient concentration to REL for mercury in your tape?

14 DR. ALEXEEFF: No.

15 SUPERVISING TOXICOLOGIST MARTY: I just discovered
16 that.

17 (Thereupon a short discussion was held
18 off the record.)

19 SUPERVISING TOXICOLOGIST MARTY: Okay, we're going
20 to get you that ratio also.

21 CHAIRPERSON FROINES: What was the airborne
22 concentration, George, do you remember?

23 DR. ALEXEEFF: About 20 nanograms per day.

24 SUPERVISING TOXICOLOGIST MARTY: Twenty to Thirty.
25 It's about one and a half to two, right in there, have been

1 the ambient average concentrations of the data that we could
2 get hold of. So if you assume X numbers of cubic meters per
3 day, you can get 20 to 30 nanograms.

4 PANEL MEMBER FUCALORO: You're not giving a
5 concentration, you're giving an uptake, is that right?

6 SUPERVISING TOXICOLOGIST MARTY: Right.

7 PANEL MEMBER FUCALORO: Okay.

8 SUPERVISING TOXICOLOGIST MARTY: That assumes, of
9 course, one hundred percent absorption, etcetera.

10 CHAIRPERSON FROINES: What's the airborne
11 concentration in the Los Angeles basin, for example?

12 SUPERVISING TOXICOLOGIST MARTY: 1.5 at North Long
13 Beach's monitoring station. Well, it's four at Riverside.

14 PANEL MEMBER FUCALORO: 4.0 nanograms per meter
15 cubed. And then what factor are you multiplying that or
16 dividing that by in order to get the nanograms per day?

17 SUPERVISING TOXICOLOGIST MARTY: Twenty cubic
18 meters per day, and that would be an adult exposure, per 70
19 kilograms.

20 PANEL MEMBER FUCALORO: And what does it come to?

21 SUPERVISING TOXICOLOGIST MARTY: So that would be,
22 if there was four nanograms per cubic meter and you were
23 inhaling 20 cubic meters and you assume 100 absorption then
24 you're getting 80 nanograms, at the peak site. And those
25 actually aren't the averages, those are the maximums

1 measured in a year, in one of the years, '98 it looks like,
2 at that particular monitoring site.

3 CHAIRPERSON FROINES: I don't want to get into
4 something that's more complicated, but what's the form of
5 the mercury that you generally think is released from these
6 sites? Is it mercury metal or is it mercury salt?

7 SUPERVISING TOXICOLOGIST MARTY: It's both. You
8 can get elemental mercury coming out of the incinerator's
9 vapor, as a vapor. You can also get chloride salts and
10 other salts.

11 CHAIRPERSON FROINES: And do you have any idea
12 what the particle size of the mercury is, of those
13 materials?

14 SUPERVISING TOXICOLOGIST MARTY: Well, for the
15 elemental, of course, it's just coming out, it escapes the
16 bag house completely. There is some collection of the
17 smaller particles in the bag houses, but not being an
18 engineer I don't know the efficiency with respect to the
19 size of the particle, nor do I know the size of the
20 particles that are coming out.

21 CHAIRPERSON FROINES: I'm just trying to think
22 about what the relative amount that's absorbed through the
23 gut or not absorbed through the gut, as the case may be
24 versus respiratory uptake.

25 I'm at a level of too much detail, so why don't we

1 go on.

2 Unfortunately this is an important issue, as we
3 all agree, because the evidence is so strong on the health
4 side.

5 SUPERVISING TOXICOLOGIST MARTY: Exactly.

6 Okay, in terms of the chemicals that were in the
7 original 11 that we wanted to move, we need to mention that
8 benzene was -- we got a lot of public comment on benzene and
9 we agreed in large respect with a lot of the comments and
10 think that the epidemiology evidence, which we were trying
11 to hang our hat on, is relatively weak, and that is the
12 evidence of parental exposure being associated with elevated
13 incidents of childhood leukemia.

14 So we have decided at this point, amongst
15 ourselves, that we would not recommend benzene for Tier 1.

16 CHAIRPERSON FROINES: Just one question and then
17 we can go on. You're comfortable with the differences in
18 childhood leukemia versus adult leukemia and benzene
19 associated leukemia, because that's obviously a key
20 question. Do you know what I'm say?

21 SUPERVISING TOXICOLOGIST MARTY: Yeah, I'm
22 probably not the right person to answer the question, but I
23 know -- yeah, we're not exactly, we're not willing to say
24 benzene doesn't induce leukemia in children, because the
25 type of leukemia kids get is different than the type of

1 leukemia seen largely in the benzene exposed workers. We're
2 not saying that. I don't think that that's something that
3 you would -- I don't think there's a lot of evidence that
4 therefore it doesn't influence rates of childhood leukemia.

5 PANEL MEMBER GLANTZ: This was the one where I was
6 quite impressed with the public comments and the arguments
7 that the evidence was not as strong as for other things. I
8 mean I don't think anybody is suggesting it be taken off the
9 list, you know, that it be dropped out of Tier 2. But I
10 think given that we can only pick five, I just think the
11 evidence on benzene is -- there are five other things where
12 the evidence is much stronger that it's important than for
13 benzene.

14 So I agree with OEHHA's recommendation. In fact I
15 think I suggested it at the last meeting or two ago.

16 SUPERVISING TOXICOLOGIST MARTY: We do have a
17 ratio of the ambient concentration. This is a statewide
18 ambient of 1.9 nanograms per cubic meter ratioed to our
19 chronic REL which is .09 micrograms per cubic meter and that
20 ratio is about .02. That's ambient, though, it's not near
21 source and the near source is what we were worried about.

22 The next thing we would like to do is the Panel
23 has not heard our presentation on dioxins yet, so --

24 PANEL MEMBER FUCALORO: Excuse me, were you going
25 to go through some of the -- I thought, and maybe you just

1 did, go through some of the chemicals that would not make
2 Tier 1, because I have here something that was handed to me
3 on acrolein. I was wondering if that was part of your
4 presentation?

5 SUPERVISING TOXICOLOGIST MARTY: That's going to
6 be part of the presentation coming up.

7 PANEL MEMBER FUCALORO: Oh, coming up. Okay. I'm
8 just kind of losing sight of where we're going at the
9 moment.

10 SUPERVISING TOXICOLOGIST MARTY: Peter, does the
11 Panel have the dioxin's handout? Okay, because that's what
12 we're going to do next.

13 (Thereupon a short discussion was held
14 off the record.)

15 SUPERVISING TOXICOLOGIST MARTY: Okay, so
16 following the dioxin presentation we're going to go and give
17 a very brief overview of the presentations you've already
18 heard on five other substances that we think are important,
19 as well as a comparison between acrolein and formaldehyde,
20 which was asked for earlier, if the Panel wants to hear it.

21 CHAIRPERSON FROINES: This has been in contrast to
22 earlier meetings, very relaxed and very well articulated and
23 I think in general, everybody is happy with the way the
24 meeting is going.

25 I think that the one thing I need to say at this

1 point is that it's five to twelve. We're going to have to
2 really move along because we don't have that much time and
3 we need time for deliberation. And so I really think that
4 you're going to have to hold down the length of the
5 presentation as much as possible, albeit trying to meet
6 Paul's criteria, you know, at the same time. And I'm one of
7 the worst persons on this of getting off on sidetracks and
8 so the Panel will also try and avoid going off.

9 Melanie, before you start on dioxin, could you
10 explain to the Panel why, if you have dioxins and coplanar
11 PCBs as part of your list, why you could not also include
12 noncoplanar PCBs?

13 SUPERVISING TOXICOLOGIST MARTY: We actually could
14 just list dioxins and PCBs. I think the issue is, for us,
15 that the environmental burden of dioxin-like PCBs is
16 significant enough to increase the overall impact of dioxin
17 and dioxin-like compounds. And what we're trying to do is
18 distinguish from the toxicology information that noncoplanar
19 PCBs do not act like dioxin. They have their own
20 developmental neurotoxicity.

21 PANEL MEMBER FUCALORO: Except if they're
22 polychlorinated, is that the issue?

23 SUPERVISING TOXICOLOGIST MARTY: It's the
24 planarity of the ring and it depends on where the chlorines
25 are in each --

1 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

2 SALMON: I'll be explaining this, I hope.

3 PANEL MEMBER FUCALORO: Because noncoplanar PCBs
4 are included here, right?

5 SUPERVISING TOXICOLOGIST MARTY: They're included
6 in the summary, yes, you're correct about that.

7 PANEL MEMBER FUCALORO: And I think the reference
8 is dioxin-like.

9 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

10 SALMON: No, there's two classes of PCBs, which I will
11 attempt to clarify.

12 PANEL MEMBER FUCALORO: Fair enough.

13 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

14 SALMON: Well, I'll try and run through this as fast as I
15 can consistent with clarity.

16 The presentation I'm giving you is about
17 specifically the dioxin-like compounds and we propose that
18 these be placed in Tier 1, because we had high concerns over
19 the toxicity about point source emissions and background
20 levels of some of these compounds and the potential for
21 bioaccumulation.

22 We found evidence of differential impacts on
23 children in relation to immunotoxicity, developmental
24 toxicity, endocrine effects and we also, since infants are
25 highly exposed via breast milk, we also have concern for the

1 established carcinogenicity of these compounds.

2 Although current air levels are low, air is the
3 primary transport medium for new emissions of dioxin-like
4 compounds. These compounds show extreme bioaccumulation and
5 environmental persistence and the current multi-pathway
6 exposures in California are high and may, in fact, exceed
7 the effects levels.

8 I'll explain briefly what I'm referring to here.
9 The dioxin-like chlorinated compound --

10 PANEL MEMBER WITSCHI: Just a question, how come
11 again we have a class of compounds?

12 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
13 SALMON: Because this -- we have listings of classes in the
14 toxic air contaminant program. We have actual listings of
15 dioxins and PCBs as named classes in the regulation. This
16 is also, as we were saying earlier, like the case of the
17 POM, which is the defining class for the polycyclic aromatic
18 hydrocarbons. Fortunately or unfortunately it's not, for
19 instance, listed in that way for reactive aldehydes or
20 something like that. That's the critical distinction.

21 However, we do actually have a problem with the
22 way the TAC listing is done for the dioxins, which is where
23 the PCBs basically cover two groups of chemicals which are
24 separate in their biological effect. And I'm referring
25 specifically here to the coplanar PCBs and not the

1 noncoplanars.

2 This is an example of a noncoplanar where the
3 presence of an ortho substituent results in the molecule
4 having a twisted confirmation in its ground state and this
5 results in different biological activity. So, for this
6 purpose I'm not referring to these compounds.

7 Sources of the dioxin-like compounds, especially
8 dioxins, include combustion, from various forms of waste
9 incineration, and there's currently considerable interest in
10 that backyard burn barrel situation. It's being looked at
11 by the US EPA and the Air Board.

12 There are some minor sources, such as metal
13 smelting and refining. Formerly bleaching of wood pulp was
14 an important source involving release to water, but this is
15 now much reduced due to the replacement of chlorine by other
16 bleaching agents.

17 There is also concern about contamination of waste
18 sites, old manufacturing sites. And in the case of
19 contaminated areas you may actually get resuspension of
20 dioxin containing particulates back into the air.

21 As far as the contamination of the environment is
22 concerned, obviously sediments form an important reservoir
23 of dioxins, and because of the bioaccumulation, the biota
24 are significantly contaminated, including both sport and
25 commercial fish and game and also meat and dairy foods and

1 human milk.

2 Most -- the figure of greater than 90 percent was
3 recently derived based on measurements by US EPA. More than
4 90 percent of the newly formed dioxins are released directly
5 into the air and then proceed to bioaccumulate in the food
6 chain. Food is the major source for both the PCB and dioxin
7 elements of this group.

8 And exposure begins at an early age with breast
9 feeding, where this study, Patandin, et al, '99 study was a
10 study of breast fed infants exposed to background
11 environmental levels of dioxins. And it's been calculated
12 that an average breast fed baby receives a considerable
13 excess of the dioxin-like compounds relative to what we
14 consider our current standard.

15 These amounts are expressed as what we're calling
16 TEQ. This is the dioxin equivalents using the toxicity
17 equivalency factors from the table. So the breast-fed baby
18 is receiving 40 pg per kilogram of dioxin equivalent per day
19 relative to the chronic or REL of 10 pg per day.

20 SUPERVISING TOXICOLOGIST MARTY: That's based on
21 measurements of dioxin equivalents in the breast milk.

22 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
23 SALMON: Also, as young children consume a greater
24 proportion of their diet in the form of dairy foods and
25 manufactured foods, which are important sources of dioxins,

1 they tend, actually, to receive rather higher exposures than
2 adults.

3 There were three times higher exposure of toddlers
4 than adults in the Patandin study. And the breast-fed
5 infants were receiving 50 times higher than the adults.

6 The effects of dioxin-like compounds, I'm going to
7 start by reviewing the effects, which have been shown in
8 humans. First, the immunotoxicology, the typical findings
9 are changes in the number and activity pattern of the T
10 cells and these changes at the cellular level are reflected
11 in functional changes in the antibody response. And both
12 the response to common diseases and also incidents of things
13 like ear infections, and also, interestingly enough, a lower
14 prevalence of allergic disease.

15 So this actually represents quite an interesting
16 disturbance of the normal development of the immune system.
17 This has been seen both in the -- for instance, the
18 Weisglas-Kuperus study. This addressed children exposed to
19 background levels.

20 And also, of course, in a more extreme form these
21 same effects are seen in infants exposed to high levels as a
22 result of specific contamination incidents, such as the Yu-
23 Cheng incident which is reported by Chao, et al.

24 PANEL MEMBER WITSCHI: I have a question, in any
25 of those upcoming human studies and the present ones, are

1 there any data which came out of the incidents in Seveso?

2 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

3 SALMON: Yes.

4 PANEL MEMBER WITSCHI: And could point those out
5 to me?

6 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

7 SALMON: I will do so.

8 There are suggestions of an adverse developmental
9 impact. These have been described by Patandin and
10 coauthors in terms of vastly reduced birth weight and
11 secondly reduced growth during the first three months of
12 life. And there's other reports also of similar effects.

13 This is the Seveso aftermath data that I wanted to
14 draw your attention to. Obviously there are a number of
15 reports now coming out on the survivors of the Seveso
16 incident. This one is of particular interest. It appears
17 that there's an alteration in the sex ratio of the offspring
18 of fathers exposed during childhood to the TCDD which was
19 emitted in Seveso.

20 So this is a developmental effect as a result of
21 childhood and it's impacting a parameter which is generally
22 considered to be an extraordinarily robust feature of normal
23 human reproduction. There's something going on here. We
24 don't understand the mechanism. It presumably is somehow
25 linked to perturbation of the endocrine system during the

1 development and maturation of the reproductive system.

2 But, beyond that, I don't wish to speculate as to
3 what the mechanism might be, but clearly this is a highly-
4 significant effect and it is a specific impact on children.
5 The fathers exposed in adulthood showed a much less
6 significant effect.

7 I'm going to attempt to briefly review the effects
8 of dioxin-like compounds on animals. There's clearly a very
9 large literature and I'm specifically picking only a few
10 recent reports and those reports which parallel the effects
11 I've pointed to in humans.

12 Firstly, immunotoxicological. Again, the changes
13 in the distribution of the T cells and changes in the
14 functional behavior of the immune system. These are in rats
15 exposed to TCDD in utero and via lactation. These effects
16 were persistent into adulthood well after the time at which
17 the dioxin to which the rats were exposed would have been
18 cleared by a metabolism and excretion. So this is a
19 persistent developmental effect.

20 Other reports showed similar findings, changes in
21 splenocyte, suppression of delayed type hypersensitivity.
22 And again these are, to a varying degree, persistent past
23 the immediate post-natal period into adolescence and
24 adulthood.

25 The developmental effects observed in rats include

1 a number of specific teratological changes in addition to
2 the more general growth retardation type of effects which
3 have been reported in humans. Interestingly, several of the
4 specific effects observed here actually do have implications
5 for the developing reproductive system.

6 Another teratological effect was the observation
7 of ototoxicity in rats exposed to a PCB mixture, A1254,
8 which includes a substantial dose of coplanar PCBs via the
9 lactational route.

10 This is a further report on the effects of dioxin-
11 like PCBs. Again we're seeing changes in both morphology
12 and function of the reproductive system. Also changes in
13 brain weight and reduction in serum testosterone. These
14 animals were exposed to specific coplanar PCBs in utero and
15 via lactation. And then these effects were assessed at
16 post-natal day 65 or 140, so they're consistent into
17 adulthood.

18 CHAIRPERSON FROINES: Can I just ask a quick
19 question. Based on the Seveso data and the sex ratios, has
20 anybody tried to do some animal experiments to look at that
21 issue?

22 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
23 SALMON: I'm not personally aware of such an experiment.

24 PANEL MEMBER WITSCHI: I would like to come back
25 to the Seveso incident, you know. I mean we have here one

1 set of data, a late effect. But at the time being this
2 created some excitement and are there no information
3 whatsoever available on what happened to this population
4 within the ten years, the first ten years after exposure?

5 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

6 SALMON: There's been a considerable amount of
7 epidemiological studies and --

8 PANEL MEMBER WITSCHI: What did they say with
9 regard to children?

10 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

11 SALMON: There are no other clear and unequivocal findings
12 that we could identify. There's a lot of interest about
13 issues like cancer and --

14 PANEL MEMBER WITSCHI: So if this acute high
15 exposure had done something substantially to children we
16 should know, but it didn't.

17 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

18 SALMON: Well, there are, unfortunately, some fairly major
19 problems with the epidemiology of the follow-up to the
20 Seveso incident. We're only now beginning to see things
21 coming out with a reasonable degree of certainty.

22 There were certainly some very significant causes
23 for concern when looking at incidences of cancer, including
24 leukemias and various measures of birth outcomes and so on.
25 But, unfortunately, I think at least partly due to the

1 epidemiological difficulties, we don't have clear
2 statistically unambiguous conclusions on all of those things
3 at this point.

4 So I think the general feeling is that there are
5 things going on in that population, but it has proved very
6 difficult to establish exactly what they are.

7 PANEL MEMBER WITSCHI: Well, this could mean two
8 things.

9 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
10 SALMON: There's a considerable folklore as to what the
11 nature of those difficulties are.

12 PANEL MEMBER BLANC: Well, let's put this in
13 context. In terms of our handout here of various slides on
14 this, we're actually quite -- you have three more slides
15 after this. So let's do those and then let's try to put it
16 into context.

17 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
18 SALMON: Right.

19 Okay, well, the report here is an interesting
20 finding. This is actually a finding of transplacental
21 cancer promotion. Rats were exposed to TCDD in utero. In
22 the 50-day offspring the first observation was
23 morphological. These were female rats and there were
24 morphological changes in the mammary gland.

25 But in addition to that the mammary gland was more

1 susceptible to the induction of mammary adenocarcinomas by a
2 single dose of DMBA. This graph actually shows the tumor
3 incidents, cumulative incidents after the single dose of one
4 microgram of DMBA.

5 This is a fairly small dose, and it's clear that
6 the TCDD rats, which were treated in utero are at 50 days
7 postpartum, still substantially more susceptible to the
8 tetranogenic effect. And at this point, 50 days postpartum,
9 we're not talking about residual TCDD here. We're talking
10 about some persistent biological change in, as noted
11 previously, the morphology, and evidently also the growth
12 regulation of the mammary gland.

13 I wanted to just briefly refer also to another
14 hormonal effect which is the thyroid hormones. The
15 noncoplanar PCBs are characterized by different impacts on
16 the thyroid hormones. But I'm going to concentrate on the
17 coplanar effect here.

18 There is, in fact, a reduction in T3 and T4
19 levels, both in the -- well, this report is specifically in
20 humans, but this is a response to the PCD, the dioxins, the
21 dibenzofurans and the coplanar PCBs. It's not, I think, 100
22 percent clear what the mechanism is, but it may involve
23 actually enhancement of T3 metabolism, and that effect has
24 also, I think, been reported in animal studies.

25 But, anyway, I'd like to summarize what we know

1 about the mode of action. The important message here is
2 that there is a so-called dioxin-like mechanism at work with
3 all these compounds which involves interaction with the so-
4 called Ah receptor. This triggers a suite of biochemical
5 responses which includes cyt P450 induction. The iso
6 enzymes varying with tissue and species.

7 That receptor interaction is also accompanied by
8 characteristic toxic responses, specifically immunotoxicity,
9 teratogenesis and carcinogenesis. And these correlate with
10 the interaction with that receptor.

11 Some of the other toxic effects do not correlate
12 strictly with the Ah interaction, so there's a possibility
13 that there's another receptor which responds to dioxin-like
14 compounds or possibly an indirect mechanism, which may
15 involve endocrine effects.

16 But we consider that all these aspects of the mode
17 of action have important implications for the both pre and
18 post-natal development of infants and children.

19 And that's the end of my presentation.

20 PANEL MEMBER BLANC: Well, two things strike me
21 about this presentation. One is that considering that
22 dioxin is one of the most elaborately studied toxins in
23 literature, the direct evidence of preferential childhood
24 effects as opposed to the fact that children as well as
25 adults have effects of dioxin is not overwhelming. There's

1 just a lot of interesting stuff which would have
2 implications for children, more or less, if you had to
3 characterize the bulk of the literature, and that's
4 particularly so for the epidemiological literature.

5 But the second thing that was even more striking
6 from your presentation is I don't see anything here on the
7 second axis, which is current airborne release. I
8 understand the point that historically most of the dioxin
9 and dioxin-like substances which are in our food chain
10 originally got there through various airborne sources,
11 although some of them probably did get there through
12 waterborne sources, particularly from the bleaching in the
13 pulp industry, where that was a waterborne release, by and
14 large, not an airborne release.

15 But where -- in the written document I see that
16 there are approximately 60 pounds a year that were released
17 in 1996, something under 60 pounds in California. I mean
18 these statements are not adequate it seems to me to
19 address -- a lot of these are historical issues. Where is
20 the evidence to say that there is currently --

21 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
22 SALMON: There are current and ongoing releases of dioxins.

23 PANEL MEMBER BLANC: You're showing less than 60
24 pounds a year. Compared to the amount that's in the
25 environment currently, that's certainly not a biologically

1 significant contribution, is it?

2 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

3 SALMON: I think actually -- well, given how long these
4 materials persist, and I think it seems that the level of
5 dioxin release into the environment has been reduced
6 somewhat, but when we're talking about the current level of
7 airborne releases, I think that is generally still
8 considered to be of concern in relation to not adding to the
9 already excessive burden.

10 PANEL MEMBER BLANC: Why doesn't that argument
11 apply to mercury then where you have, what is it, a thousand
12 pounds of mercury going up into the air?

13 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

14 SALMON: It's difficult to compare -- I mean a thousand
15 pounds of mercury is not toxicologically equivalent to a
16 thousand pounds of dioxins.

17 PANEL MEMBER BLANC: No, what about 50 pounds of
18 dioxin?

19 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

20 SALMON: No, we're talking about a great many orders of
21 magnitude difference in terms of the effective
22 concentrations here. We're talking in the dioxin case about
23 being concerned about exposures down to the pico- or
24 fentogram level.

25 PANEL MEMBER FUCALORO: Then that should show up

1 in the ratio, right?

2 SUPERVISING TOXICOLOGIST MARTY: We don't have
3 ambient concentrations and dioxin is just too expensive to
4 monitor.

5 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

6 SALMON: Yeah, that is an additional --

7 PANEL MEMBER WITSCHI: I think to bring dioxin
8 down to the -- I mean guinea pigs are not humans and the
9 other way around. We know this. I wouldn't be so sanguine
10 in declaring dioxin the ultimate and most important and
11 toxic agent that's around.

12 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

13 SALMON: It's not clear exactly how potent it is in humans
14 and it may be true that humans are less susceptible than
15 some experimental animals, but I think that we are concerned
16 about the levels currently being emitted.

17 SUPERVISING TOXICOLOGIST MARTY: I think that a
18 very important point that needs to be hammered home is that
19 breast milk is the major source of exposure to a chemical
20 which we know has impacts in infants and children. We are
21 exposed in the first year of life to more than we're ever
22 exposed to in the whole rest of our lifetime. To me that's
23 enough to put it on the list.

24 PANEL MEMBER BLANC: No, it's not, unless there is
25 air -- I mean --

1 SUPERVISING TOXICOLOGIST MARTY: There are
2 airborne exposures.

3 PANEL MEMBER BLANC: No, airborne exposures which
4 are a significant part of the source of the current total
5 exposure. I mean that's why I'm trying to be consistent,
6 I'm trying to understand this in the context of mercury, for
7 example, or arsenic.

8 SUPERVISING TOXICOLOGIST MARTY: All of the stuff
9 that's in the environment now, the vast majority of it came
10 from airborne exposures.

11 PANEL MEMBER BLANC: Okay, and where did --

12 SUPERVISING TOXICOLOGIST MARTY: Okay, we've cut
13 back on that, which is a good thing.

14 PANEL MEMBER BLANC: I'm not arguing with that.

15 SUPERVISING TOXICOLOGIST MARTY: But we still have
16 sources and, in fact, both ARB and US EPA are doing a lot of
17 work right now to further characterize the sources of
18 dioxins. So I think it's a very important issue.

19 There is some modeling work, which unfortunately I
20 don't have in front of me, that was done by the Regional
21 Water Quality Control Board, after the San Francisco Bay was
22 declared impaired because of dioxin contamination of fish,
23 which indicated that the sources of dioxins to the Bay were
24 motor vehicles. That's the primary source of dioxins in the
25 San Francisco Bay.

1 Now it's a model and so, of course, it's not
2 exactly correct, but it's a pretty interesting indication
3 that airborne exposures are continuing to contaminate the
4 environment and that we already have an environment that
5 is --

6 PANEL MEMBER BLANC: But couldn't I say exactly
7 the same thing about mercury? Couldn't I say exactly the
8 same thing about mercury and how am I supposed to decide
9 proportionately comparing the 50 pounds per year as in --

10 SUPERVISING TOXICOLOGIST MARTY: Well, I don't
11 think that the stuff that's in the fish now, the mercury in
12 the fish, it wasn't originally airborne.

13 PANEL MEMBER BLANC: Yeah, but we're not talking
14 about history, we're talking about what the future --

15 SUPERVISING TOXICOLOGIST MARTY: I think we're
16 talking about both. I'm talking about both anyway, because
17 I just think that the airborne exposures from dioxin are the
18 most important.

19 PANEL MEMBER BLANC: Historically.

20 SUPERVISING TOXICOLOGIST MARTY: And they continue
21 to be even more important because they've cut back on
22 exposures from pulp mills by changing the bleaching process.

23 PANEL MEMBER FUCALORO: So we are to consider, for
24 example, when we look at exposure levels, we're to consider
25 ingestion if, in fact, it originally was ample?

1 SUPERVISING TOXICOLOGIST MARTY: Absolutely.

2 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

3 SALMON: Can I just intrude a point here?

4 One of the points that I was making in relation to
5 the sources of current human exposure is that the primary
6 approximate source of the body burden of dioxin, which most
7 people carry around with them, is the food chain.

8 Now the current airborne exposures are an
9 important contributor to the food contamination. The
10 material turning up in the meat and the dairy products is
11 not coming primarily from the old waste sites, the old river
12 sediments, the old chemical factory sites. It's coming from
13 airborne emissions, because most of the cows grow in areas
14 which are not close to the traditional core contamination
15 sites, which is where a lot of the historical contamination
16 is located.

17 They're receiving constant input of dioxin into
18 the immediate -- you know the grass, it's landing on the
19 grass, it's --

20 PANEL MEMBER BLANC: And there is data in this
21 document that support that?

22 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF

23 SALMON: We have not attempted to duplicate the very
24 considerable amount of work which was done by the US EPA in
25 their recently approved and released dioxin 2000 report, but

1 we do rely on that to a considerable extent. We have not
2 ourselves had access to such comprehensive studies as what
3 they have reported.

4 However, this is an area of active of work and, in
5 fact, the Air Board and the various regional -- you know the
6 state board and the regional boards and the water quality
7 boards are working on this.

8 PANEL MEMBER BLANC: So your argument would be
9 even if there was one pound released per year in California
10 that would be enough to make you want to list it on the top
11 five?

12 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
13 SALMON: I would be very concerned about minimizing. Given
14 that we are currently into the effect range in terms of what
15 we're exposed to and what we carry around in our bodies, I
16 would be very concerned to reduce the emissions as far as
17 possible, yes.

18 PANEL MEMBER BLANC: So as long as there were any
19 emissions in California you would put it -- I mean I'm just
20 trying to get a sense again, since we're being asked to do a
21 relative weighting, at least we're being asked to evaluate
22 your relative weighting, I have to be able to understand the
23 logic behind your relative weighting.

24 SUPERVISING TOXICOLOGIST MARTY: Maybe I can shed
25 a little bit of light. The emissions that are in this table

1 are stationary source emissions, so that doesn't include all
2 the mobile source emissions of dioxins.

3 CHAIRPERSON FROINES: Can I interrupt long enough
4 to raise an issue?

5 I actually think that there are two questions, one
6 of which is absolutely predictable that this discussion on
7 exposure was going to occur, because the exposure question
8 is highly questionable, I think, and so what Paul is doing,
9 I think, had to occur, has to occur.

10 But the corollary to that is Peter's question,
11 which is that, you know, I think the evidence on
12 carcinogenicity at this point is relatively strong,
13 particularly with respect to soft tissue sarcomas at least.
14 But clearly there seem to be some significant species
15 differences in the toxicity of the dioxins relative to
16 humans so that the exposure question and the differential
17 toxicity between animals and humans, those two are actually
18 coupled.

19 So when Paul is asking you the question about one
20 pound and you answer yes, that has something to do with both
21 persistence in the environment, but also a perception about
22 the level of toxicity of the compound --

23 PANEL MEMBER BLANC: Differentially.

24 CHAIRPERSON FROINES: -- and differential
25 toxicity. So the issues are linked.

1 I had one question that hopefully might shed some
2 light, but I have a feeling I know the answer. And that is
3 in your diesel document, of course, where you do this thing
4 which I must admit I'm not a great lover of, but I'll live
5 with it, where you list in table one the various toxic air
6 contaminants found in diesel exhaust, you list chlorinated
7 dioxins.

8 And so the obvious reader of this document has
9 some reason to believe that chlorinated dioxins are released
10 by diesel exhaust and the question then becomes what do we
11 know about that because that's obviously an important issue,
12 and the corollary question would obviously be is there
13 anything in gasoline? But at least with respect to diesel
14 the question is does this constitute a significant release?
15 Does it constitute a negligible release? Is it really no
16 releases?

17 I don't know the answer to it. And this could
18 represent an important source of dioxin to the degree that
19 there's any being released at all.

20 (Thereupon a short discussion was held
21 off the record.)

22 CHAIRPERSON FROINES: The problem is if you list
23 it in your document it's likely to get asked.

24 PANEL MEMBER FUCALORO: Just again. It's very
25 toxic. I mean you spend some time describing the toxicity,

1 and I'm not even looking at the differential toxicity at the
2 moment, but just toxic in general.

3 We're finding it in human beings. We're finding
4 concentration in human beings. And now the question as to
5 what's the original source of it, whether it's airborne at
6 one point or not, that's still undecided, correct? Remember
7 where there are ratios that we were asking before, 50 to
8 one, or something like that, there's no estimate as to what
9 those ratios might be or any guess at what they might be?

10 SUPERVISING TOXICOLOGIST MARTY: No. We don't
11 have ambient concentration data --

12 PANEL MEMBER FUCALORO: Is it your guess that most
13 of these materials showing up in human beings are originally
14 airborne?

15 SUPERVISING TOXICOLOGIST MARTY: Yes.

16 CHAIRPERSON FROINES: But, Melanie, not wanting --
17 I don't know the literature here so I'm asking a question
18 out of ignorance. There's a fair amount of herbicide use
19 that's occurred in California over the years too, so that
20 it's not as though there hasn't been a nonairborne source of
21 dioxins into the environment.

22 I don't know how much herbicide use has been used
23 with dioxin as a contaminant, but that's clearly another
24 source. I don't know, for example, around railroads how
25 much, you know of these compounds have been used to clear

1 railroad tracks and all the other things that we know that
2 happens with herbicide use.

3 So I think one has to be a little careful to
4 ascribe it all to airborne releases, given the herbicide
5 use. And I have no idea how much is used over the years.

6 PANEL MEMBER FUCALORO: You see in some ways the
7 concern here, I mean, is that five slots makes each slot a
8 very dear commodity.

9 (Laughter.)

10 PANEL MEMBER FUCALORO: And so if you take up a
11 slot with this, you don't have something else that may also
12 be very adverse to human health.

13 SUPERVISING TOXICOLOGIST MARTY: I understand
14 that.

15 CHAIRPERSON FROINES: Let me just say one thing.
16 We spent yesterday with an all day meeting of our particle
17 center with folks from ARB in a very very successful
18 meeting. And one of the things that we learned is that ARB
19 is about to start doing fairly extensive monitoring for
20 dioxins. And so the state is actually in a position where
21 it's going to begin to do monitoring to give us better data
22 on this particular issue.

23 But it does seem to me, going back to Paul's
24 original thrust of his questions that we are laboring here
25 in terms of the current state of the problem.

1 SUPERVISING TOXICOLOGIST MARTY: I agree that lots
2 more information needs to be developed in terms of what the
3 sources of dioxins are into the air. But I think it's
4 pretty clear from US EPA's work that currently the majority,
5 over 90 percent of the sources of emissions in to the
6 environment, are coming from the air.

7 The cows that you eat, that you are eating, which
8 are contaminated with dioxin, they're contaminated because
9 they're eating grass which keeps growing and the input is
10 airborne deposition.

11 So to me the dioxins that you're getting now in
12 your food are largely from airborne sources with the
13 possible exception of fish near --

14 CHAIRPERSON FROINES: Yeah, but I get radioactive
15 iodine from China's atom bomb testing from 20 years ago. So
16 what you just said doesn't necessarily carry a lot of water.
17 I mean we get dioxins, airborne, from places all over the
18 world that the winds blow that material over California and
19 so there's deposition in California.

20 So we have deposition from Japanese dioxin.

21 SUPERVISING TOXICOLOGIST MARTY: We also have
22 deposition from California generated dioxin. I mean there's
23 no question about that.

24 CHAIRPERSON FROINES: So, yes, there is airborne
25 exposure to dioxins. Paul's question is, in fact, trying to

1 get at how much and is it significant. And am I still
2 waiting for somebody to suggest what the -- tell me that
3 either you don't know about diesel or you do and somebody is
4 going to give us a number or what?

5 SUPERVISING TOXICOLOGIST MARTY: I would have to
6 have Air Board people who are the ones that listed dioxins
7 as being diesel exhaust come and talk about it. I can say
8 one thing that the modeling that I mentioned earlier from
9 the Regional Water Board, that attributed a lot of the
10 dioxins in the Bay to vehicular traffic. They were really
11 talking about truck traffic. But, you know, I don't have
12 that in front of me.

13 I think it's --

14 PANEL MEMBER GLANTZ: Do you know anything about
15 diesel versus gasoline?

16 SUPERVISING TOXICOLOGIST MARTY: I don't, but I'm
17 sure the Air Board does.

18 PANEL MEMBER GLANTZ: Does anybody from the Air
19 Board, can they comment on that?

20 CHAIRPERSON FROINES: There's nobody from the Air
21 Board here who's familiar with this area.

22 PANEL MEMBER BLANC: Well, let me ask, maybe part
23 of the problem is the ellipses in this section of this
24 document. But, you know, it goes right from the toxics
25 inventory to the statement extracted from the EPA document

1 that 90 percent of dioxin currently coming into the
2 environment comes from airborne sources, which is a
3 statement. But didn't the EPA base that on air monitoring
4 in other states?

5 I mean I would be more sympathetic if there was a
6 paragraph of data that said airborne dioxin monitoring from
7 35 states in every state in which it's been done has found
8 ambient levels and these ambient levels range from X to Y,
9 and that would translate into, you know, this kind of --
10 that would be equivalent to X amount of pounds released in
11 total in California to get to that kind of ambient level.

12 Because all you have here is the, in italics, the
13 two-sentence statement from the -- I'm not asking you to
14 recapitulate the EPA document, but you're asking us -- this
15 comes back to a recurring issue. You're asking us to make a
16 scientific comment on a document, so I can't make a
17 scientific comment on, you know, here's what the EPA
18 summarized, you know.

19 I need to have something more than that,
20 especially if it's linked to -- the only hard data it's
21 linked to shows 50 pounds of release.

22 SUPERVISING TOXICOLOGIST MARTY: Well, let me come
23 back to another argument and that is that the statute
24 requires us to look at not just differential toxicity, but
25 differential exposure. And it's unarguable that a breast

1 fed infant has much more exposure than a formula fed infant
2 and than an adult.

3 PANEL MEMBER BLANC: Well, they have to have
4 exposure to something which has -- or are you saying because
5 it's a toxin in general? Okay, but still you have to -- the
6 missing piece is the airborne component piece.

7 SUPERVISING TOXICOLOGIST MARTY: And what we're
8 arguing there is that the dioxin that's in my body now was
9 largely initially airborne, that's the argument.

10 PANEL MEMBER BLANC: But where is the data to
11 support that argument other than the statement from the EPA
12 in terms of the current situation?

13 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
14 SALMON: There is an extensive report which we cite, which
15 is the US EPA report.

16 PANEL MEMBER BLANC: But do they have some
17 airborne ambient data?

18 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
19 SALMON: They came to that conclusion on the basis of a
20 combination of measurements and their modeling.

21 CHAIRPERSON FROINES: I think there are two things
22 to say.

23 One, as you know, I communicated with Bill Glaze,
24 who was the chair of that committee. And Bill sent me an e-
25 mail back saying that those issues were very controversial.

1 And I just want to say one thing -- and so I think
2 that the problem for us, as a panel, is that -- and this has
3 come up before and I don't want to make an issue of it, but
4 the citing of somebody else's secondary review doesn't help
5 this Panel with the information.

6 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
7 SALMON: I think our hope was that we were trying to cite
8 the EPA's primary work of undertaking the measurements and
9 analysis. We may not perhaps have reflected that as
10 completely as we should have done in our citation, but that
11 was the intended reference.

12 CHAIRPERSON FROINES: But the point is that what
13 Paul is suggesting is that there's inadequate description of
14 what's in the EPA report for him to --

15 PANEL MEMBER BLANC: Some of it's on the page
16 preceding, I see. So it may be partly a cut and paste issue
17 to an extent.

18 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
19 SALMON: I'm sure that given a little more time we could do
20 a better job of describing what it is we're referring to.

21 CHAIRPERSON FROINES: I just want to make --
22 Melanie obviously feels quite strongly about the breast milk
23 issue and I can understand that, and support her view on it.
24 But I think that the issue of exposure is a question of is
25 there current exposure or anticipated exposure that will

1 constitute a public health problem in the future?

2 That's different than saying there has been
3 exposure and now it's in the breast milk. Those are
4 different ways of looking at the problem. And I think that
5 we're really talking about exposure in terms of whether or
6 not it constitutes a current and potentially ongoing public
7 health problem from the standpoint of exposure.

8 So I think it's not sufficient to say it has
9 occurred in the past and we have to live with it and it does
10 create an ongoing problem in that context, which I would
11 agree with. But the ongoing problem of it being in the air
12 is really one of the issues that we need to worry about.

13 AIR TOXICOLOGY AND RISK ASSESSMENT UNIT CHIEF
14 SALMON: I think there's clearly more work being done and
15 hopefully we will have better detail in the future. But I
16 think the fact that you indicated there's a fair amount of
17 controversy about some of these issues on dioxin. But
18 nonetheless, I think most of the people who are contending
19 nonetheless believe that there is a problem with continuing
20 emissions of dioxins into the air and then getting
21 specifically into the food chain.

22 This is one of the reasons why both the Air Board
23 and the federal government have been paying so much
24 attention to the issue of backyard trash burning, because
25 this is, I believe, considered to be a substantial source

1 and one which they feel, you know, needs more attention and
2 better control.

3 CHAIRPERSON FROINES: Let me ask you a favor here.
4 George and Melanie, let me just ask you a favor. For the
5 moment, the three of you take our place and sit at this
6 table and in other words put yourself in our place.

7 PANEL MEMBER GLANTZ: Then they could give us a
8 hard time.

9 (Laughter.)

10 CHAIRPERSON FROINES: No, wait a second, I'm not
11 joking and we're not giving them a hard time. We're trying
12 to talk carefully through an important issue.

13 The question is this is a compound or compounds
14 that have been suggested to be in Tier 1, to be the five
15 listed, and we have to make a decision. The decision for us
16 is not whether or not it should be listed in Tier 1. That's
17 really Joan Denton's decision.

18 The decision that we have to make is is the
19 presentation of the scientific information sufficient for us
20 to approve the document that we have in front of us. That's
21 the decision we have to make.

22 And so that the level of evidence that's before us
23 is really what's the question that's being raised. And so
24 by putting yourself in our place, help us figure out how can
25 we get to the place where we say, yes, this is sufficient

1 information or suggest a process by which we can get there
2 on this one, because obviously it's troubling.

3 So that it seems to me to be the issue that we
4 have to worry about at this point.

5 PANEL MEMBER BLANC: I think another --

6 PANEL MEMBER GLANTZ: Let them answer the
7 question.

8 PANEL MEMBER BLANC: Is there an answer to that?
9 That's not a -- it wasn't a question.

10 CHAIRPERSON FROINES: I think we should break for
11 lunch and I think we should go away and come back and
12 Melanie and George and Andy can suggest what they think is
13 the best way to think about this in terms of bringing it to
14 closure.

15 PANEL MEMBER FUCALORO: I lost my appetite knowing
16 there's dioxins --

17 PANEL MEMBER GLANTZ: I'm a little concerned -- I
18 mean I'm always happy to eat, except not fish, I guess.

19 (Laughter.)

20 PANEL MEMBER GLANTZ: Although I used to like
21 fish. But, you know, I think it's important that we come to
22 closure on the findings today and I'm a little worried that
23 we're getting into such a level of detail on everything
24 we're talking about that we're going to be here until
25 midnight, and I mean, I'd just like to raise that. And I

1 think that, you know, everyone appreciates that you can
2 always ask more questions and raise more issues and I mean
3 it will be interesting to see what people think around the
4 table, which two of these should be thrown out or which
5 should be added.

6 But I think it's also important to remember that
7 this isn't the end of the process, that, you know, we will
8 come up with the five that the Legislature says we have to
9 come up with by July 1st.

10 But the law also says you can add to the list as,
11 you know, subsequently. And so I think that some of these
12 things, if people are sufficiently troubled about one
13 chemical or another being in the top five I think it's going
14 to be hard to take two out of this list. Some of these
15 requests for additional information, I think, are things
16 that we can deal with later.

17 CHAIRPERSON FROINES: But wait a second. I was
18 being very careful in what I said. We don't choose which
19 two chemicals to take out. That's not our job. That's not
20 legislatively mandated for us to do. It is our job to
21 approve or disapprove the documents that we have before us,
22 that's what we do.

23 Our job is not to make Joan Denton's decision.
24 Joan Denton makes that decision. So I think it's important
25 at this stage for everybody in this panel to recognize that

1 what we're doing is saying that these documents we approve
2 or disapprove or say go back and start over again, or
3 whatever, but that's what we're doing.

4 We are not the decision-makers in this law on
5 whether dioxin is Tier 1 or Tier 2.

6 PANEL MEMBER FUCALORO: In that spirit, John, I
7 just draw your attention to the paragraph on page six,
8 where, frankly maybe it's -- I was very good in reading
9 comprehension, but I'm having a little trouble understanding
10 exactly what's being said here.

11 It says, "Fortunately between 1987 and 1995
12 emission of dioxins into the environment has decreased by
13 almost 80 percent from the level reported in the seventies."
14 That's clear. "This decrease is primarily due to decreased
15 emission of dioxin and related compounds into the
16 atmosphere." What is unclear is why was there a reduction
17 in those emissions? Okay, it's not clear.

18 Then it says, "Because of new regulations
19 promulgated by US EPA in '95 from municipal waste combustors
20 and in '97 from medical waste incinerators, levels of
21 dioxins emitted into the atmosphere from the two sources is
22 expected to result in a greater than 95 percent reduction in
23 dioxin emissions into the air in the United States."

24 Now 95 percent based upon the seventies or the
25 later number? I mean this is just a matter of clarity. I

1 mean it's not argument here that I'm trying to make. That
2 is unclear to me and I think that really has to be made
3 clear, exactly why, to repeat, why was this -- what resulted
4 in the reduced emission of dioxins in the period from '87 to
5 '95? And the expectation of a 95 percent reduction, is that
6 an additional 95 percent reduction or is it based on the
7 1970?

8 That information must be available.

9 CHAIRPERSON FROINES: We actually have been over
10 that in a previous meeting and Tony didn't remember it, but
11 we did go over it. At some meeting we have done it.

12 But I don't want to get into that and I don't want
13 you to answer that question.

14 If you remember years ago when we did lead, Stan
15 took approximately 36 hours, as far as I could tell, to go
16 through all of his comments on lead. It felt like 36 hours,
17 it was probably two. But whatever it was it went on. And
18 what happened was you then went back and made changes to
19 your document, but in the process we had provisionally
20 accepted your document, even though Stan had just incredible
21 numbers of changes.

22 My question for you isn't about the substance of
23 the issue, it's about can you go back with this document and
24 develop a discussion of the exposure that would meet the
25 criteria in a sense that Paul's asking about and can you say

1 that yes, that there is an information, for example, in the
2 EPA document that could be put in. And then the Panel will
3 feel more satisfied with saying, okay, we will approve the
4 issue with respect to -- we'll approve the document with
5 respect to dioxin, given the potential changes. And the
6 Panel has to agree with what I'm suggesting.

7 I'm suggesting an approach that can move us off
8 dead center, that's all.

9 I'm using the Stan Glantz model here.

10 (Thereupon a discussion was held off the
11 record.)

12 CHAIRPERSON FROINES: Melanie, is that --

13 SUPERVISING TOXICOLOGIST MARTY: We can do that.
14 We can try to develop the argument.

15 PANEL MEMBER BLANC: And I guess I want to echo,
16 Stan, something that John alluded to as well in your
17 comment. And that is that I don't want you to have the
18 impression or to give others the impression that we are
19 somehow, quote, "Giving them a hard time," unquote.

20 PANEL MEMBER GLANTZ: Oh, I was joking.

21 PANEL MEMBER BLANC: But it's not a good subject
22 to joke on because there was a lot of, you know, morale
23 problems in follow-up to the last meeting, apparently,
24 which, you know, are understandable, but are related to the
25 necessary role that we have to take which can't always be

1 soft-edged in certain situations. And sometimes the
2 necessity for taking, you know, a somewhat demanding role is
3 necessary in order to fulfill our obligations, at least as I
4 understand them to be.

5 The reason why we have to be very cautious not to
6 have that veer over into a misinterpretation of giving
7 people a hard time or being difficult or being ornery or
8 being overly aggressive or inappropriately aggressive is
9 because that if we start to stray into that kind of
10 territory we may inadvertently in substance or appearance
11 have the Panel not fulfill its responsibilities fully.

12 PANEL MEMBER GLANTZ: Well, actually at the risk
13 of delaying lunch, I'd like to actually offer a comment on
14 that, it was a point I wanted to make later, and it was a
15 joke. Just for the record, I was just making a joke here.

16 But I think if you look at the record and some of
17 the reporting of these meetings and the fact that the OEHHA
18 staff have been questioned very vigorously, we'll say, over
19 the course of the development of this document, I think it's
20 important to actually clarify a couple of things about the
21 way this process on this particular document has run.

22 First of all, I think that the Panel in being very
23 very vigorous in their questioning of OEHHA as this document
24 has evolved has been totally appropriate. The thing which
25 is different about this one, from most everything else I can

1 remember from having been on this Panel is that the
2 Legislature adopted a very short deadline for the production
3 and approval of this document. And I think, based on my
4 prior experience on this Panel over a period of many years,
5 this thing got produced in lightning speed and so I think
6 what ended up coming in front of this group wasn't as
7 polished as the typical OEHHA document has been, simply
8 because they didn't have time to go through as many
9 iterations as they normally would before it would come here.

10 Now I think that was appropriate because I think
11 we should meet the deadline established in the law if we at
12 all can. But I also -- and I think as a result we were
13 seeing and we were commenting on things that were rougher
14 than usually get to the public meetings. And I think that's
15 something that everybody just needs to recognize.

16 And I think the Panel, because the Panel and the
17 public and the media who have reported on these meetings
18 need to recognize that. I mean I happen to think,
19 notwithstanding the discussion so far today, which I pretty
20 much concur with everything that was said, but the document
21 that we're looking at now, the June draft is far, far, far
22 superior to the previous iterations.

23 Now does that mean there's -- I mean I think
24 there's a bit more work that needs to be done, but I think
25 in the end we're going to have a document which is up to the

1 same high quality standards of everything else we've dealt
2 with in here. And I think it's just important to put that
3 on the record.

4 I was going to give this little speech later, but
5 I think it's very important to put that on the record. And
6 I think the people at OEHHA have been working very very hard
7 on this to try and meet the criticisms and the issues that
8 the Panel has made and that they've imposed on themselves.

9 And so I just think by the time we finally approve
10 this thing, I think it's going to be up to the usual
11 standards that we work to. And I don't want people to feel
12 that because of the vigorous questioning and vigorous
13 discussion that's occurred, based on earlier drafts of this
14 document, that somehow the final product, as it emerges, is
15 somehow deficient as a result, because I don't think it will
16 be by the time we're done with it and that they're done with
17 it.

18 CHAIRPERSON FROINES: I think that the comments by
19 Stan and Paul are extremely useful and basically reflect the
20 fact that this Panel has the highest regard and respect for
21 OEHHA historically and currently and will have in the future
22 and so that everybody needs to understand that that's the
23 foundation upon which we operate from, that we have nothing
24 but the highest regard for OEHHA and operate from that
25 vantage point.

1 And also we're going to be back from lunch at 1:30
2 and we'll proceed.

3 PANEL MEMBER BLANC: I think it would be
4 reasonable to say 1:45, so it's a reasonable timeframe, so
5 we're not setting it up to be late in advance. Because I
6 can just tell you even to get the food out of this
7 cafeteria --

8 PANEL MEMBER FUCALORO: But if he says 1:45 he
9 means exactly 1:45 and not 1:52, okay.

10 CHAIRPERSON FROINES: 1:40.

11 (Thereupon the lunch recess was taken.)

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1 AFTERNOON SESSION

2 SUPERVISING TOXICOLOGIST MARTY: What we're going
3 to do next is go over -- okay, George is telling me that Jim
4 Aguila from the ARB has a comment or two on what they're
5 doing monitor dioxin in California now if you want to hear
6 that.

7 CHAIRPERSON FROINES: Briefly.

8 PANEL MEMBER FUCALORO: Seventeen words or less,
9 Jim.

10 MR. AGUILA: Okay, I'll do my best.

11 CHAIRPERSON FROINES: We're still interested in
12 knowing about the dioxin levels from diesel.

13 MR. AGUILA: What I can do is share with you what
14 information the Air Resources Board has on dioxins. And
15 basically, as Melanie indicated, the information that was
16 shared in the report was based on the hotspots data, which
17 again is stationary source information.

18 We don't have any ambient data at this point, but
19 we have done some testing in connection with the EPA's
20 development of max standards for the refinery catalytic
21 cranking units. And I believe in 1999 the Air Resources
22 Board did some source testing in Chevron and Tosco
23 refineries which indicated that there were low dioxin levels
24 in the beaker gram per thousand barrels --

25 CHAIRPERSON FROINES: In the oil?

1 MR. AGUILA: Excuse me?

2 CHAIRPERSON FROINES: In the oil?

3 MR. AGUILA: Actually the catalytic cranking unit
4 is a -- it's a unit that produces reformate, which is a high
5 octane blending component for gasoline. And based on that
6 information the Air Resources Board has developed a dioxin
7 testing program, which basically has two parts.

8 There will be an ambient monitoring component
9 which is scheduled to start in the fall of this year. There
10 will be four sites selected in the Bay Area and four sites
11 in the South Coast as well.

12 In addition to that there's also going to be
13 another component to that program which is going to look at
14 sources, dioxin sources and source characterization testing.
15 Right now the Air Resources Board is in the process of
16 selecting those sites and they're looking at sites like
17 refineries, medical waste incinerators, hazardous waste
18 cleanup sites. And again that testing will start later this
19 year as well.

20 There will also be a mobile source component to
21 that, but because there isn't any source test method it's
22 going to take a little bit longer and I believe the
23 timeframe on that will be about 2003.

24 So basically that's the information we have. We
25 also have some information from a contract study, where they

1 looked at a single engine diesel engine. And that again
2 showed trace levels of dioxins as well.

3 CHAIRPERSON FROINES: The question is what's the
4 chlorine source for diesel?

5 MR. AGUILA: I guess it's speculated that it could
6 be from the engine oil or it's even a component in the crude
7 oil itself. And there is some crude oil that's even
8 transported by tanker truck and you get the sea water in
9 there, so that's a potential source.

10 CHAIRPERSON FROINES: Thank you very much. We
11 appreciate it very much.

12 SUPERVISING TOXICOLOGIST MARTY: Okay, what we
13 were planning on doing was giving a very brief overview of
14 five of the chemicals that we've already presented to the
15 panel just as a refresher before your discussion, in
16 preparation for your discussion.

17 And we also were asked a couple of meetings ago to
18 do a comparison of the information on acrolein and
19 formaldehyde in terms of the guinea pig hyper responsiveness
20 model, so we have that information if you want to hear that
21 too.

22 CHAIRPERSON FROINES: I think we're particularly
23 interested in the formaldehyde point. I think we're
24 generally familiar with the acrolein, so I wouldn't spend
25 too much time on it. But I think the formaldehyde is the

1 one for which the evidence is the shakiest, from what we
2 think at this point, but --

3 SUPERVISING TOXICOLOGIST MARTY: Okay, well we can
4 just skip acrolein and just talk about formaldehyde.

5 CHAIRPERSON FROINES: It's whatever you think.

6 PANEL MEMBER GLANTZ: Well, can I just ask a
7 question of the Panel, because I just am worried that we're
8 going to run out of time for the most important part of the
9 discussion.

10 Of the things in the findings, the draft findings
11 that we've prepared, of the things that are currently
12 listed, you know, in item number eight, which is what we've
13 been calling Tier 2, is there anything there that anybody
14 thinks ought to be moved up? Because if not, then we don't
15 need to further discuss them and we can just go on.

16 Formaldehyde is in the Tier 1 list right now. But
17 I mean, the things that are listed as ones that we say are
18 concerned but not in the top five, it's arsenic, benzene,
19 carbon disulfide, glycol ethers, manganese, mercury,
20 methylene chloride, methyl bromide, PCBs and vinyl chloride.

21 So is there any of those that anybody seriously
22 thinks ought to be moved up?

23 CHAIRPERSON FROINES: Peter.

24 PANEL MEMBER BLANC: But the caveat is that
25 assuming that they stay in the lower Tier I think that it is

1 -- I agree the assumption is, however, that there is
2 modifying language that makes it clearer that some of these
3 are of more concern than perhaps others.

4 PANEL MEMBER GLANTZ: Right. Well, I think that a
5 separate question is do we want to drop any of them from
6 Tier 2. But the question -- I don't think so either, but
7 the question here is so there's nothing that's currently
8 listed as Tier 2 that anybody wants to move up. So I don't
9 think we really need to talk about that at all.

10 PANEL MEMBER BLANC: That wasn't what she was
11 about to talk about. She was about to --

12 PANEL MEMBER GLANTZ: Well, yes, she was actually
13 and then there was the question of formaldehyde versus
14 acrolein.

15 SUPERVISING TOXICOLOGIST MARTY: Stan, right now
16 in the table in the document we still have the old Tier 1,
17 Tier 2.

18 PANEL MEMBER GLANTZ: I know, I'm talking about
19 the findings.

20 SUPERVISING TOXICOLOGIST MARTY: Okay, I'm sorry.

21 PANEL MEMBER GLANTZ: I'm talking about the
22 findings list.

23 Okay, so having said that, I think formaldehyde --
24 I agree with John that we should talk about formaldehyde
25 because that's -- I think now we should be talking about

1 basically -- we've got six of them listed in Tier -- there
2 are six of them listed in the findings and we can only list
3 five, so the real question is what goes.

4 CHAIRPERSON FROINES: I think for purposes of
5 time, Melanie, you probably should focus on three chemicals
6 or three substances. One being formaldehyde, clearly, the
7 second being changes you've done with respect to PAHs in
8 diesel, seem to be the ones. The acrolein one, I think
9 unless there is any new information or comparative
10 information, you can include it or not include it as you
11 wish.

12 SUPERVISING TOXICOLOGIST MARTY: Okay.

13 Well, let's go ahead with formaldehyde and Stan
14 Dawson is going to make a very brief presentation on a
15 synopsis of what we're thinking on formaldehyde.

16 DR. DAWSON: This is mostly a reminder of what we
17 went through last time and a little bit of a new synthesis.

18 Formaldehyde was placed in Tier 1 because human
19 data suggests that children are more sensitive than adults
20 to formaldehyde. Studies in children indicate adverse
21 respiratory effects from formaldehyde, concentrations of 13
22 to 26 ppb compared to a NOAEL of 26 ppb and a LOAEL of 75
23 ppb for adults. And that's, of course, a continuous
24 equivalent derived from occupational studies in the chronic
25 REL.

1 And the next point is that data in guinea pigs
2 show increased bronchoreactivity at very low concentrations
3 after eight-hour exposures and extensive exposure data for
4 formaldehyde exist, mean in-door values exceed the chronic
5 REL of 2 ppb by four to 20 times.

6 Next I have a slide of the concentrations, data
7 furnished by compilation in the Air Resources Board. You
8 can see a number of different locations there and throughout
9 California and averages. The conventional homes and
10 manufactured home samples are throughout California, 11 and
11 45, and then some US data on public.

12 The schools was a kind of a special sample the ARB
13 got from the data they could glean at very selected schools,
14 but both northern and southern California, with 26 ppb.
15 These, of course, are all mean values as you can see. And
16 then some values in vehicles.

17 And finally the outdoor value was 3.6.

18 PANEL MEMBER WITSCHI: So why would the vehicle
19 values be higher in L. A. than they are in Sacramento?

20 SUPERVISING TOXICOLOGIST MARTY: That's just the
21 measurements that were made by the Air Board in their in-
22 vehicle study.

23 PANEL MEMBER FUCALORO: Sacramento has cleaner
24 cars.

25 (Laughter.)

1 SUPERVISING TOXICOLOGIST MARTY: You know, there
2 could be a lot of reasons, including congestion on the
3 freeway, you know, that sort of thing, but that's what they
4 got.

5 PANEL MEMBER WITSCHI: Well, no, but they're much
6 higher.

7 CHAIRPERSON FROINES: Could you go back to that
8 just for a second?

9 PANEL MEMBER WITSCHI: Yeah, I mean first of all
10 there must be something in the car that emanates
11 formaldehyde because otherwise it can't be much higher than
12 it is in outdoor air. And so why are the cars in Sacramento
13 different from the ones in Los Angeles?

14 SUPERVISING TOXICOLOGIST MARTY: I think that if
15 you look at those studies the inside concentrations were not
16 very different than the outside concentrations, and so we're
17 talking about freeways.

18 PANEL MEMBER FUCALORO: Somebody could have been
19 smoking.

20 SUPERVISING TOXICOLOGIST MARTY: I don't think --
21 no, these wouldn't be cigarette smoke because the people
22 driving the cars were ARB employees.

23 PANEL MEMBER FUCALORO: And you also don't know
24 when they were done, for example, the time of year and also
25 the years apart that they were done.

1 DR. DAWSON: Actually there's more documentation
2 in the document that you have that gives the footnotes.

3 Now the four studies that we are relying on for
4 discussing differential effects, the main four human
5 studies, are described here. The first study, the Chris
6 Krzyzanowski study, is a large study and it's in homes and
7 it has both -- the main thing to point out is it has both
8 children and adults in the study. So there are around 600
9 adults and 300 children.

10 And then two of the other three studies are in
11 homes and they are a good deal smaller. And, of course, one
12 of them is at schools.

13 Now, this is a summary of all these results in
14 these four studies of relevance here. First of all the
15 study is listed on the left and then the objective
16 respiratory effects are listed for each study.

17 You can see that the bronchial -- well, the two
18 pluses means that that's highly significant, that this is
19 the chronic bronchitis and then asthma and the peak
20 expiratory flow rate are also significant. Note over on the
21 far right-hand column that reported symptoms are
22 nonsignificant. This, of course, is -- these effects are
23 correlated with formaldehyde exposure.

24 And incidentally they're corrected for various
25 possible confounders.

1 And then the Garrett study you can see was not
2 significant for asthma but it was for the atopy, as
3 indicated by a skin test. And the Wantke data in schools in
4 Vienna was highly significant for specific formaldehyde
5 response in the RAST test, and that was with a change in
6 schools.

7 Now also, Wantke should point out, they found a
8 fair amount of correlation among affected children with
9 symptoms, but what was statistically significant was the
10 finding that, with a change in schools, that the symptoms
11 were reduced. And the previous slide showed that the
12 formaldehyde levels had dropped by something over a factor
13 of two.

14 And finally the Franklin study of very significant
15 changes in the exhaled NO, an indication of lower airway
16 inflammation.

17 SUPERVISING TOXICOLOGIST MARTY: I think we need
18 to point out that the only study that looked at adults and
19 kids was the Krzyzanowski study and that for adults there
20 were no significant outcomes that were associated with the
21 formaldehyde bubble.

22 DR. DAWSON: Right and that's the footnote down at
23 the bottom.

24 PANEL MEMBER WITSCHI: I have another question.
25 There was also an experiment with formaldehyde and these

1 were with homes, I think, in Canada which had been insulated
2 with urea-formaldehyde. And there was a fair amount of
3 epidemiologic studies on that one. Do those studies
4 indicate anything about children?

5 DR. DAWSON: Nothing turned up in our literature
6 search and I looked pretty hard.

7 PANEL MEMBER WITSCHI: Well, did you look at those
8 studies, I think it was in the Toronto area?

9 DR. DAWSON: No, I didn't specifically. Our
10 search was four studies that would be relevant for children.

11 PANEL MEMBER WITSCHI: Well, these were some
12 pretty substantial exposures and there were some, when the
13 whole thing came up, you know, there were some very
14 concerned -- so extensive studies were being done. My point
15 is if those studies do not mention anything explicit in
16 children, whatever is its worth?

17 PANEL MEMBER BLANC: I was under the impression
18 that you were also going to come back with some of the data
19 on controlled human exposures in terms of persons with and
20 without airway reactivity?

21 SUPERVISING TOXICOLOGIST MARTY: We actually had
22 that data in our original document. This is with respect to
23 whether or not formaldehyde exacerbates asthma, and that
24 data in adults indicates that you probably have to have
25 presensitisation to fairly high concentrations such as you

1 would experience occupationally in order to get people to
2 respond to formaldehyde in terms of exacerbating asthma.

3 PANEL MEMBER BLANC: So there's no experimental
4 evidence that asthmatics are more sensitive to the irritant
5 effects of formaldehyde than non asthmatics in controlled
6 human studies?

7 SUPERVISING TOXICOLOGIST MARTY: That's right. We
8 have a little bit of animal model information, but --

9 PANEL MEMBER BLANC: That just shows that if you
10 expose animals to enough formaldehyde you can induce airway
11 responsiveness which is a different question and should be
12 universally true of almost any irritant.

13 So, to summarize the only study that you have that
14 suggests any preferential effect among children is a single
15 analysis based on home monitors of formaldehyde.

16 SUPERVISING TOXICOLOGIST MARTY: Right.

17 PANEL MEMBER BLANC: And other than that you have
18 no other human studies that suggest a preferential effect
19 based on age and you have no animal data that suggest a
20 preferential effect of age?

21 SUPERVISING TOXICOLOGIST MARTY: We don't have any
22 studies that looked at both kids and adults in the same
23 study, but when you look overall at the studies we have
24 found studies that measured respiratory impacts in kids at
25 fairly concentrations and they were lower than the

1 concentrations we found when we looked for our chronic REL
2 in adult studies.

3 Now they're not measuring the same things, so it's
4 real hard --

5 PANEL MEMBER BLANC: One is measuring the RAST
6 level and in that study one of the things that was
7 impressive was the number of children who had positive
8 specific RAST to formaldehyde, if I understood your synopsis
9 of the study?

10 SUPERVISING TOXICOLOGIST MARTY: Yes.

11 PANEL MEMBER BLANC: Is that correct?

12 SUPERVISING TOXICOLOGIST MARTY: That's what they
13 said.

14 PANEL MEMBER BLANC: And wasn't it something like
15 20 percent of the kids had a positive RAST to formaldehyde?

16 SUPERVISING TOXICOLOGIST MARTY: Yes.

17 PANEL MEMBER BLANC: I mean there's a biological
18 plausibility issue there. That's such a prevalence of --
19 for specific sensitivity of formaldehyde it's not been
20 elsewhere reported to my knowledge. Not only that, the
21 whole issue of measuring specific sensitivity to
22 formaldehyde has proved to be quite difficult and I'm not
23 sure that there actually is a reliable RAST method for
24 formaldehyde specific IGE.

25 I mean you have to do albumin conjugates and it's

1 very difficult to interpret. It's kind of like the TDI
2 literature only worse, which was why it's been so difficult
3 to even establish convincing occupational specific
4 sensitization to asthma. Even the Burge cases are somewhat
5 problem ridden.

6 SUPERVISING TOXICOLOGIST MARTY: We agree that
7 that study is kind of standing out there by itself.

8 PANEL MEMBER BLANC: So, in summary then, you
9 don't have any studies that do a head-on comparison between
10 children and an adult with a comparative exposure except for
11 the study from Arizona?

12 SUPERVISING TOXICOLOGIST MARTY: Right.

13 PANEL MEMBER BLANC: And you have negative human
14 data suggesting differential responsiveness among people
15 with airway hyper responsiveness so it's difficult to invoke
16 the argument that it's worse for asthmatics and more
17 children are asthmatic and have smaller airways, therefore
18 it would be worse for children?

19 SUPERVISING TOXICOLOGIST MARTY: Right. I don't
20 think we can invoke that argument for formaldehyde.

21 PANEL MEMBER BLANC: So you have good -- on the
22 axis of exposure you have fairly good exposure data. You
23 know that there is a fair amount of exposure out there, but
24 on the preferential childhood sensitivity it's actually this
25 -- I would characterize this evidence as weaker than any of

1 the others in this category and weaker than many of the ones
2 in the Tier 2, far weaker.

3 SUPERVISING TOXICOLOGIST MARTY: Yes, I think you
4 can say that.

5 PANEL MEMBER BLANC: So that having come this far
6 in the process and looking back, I'm looking ahead now to
7 the draft findings, where one of the six is going to go. It
8 seems like to, quote, a new television personality if I were
9 speaking to formaldehyde, "You are the weakest link."

10 (Laughter.)

11 PANEL MEMBER BLANC: Would that be a reasonable
12 summary of the data?

13 SUPERVISING TOXICOLOGIST MARTY: I think that is
14 reasonable. I think it's reasonable and I think it's
15 important to point out that, you know, one of the things we
16 are concerned about with formaldehyde is there's just huge
17 exposure indoor and even outdoor, depending on where you
18 are. And so we felt even though we had just a little bit of
19 evidence that kids might be differentially impacted, we just
20 thought it was an important compound to reckon with.

21 But I would agree with you, out of all those --

22 PANEL MEMBER BLANC: I think it was appropriate to
23 reckon with it --

24 (Laughter.)

25 PANEL MEMBER BLANC: -- but I think once you've

1 reckoned with it and if you think about it logically there
2 is a difference -- you know, something, if there's a lot of
3 it out there and it's been fairly well studied, and this is
4 all the evidence there is for it, that's a different issue
5 than some of the other toxins we've talked about where the
6 biological data in experimental systems are very convincing.
7 But where there's no air data and we're simply assuming that
8 there's not an exposure, but we don't know it and it hasn't
9 really been studied very well in humans, so it would have
10 been hard to see the effect even if it's there.

11 So, you know, if I think of all these things
12 relatively speaking, I'm not quite as concerned about
13 formaldehyde for that reason, because I think something
14 would have appeared. And I think that not only that, but
15 the one study that you have has enough, you know, issues in
16 terms of potential interpretation that to rest as much on it
17 in terms of the differential effect, particularly when they
18 had so much confounding with environmental tobacco smoke.
19 And although they say in the paper that they included that
20 in their predictive model, they don't ever provide the
21 parameter estimates for it.

22 SUPERVISING TOXICOLOGIST MARTY: That's right.

23 PANEL MEMBER BLANC: And the description of the
24 statistical methods imply that they took into account the
25 fact that each person is contributing multiple observations

1 because they have multiple peak flow readings from the same
2 individuals and they used a fixed and random effects model.
3 So I'm assuming that each person was controlled for in terms
4 of how many data points they contributed.

5 But, basically, what you have is, you know, ten
6 people in the highest exposure category, ten people in the
7 medium and then a hundred in the lower. So a very lot is
8 being driven by a very few people and when they show you the
9 raw data all of the asthma in the children are in children
10 who have ETS exposure and formaldehyde exposure.

11 So generalizing to formaldehyde alone is a
12 challenge.

13 SUPERVISING TOXICOLOGIST MARTY: I would agree
14 with that.

15 DR. DAWSON: I would just say that one saving
16 grace is that the other three studies come in at somewhat
17 similar levels of formaldehyde levels.

18 PANEL MEMBER BLANC: But they're not looking at a
19 preferential effect.

20 DR. DAWSON: Yeah, right.

21 PANEL MEMBER BLANC: And they're not actually
22 looking at the effect that we would tend to think mattered,
23 which is we're not really concerned about formaldehyde
24 sensitization, what we're concerned about is the irritant
25 effect of formaldehyde in children, the nonspecific irritant

1 effect. Not the fact that 20 percent of the population has
2 become sensitized to formaldehyde, which is not particularly
3 biologically plausible based on other data.

4 So that really makes me concerned about the
5 Viennese study.

6 SUPERVISING TOXICOLOGIST MARTY: Uh-huh.

7 PANEL MEMBER BLANC: I don't think there's any
8 corroborative data from any other epidemiologic study
9 suggesting that 20 percent of children are sensitized to
10 formaldehyde or even one percent of children.

11 SUPERVISING TOXICOLOGIST MARTY: We didn't find
12 anything.

13 DR. DAWSON: But I would have thought that the
14 Garrett study which has the skin test in it would be
15 somewhat related to the RAST.

16 PANEL MEMBER BLANC: It's hard with an irritant to
17 do skin scratch testing, too. I mean it's really -- it's a
18 very difficult compound to study in terms of specific
19 sensitization and very controversial. All the aldehydes
20 are. Glutaraldehydes presented the same problem.

21 Well, anyway, I think we have consensus. I don't
22 think we need to belabor the point.

23 SUPERVISING TOXICOLOGIST MARTY: Okay. The
24 other --

25 DR. DAWSON: Oh, wait, we're not quite finished.

1 We do have a summary and the first two bullets really just
2 recapitulated what we just said about the Krzyzanowski study
3 and the other three.

4 The third bullet I wanted to add that was the
5 studies of guinea pigs show that airways are hyper
6 responsive. And of course there's the simple hyper
7 responsiveness in the sense of Amdur's 1960 studies of
8 airway resistance and compliance at about 350 ppb. And then
9 the Swiecechowksi study, which gets into both that kind of
10 simple resistance and also the resistance as mediated by
11 acetylcholine and then finally the Riedel study, even more
12 recently, which looks a little bit more at the mechanistic
13 aspects.

14 SUPERVISING TOXICOLOGIST MARTY: We were going to
15 come back to the Panel with a little bit of information on
16 the acrolein versus formaldehyde guinea pig model hyper
17 responsiveness. I'm not sure we need to do that.

18 Okay.

19 PANEL MEMBER GLANTZ: Can I just ask a question.
20 Did we just agree to move formaldehyde down to Tier 2?

21 PANEL MEMBER BLANC: That's what I heard Melanie
22 saying.

23 PANEL MEMBER GLANTZ: Okay, but I want to see if
24 the Panel thinks that.

25 PANEL MEMBER FUCALORO: Based upon differential --

1 or lack of evidence --

2 PANEL MEMBER GLANTZ: For the reasons that Melanie
3 said, I mean, I agree with doing that. Okay.

4 CHAIRPERSON FROINES: Paul, somebody said there's
5 a consensus, which since nobody said anything in opposition
6 to it, I took it as being what --

7 PANEL MEMBER FUCALORO: But that was what the
8 consensus was for, as I understood it, and I agreed and I
9 just repeated what I thought.

10 CHAIRPERSON FROINES: I'll put it affirmatively.
11 There is a consensus of the Panel that formaldehyde be moved
12 to Tier 2.

13 PANEL MEMBER BLANC: Well, it may be consistent
14 with what you had stated earlier, the consensus of the Panel
15 is that we accept the revised recommendation of OEHHA that
16 it be moved to Tier 2, because what you're saying is you're
17 going to revise your document to reflect that
18 recommendation, and I think we would find that
19 scientifically valid.

20 SUPERVISING TOXICOLOGIST MARTY: Right.

21 CHAIRPERSON FROINES: I'm glad you said that,
22 because I really want every motion that we do is basically
23 around our approval or disapproval or critique of the
24 scientific information presented to us. It's your decision
25 about what you list. We simply review the basis of that

1 decision and I want to make sure that on every compound or
2 compounds we talk about that that's the criteria that we're
3 using.

4 PANEL MEMBER GLANTZ: I just want to make one
5 thing clear, though, and this is something we went around
6 and around about in trying to get ready for the meeting.
7 The list, the Tier 1 and Tier 2 lists in the document that
8 was circulated, the June of 2001 draft, are the same lists
9 that have been there for a while. And that table in this
10 report doesn't reflect all of the changes and discussions
11 that have gone forth over the last little while.

12 SUPERVISING TOXICOLOGIST MARTY: That's correct.
13 It's the original table from back in March.

14 PANEL MEMBER GLANTZ: Right and, in fact, I had
15 tried to convince them to put it in blank for that reason.
16 But I think consistent with the discussion the findings that
17 were put in front of you reflect, and correct me if I'm
18 wrong, George and Melanie, reflected OEHHA's recommendations
19 at the time they were drafted.

20 So if the table were to have been redone before
21 this draft document was put forward it would have agreed
22 with the findings.

23 CHAIRPERSON FROINES: I'm sorry, what table are
24 you looking at?

25 PANEL MEMBER GLANTZ: Well, I'm looking at the

1 list of findings. If you look in the document --

2 SUPERVISING TOXICOLOGIST MARTY: On page 37.

3 CHAIRPERSON FROINES: But I'm looking at the most
4 current table that's before us which is a table that --

5 PANEL MEMBER BLANC: Table 1.

6 CHAIRPERSON FROINES: -- is dated -- is Table 1,
7 that lists proposed TACs that disproportionately impact
8 infants and children, and I think this is the table that
9 we're currently operating under.

10 PANEL MEMBER GLANTZ: Okay. Well, the findings
11 are consistent with this table.

12 CHAIRPERSON FROINES: That's correct.

13 PANEL MEMBER GLANTZ: Right. Melanie, --

14 CHAIRPERSON FROINES: And that the findings are
15 not consistent with what's in this document.

16 SUPERVISING TOXICOLOGIST MARTY: That's correct.

17 PANEL MEMBER GLANTZ: That's right. Okay.

18 CHAIRPERSON FROINES: For the Panel we are
19 operating under Table 1.

20 PANEL MEMBER GLANTZ: Okay, which was the separate
21 handout table.

22 SUPERVISING TOXICOLOGIST MARTY: That's right.

23 PANEL MEMBER GLANTZ: Okay and the findings were
24 drafted to be consistent with this table.

25 CHAIRPERSON FROINES: And let me just say that

1 procedurally speaking in Table 1 formaldehyde was listed as
2 Tier 1 and that based on the discussion with the Panel that
3 OEHHA has decided to move formaldehyde from Tier 1 to Tier 2
4 and the Panel agrees with that -- that the scientific basis
5 of that decision is consistent with the evaluation of the
6 Panel.

7 PANEL MEMBER BLANC: And furthermore it appears,
8 Melanie, it appears that if I understand correctly that your
9 current recommendation on the draft Table 1, that in
10 addition to the change that Dr. Froines has just alluded to
11 where the term Tier 2 would replace Tier 1 in the
12 formaldehyde OEHHA recommendation column, you would change
13 the wording on the substances that say possible Tier 1
14 candidate to Tier 1. And on the one that says probable Tier
15 2 to simply say Tier 2, and those would be the other
16 modifications to this table that you'd be proposing to us.

17 SUPERVISING TOXICOLOGIST MARTY: Right, that's
18 correct.

19 PANEL MEMBER BLANC: And is it your proposal then
20 that this table, which is currently labeled Table 1, would
21 essentially be inserted into the document and replace the
22 current table that's on page 37 of the draft document?

23 SUPERVISING TOXICOLOGIST MARTY: That's right.

24 CHAIRPERSON FROINES: Now, Melanie, one further
25 question. I don't know if we're at a place where you are

1 making a new recommendation with respect to acrolein?

2 SUPERVISING TOXICOLOGIST MARTY: Yes, well, when I
3 put that table together for the Panel we wanted -- OEHHA
4 wanted to hear the discussion today and to get a chance to
5 gather up all of the Panel's comments before we formally
6 made a new table with these new proposals. And I'm sorry, I
7 don't have the table in front of me, but I believe I put for
8 acrolein, potential for Tier 1.

9 PANEL MEMBER BLANC: That's why I used the wording
10 I just did, that would take that into account.

11 CHAIRPERSON FROINES: Right. And so what I'm
12 saying is that it seems to me at this point that, based on
13 the evidence that the Panel has reviewed with respect to
14 acrolein, I think that, based on the review of that evidence
15 the Panel is comfortable or would conclude that a decision
16 to move it to Tier 1 would be appropriate.

17 SUPERVISING TOXICOLOGIST MARTY: Okay.

18 PANEL MEMBER BLANC: And the same is true for the
19 other one that is worded possible Tier 1, which is diesel
20 exhaust particulate, and it's also true for the one that
21 says probable Tier 2, which is a change, which is benzene.
22 In other words what you're proposing now is that the word
23 probable be deleted, the word possible candidate be deleted
24 and it simply say Tier 1 and Tier 2 respectively, and
25 formaldehyde where the actual Tier is changed.

1 CHAIRPERSON FROINES: Well, we haven't gotten to
2 diesel yet, so let's --

3 SUPERVISING TOXICOLOGIST MARTY: Okay.

4 PANEL MEMBER FUCALORO: But that's in anticipation
5 of what's going to happen, because now we have five
6 substances in Tier 1.

7 CHAIRPERSON FROINES: But I don't want to start
8 making changes before we've heard the presentation on the
9 chemical.

10 SUPERVISING TOXICOLOGIST MARTY: Okay.

11 PANEL MEMBER BLANC: Are we anticipating another
12 presentation on diesel?

13 SUPERVISING TOXICOLOGIST MARTY: We gave a
14 presentation at the last meeting. We do have a quick
15 overview presentation if you want to see it again just to
16 remind people of where we were.

17 CHAIRPERSON FROINES: I think that the important
18 issue that came up at the last meeting was around the asthma
19 and the adjuvancy if there is such a word, adjuvancy, the
20 adjuvant effects of diesel. And we requested a considerable
21 improvement in the literature associated with diesel to the
22 degree that you felt that was appropriate. And so what we
23 need is a review and a discussion of where you've come to on
24 that issue, because you've obviously evaluated a lot of new
25 information and whatever your perspective, I think it's

1 important for us to hear what that perspective is.

2 SUPERVISING TOXICOLOGIST MARTY: Okay.

3 We did consider that diesel exhaust particulate,
4 that there was a lot of evidence that diesel exhaust
5 particulate enhances allergic response, and this is in both
6 animal models and in humans. And it's by internasal
7 installation as well as by inhalation.

8 So we did have that argument in the original draft
9 of the document and what we did was we expanded discussion
10 of those studies in this draft.

11 So I think we still think that that's a very
12 important issue for diesel and it's probably the strongest
13 piece of evidence for diesel because enhancement of allergic
14 response can mean exacerbation of allergic airway disease,
15 including asthma -- I'm getting ahead of myself.

16 So we initially suggested diesel exhaust be in
17 Tier 2 because of the enhancement of allergic response,
18 evidence of respiratory health impacts in traffic studies,
19 which we reviewed at the last meeting, and because diesel
20 exhaust particulate is a component of PM10 and PM10 has a
21 number of studies associating it with exacerbation of asthma
22 in infant and actually child morbidity and mortality,
23 including respiratory symptomology. And in addition diesel
24 exhaust contains polycyclic aromatic hydrocarbons which we
25 are concerned about and are a candidate for Tier 1.

1 And on top of that, in terms of dosimetry children
2 in the same environment as adults will receive a higher
3 particle dose per lung surface area because of their larger
4 breathing rates and smaller airways and then the dynamics of
5 deposition.

6 There are probably 60 or 70 studies now looking at
7 co-exposure to diesel exhaust particulate, along with
8 allergen, and a number of parameters have been measured that
9 indicate the release of pro-inflammatory --

10 CHAIRPERSON FROINES: Excuse me, just a second.
11 Okay.

12 SUPERVISING TOXICOLOGIST MARTY: So there have
13 been a number of studies and these are studies in people as
14 well as in animals, showing that when you co-expose a person
15 by internasal installation or an animal to allergen and to
16 diesel exhaust particulate that you could enhance the
17 animal's or the person's response to the allergen. And this
18 has been measured in a number of ways, including looking at
19 enhanced IgE and IgG response to the aeroallergen and
20 increases in pro-inflammatory cytokines and chemokines in
21 lavage fluid.

22 There's also studies that have been done that have
23 shown potentiation of histamine release upon exposure to the
24 allergen in the presence of diesel exhaust particulate
25 relative to just exposure to the allergen itself. That's

1 what all of these things are relative to.

2 And all of these things have implications for
3 exacerbating allergic airway disease including asthma. In
4 addition diesel exhaust particulate enhances the development
5 of new allergy and this has been shown in an animal model.
6 And this has implications for increasing asthma prevalence.

7 We also went over some of the traffic studies that
8 have correlated increased respiratory symptoms and decreased
9 lung function in kids to track traffic density to
10 measurements of fine particulate soot in several cross
11 sectional studies. And in one of those studies it appears
12 that the children in the household were more sensitive to
13 the decrements in lung function than the adults.

14 Diesel is a source of PM10. There are numerous
15 studies associating PM10 with exacerbation of asthma.
16 There's a half dozen studies or so looking at neonatal
17 infant and child mortality and found in association with
18 both short-term acute episodes as well as longer term
19 exposures to PM10.

20 There have been studies associating decreased lung
21 function in kids with PM10 exposure and then again children
22 experience higher particle loads per lung surface area than
23 adults breathing in the same environment.

24 Diesel exhaust particulate contains PAHs and
25 nitro-PAHs. These PAHs have been associated with

1 developmental toxicity in a number of studies, including
2 reduced birth weight and dismorphogenesis.

3 There is demonstrations of increased
4 susceptibility of the fetus or neonate to the genotoxicity
5 of PAHs. Insofar as we know PAHs probably contribute to the
6 carcinogenicity of diesel exhaust and the PAHs on the diesel
7 exhaust are bio-available.

8 This is just a quick slide on exposures. The
9 first bullet is our chronic REL of five micrograms diesel
10 exhaust particulate per cubic meter of air. The ambient air
11 is somewhere around two micrograms per cubic meter, a
12 statewide average.

13 The ARB has measurements that show near a freeway
14 they got up to ten micrograms per cubic meter and then in
15 their in-vehicle exposures they got three to twenty-three
16 micrograms or so of diesel exhaust particulate per cubic
17 meter as measured by elemental carbon.

18 So, the point is there are exposures and some of
19 the exposures are higher than our chronic REL.

20 PANEL MEMBER BLANC: So in terms of modifications
21 during your previous presentation there's even more evidence
22 than previously cited in terms of the adjuvant effect or the
23 possible role of diesel particulate in sensitization --

24 SUPERVISING TOXICOLOGIST MARTY: Yes.

25 PANEL MEMBER BLANC: -- and a potential for

1 inducing asthma?

2 SUPERVISING TOXICOLOGIST MARTY: Yes.

3 PANEL MEMBER BLANC: And did you find any
4 additional information on exacerbation of preexisting asthma
5 or that basically is the same data that you had before?

6 SUPERVISING TOXICOLOGIST MARTY: That's basically
7 the same data that we had before.

8 PANEL MEMBER BLANC: I think one other thing that
9 the record should indicate is that there certainly has been
10 discussion today that, not only polycyclic aromatic
11 hydrocarbons that are recommended for Tier 1, but also
12 dioxins may be present in diesel exhaust. I understand
13 there's a big questionmark with that, but since the issue
14 was raised and then confirmed by the later speaker, I think
15 that that has to be taken into note, particularly given how
16 strongly you were on record that any reducible exposure was
17 important even if a small percentage.

18 Another issue, as a minor modification of the
19 document, although it may already be in there, would be in
20 terms of preferential childhood exposure if it's assumed
21 that heavy density roadways are a significant hotspot, as it
22 were, for diesel particulate I think the argument could be
23 elucidated that because lower socio-economic strata are
24 associated with living areas closer to freeways and because
25 the number of children per thousand population are greater

1 in poorer areas that, in fact, disproportionately in
2 California, children would tend to live nearer to freeways
3 than adults, as a percent of the adult population compared
4 to the percent of childhood population. If that argument
5 makes -- I don't know if I've --

6 SUPERVISING TOXICOLOGIST MARTY: Yeah, that makes
7 a certain amount of sense. I think I might add to that that
8 the incidence of asthma in African-American kids is higher
9 than in white kids and they tend to be in a lower level
10 generally.

11 PANEL MEMBER BLANC: But that would be an
12 ideologic leap, but clearly you know -- I think the data,
13 the census data allow you to say unequivocally that more
14 children live near high density roadways. I mean I don't
15 have the data at my fingertips, but I think those data
16 should be available.

17 SUPERVISING TOXICOLOGIST MARTY: We can look for
18 the data.

19 PANEL MEMBER FUCALORO: Based upon the average
20 number of children per thousand in poorer groups --

21 PANEL MEMBER BLANC: So, in summary, therefore,
22 you would say that the biological plausibility, the
23 reproducibility and the strength of the association are
24 present on the scientific side and on the exposure side
25 there is very clearcut exposure to the total population and

1 some evidence, indeed, of higher exposure and higher
2 delivered dose to children?

3 SUPERVISING TOXICOLOGIST MARTY: Yes.

4 CHAIRPERSON FROINES: Peter? Stan?

5 PANEL MEMBER GLANTZ: No, I agree with that and I
6 looked over the rewritten section in the report on diesel
7 and thought it was much better than when we last discussed
8 it. And these issues -- some of these things you just
9 raised Paul are sort of -- aren't in there, but the key
10 points from the presentation are cleared out much -- or
11 spelled out much more clearly than they were in the previous
12 draft.

13 CHAIRPERSON FROINES: Tony?

14 PANEL MEMBER FUCALORO: No, that's fine.

15 CHAIRPERSON FROINES: I'll be the negative side of
16 this comment.

17 There are still references missing, but I'm not
18 going to -- but you've got a lot of references, so I'm not
19 going to raise that further.

20 PANEL MEMBER BLANC: They would only tend to make
21 the argument stronger in your opinion?

22 CHAIRPERSON FROINES: Yeah.

23 But there are some issues that I think I just want
24 to point out that I have trouble with. First, I really do
25 support, because it's research that we're doing, the points

1 about IgE and IgG and other responses, so I would certainly
2 support that.

3 I still, and at the last meeting the entire Panel
4 rejected my point of view and so I'm a minority here, but I
5 really don't agree with this notion that argues that because
6 diesel is a constituent of PM10, therefore, diesel must be
7 causally related to the same effects that are seen in PM10.

8 I think that one can make that argument. I think
9 that that argument is too speculative for me. I can't
10 accept it and so that one I have trouble with.

11 And I just want to mention here. It says "Ostro,
12 et al, 1996." Now in the references that is an Ostro
13 reference to a -- that --

14 PANEL MEMBER BLANC: I thought there were two
15 Ostros.

16 CHAIRPERSON FROINES: Pardon me, that Ostro, 1996
17 is not in the references. It's not there.

18 Secondly Ostro, et al, 1996, for which the
19 reference is missing in a mortality PM time series study in
20 Santiago, Chile cites Sandoval, 1985. Sandoval reference is
21 missing. It's also not there.

22 Diesel vehicles account for 87 percent of black
23 smoke emissions in London," Quarg, 1993. Is there an
24 epidemiologic study that that relates to, because I didn't
25 find it?

1 You don't have to respond, this is stuff that you
2 can follow up on.

3 "Ostro, 1995, found significant associations
4 between PM10 and asthma symptoms." That's a proceedings,
5 it's not a peer reviewed article. We generally --

6 PANEL MEMBER BLANC: You may not be absolutely
7 correct on that, because that's published in "Inhalational
8 Toxicology" and often proceedings in journals are, indeed,
9 peer reviewed, so I wouldn't be too --

10 CHAIRPERSON FROINES: I know that proceedings and
11 I know that conference.

12 PANEL MEMBER BLANC: And it was never peer
13 reviewed?

14 CHAIRPERSON FROINES: And I don't believe it was
15 peer reviewed, no. It's Bob Fallon's conference at U. C.
16 Irvine.

17 Then there is an Ostro 2001 cited for which there
18 is no reference to be found. So that section needs some
19 additional cleaning up.

20 SUPERVISING TOXICOLOGIST MARTY: Okay, my
21 apologies.

22 CHAIRPERSON FROINES: It's all right, it's all
23 right, people make mistakes.

24 Nobody on this Panel has ever made a mistake
25 but --

1 PANEL MEMBER BLANC: Other people might.

2 (Laughter.)

3 CHAIRPERSON FROINES: "In a large study in 12
4 southern California communities, asthma, bronchitis," so on
5 and so forth, and "were associated with PM10 pollution.
6 Though because of pollutant covariation these effects could
7 not be ascribed exclusively to PM," and you quote John
8 Peters' studies.

9 Now I think it's very important to emphasize that
10 the John Peters study demonstrates that there are changes in
11 lung function growth. They do not argue that there are
12 changes in lung function, as your document proscribes. And
13 it's lung function growth and it's important to look at the
14 data, not only in terms of the measures of FEV1, but there's
15 also data now available on MMEF on lung functions that look
16 at airways and those results are somewhat different.

17 But the point I want to make is that what John
18 Peters shows is that there are chronic effects of lung
19 growth in Southern California, in the 12 Southern California
20 communities. He does not demonstrate a causal relationship
21 between those chronic effects in children and any pollutant
22 whatsoever. He shows that there are relationships with
23 PM10, with PM2.5, with acid, with NOX and he shows no effect
24 with ozone, but they also have some data showing some ozone
25 effects.

1 So that what you have -- and clearly all these
2 measurements are correlated with one another, so at this
3 point what you can say is that children are adversely
4 affected, in terms of their lung function growth, by air
5 pollution and we don't know what causes it at this point.

6 So to associate it with PM10 is just not on. It's
7 just not a conclusion, and if you ask John Peters, he would
8 not make that conclusion anymore than I would make that
9 conclusion.

10 So that I think it's not appropriate to suggest
11 that that one finding, where they do find -- they draw a
12 regression line and they see lung function changes, lung
13 function growth changes with PM10, to then ascribe that to
14 being a causal relationship.

15 PANEL MEMBER FUCALORO: Why, because you don't
16 know if it's diesel or PM10?

17 CHAIRPERSON FROINES: You don't have any idea
18 what's causing those lung function growth changes. You
19 don't know if it's nitric oxides, nitric oxides with CO,
20 nitric oxides with acid, nitric oxides --

21 PANEL MEMBER FUCALORO: So they haven't --

22 CHAIRPERSON FROINES: No, they don't know.

23 PANEL MEMBER BLANC: So, John, I just want to try
24 to convert your comments into sort of guidance and see if I
25 understand what you're saying.

1 I think what I hear you saying is that there are
2 so many areas of the diesel story which are so solid in
3 terms of the preponderance of scientific evidence that in
4 the area of PM10 it would be prudent to emphasize the
5 caveats in that particular portion of the argument, because
6 the case does not stand on that. It's an interesting area
7 that warrants further data and as long as you couch it as
8 being speculative, it's certainly worth alluding to and if
9 the document didn't comment on PM10 and some of the PM10
10 data, it would be deficient.

11 But it should simply be perhaps highlighted as,
12 you know, -- although of a speculative nature it is
13 important to note that there have been associations with
14 PM10. Two important pieces we don't have is the diesel
15 contribution to PM10, the important factor in this. And
16 secondly, to a certain extent, the causal association
17 between PM10 and some of these outcomes remains to be
18 established, although there is certainly a great deal of
19 concern that that is the -- am I summarizing your comments?

20 CHAIRPERSON FROINES: Yeah, I would say that the
21 -- my comments are about evidence. They're not subjective.
22 I think that there is significant evidence of enhanced
23 allergic responses and I would certainly agree with you and
24 I think your document has improved, especially in dealing
25 with that area.

1 I think the Brunekreef studies and others on --
2 the traffic studies that are in the literature also support
3 those conclusions.

4 Certainly the PAH, -- the fact that diesel
5 contains PAHs is also evidence that has relevance. And the
6 higher particle dose per lung in children also has
7 relevance.

8 I think that the issue of it being a compounded
9 PM10 and PM10 effects, we have an entire particle center
10 that's studying these kinds of issues at this point and I
11 suspect that diesel, as a component of PM10 may have effects
12 on asthma and other respiratory changes. But I think that
13 in the current document that it is more speculative than I
14 think the evidence would allow for.

15 PANEL MEMBER BLANC: Where it's not explicit
16 enough about those parts of it which are speculative is more
17 what -- you're not saying that it's irrelevant, you're just
18 saying it should be couched appropriately?

19 CHAIRPERSON FROINES: That's right. And I think
20 that they did, by suggesting that there is auto-correlation
21 they do, sort of, acknowledge it. But I think that one
22 could take that a step further.

23 SUPERVISING TOXICOLOGIST MARTY: Okay.

24 PANEL MEMBER BLANC: And also, you know, as the
25 evidence begins to emerge that PM2.5 is even more important

1 that's only going to weigh the diesel argument more
2 strongly, isn't it? Because isn't the diesel particulate
3 even a bigger proportion of the PM2.5?

4 SUPERVISING TOXICOLOGIST MARTY: Yes, it is.

5 CHAIRPERSON FROINES: Well some of us even think
6 that the ultrafine fraction is even more important.

7 PANEL MEMBER BLANC: And won't that even more
8 weight towards diesel?

9 CHAIRPERSON FROINES: Well, but it's -- having
10 spent two days at a gasoline conference, the issues are very
11 complicated.

12 PANEL MEMBER BLANC: I understand, but as long as
13 it's put in the right context I think it's -- it shouldn't
14 be not mentioned, it should just be put in context.

15 PANEL MEMBER WITSCHI: Just out of curiosity,
16 John, how did John Peters hope, in view of the fact that he
17 uses lung growth, which is an integrated measurement over
18 time caused by integrated exposure, how did he ever hope to
19 attribute anything to any part of this mixture, if you are
20 dealing with internal exposure?

21 CHAIRPERSON FROINES: Well, my view of John
22 Peters' study -- I'm a wild enthusiast for John Peters'
23 study. I think it represents one of the most important
24 studies that's ever been done, precisely because it shows
25 long-term effects, chronic effects on lung function growth

1 in children.

2 And I think showing the health outcome is
3 absolutely crucial and important. I think it also
4 demonstrates very clearly how difficult it is to then
5 assign, however, those effects to a specific pollutant.

6 I mean here we are dealing with 10,000 chemicals
7 and obviously how we sort that out is very difficult. I
8 don't think you can sort out the answer to that question
9 using epidemiology. I think the only way you can do it is
10 through hypothesis based studies in the world of toxicology.
11 It's the only thing that gives you a sufficient laser or
12 scalpel to test hypotheses, because epidemiologic studies,
13 as you know, tend to be broad in nature and are not very
14 precise in terms of establishing causal relationships.

15 Now if you're lucky enough the way Stan is you can
16 demonstrate cigarette smoke can do it, but it's -- I'm
17 finished.

18 PANEL MEMBER BLANC: All right. Thanks.

19 Now I think this would be, maybe timing-wise, just
20 a clarification on the form that you see the document taking
21 in terms of the written basis for the Tier 2 substances that
22 were presented orally today, but for which we --

23 PANEL MEMBER GLANTZ: Can I just raise one thing
24 before you get on to that?

25 I just want to absolutely for the record make sure

1 that we've nailed down the lists. Then we can go on to talk
2 about the document.

3 CHAIRPERSON FROINES: No, we're not ready for
4 that.

5 PANEL MEMBER GLANTZ: Okay.

6 CHAIRPERSON FROINES: I appreciate both of your
7 attempts to move this along, but right now we have a very
8 important issue we have to address before we get to this.

9 PANEL MEMBER GLANTZ: Okay.

10 CHAIRPERSON FROINES: And that is that these
11 documents that we've received, including these six
12 documents, this document and the table, Table 1, we've all
13 seen. The public, however, has had a limited timeframe to
14 see it and the public has not been given the opportunity yet
15 to make comments about the quality and nature of these
16 documents. And because of that there's been no response
17 either from OEHHA about the public comments, so what we need
18 right now is to learn as a legal procedural matter how we
19 then proceed from here.

20 PANEL MEMBER FUCALORO: I just keep losing sight
21 of the road map here. Once we hear from this, what do we do
22 then?

23 PANEL MEMBER GLANTZ: Well, let's hear it first.

24 CHAIRPERSON FROINES: I'll tell you what our --

25 PANEL MEMBER GLANTZ: Let her just tell us.

1 PANEL MEMBER FUCALORO: I mean in other words it's
2 uncertain depending upon what's going to be said now?

3 PANEL MEMBER GLANTZ: No.

4 CHAIRPERSON FROINES: No. What she's going to
5 tell us is going to be procedurally how to deal with the
6 question of public input. And I could tell you what I think
7 we should do, but I think that we should get an official
8 position.

9 PANEL MEMBER BLANC: Well, let's hear the
10 presentation.

11 MS. HECK: Thank you. Colleen Heck for OEHHA.

12 What I would envision happening, procedurally from
13 here, would be that, as you pointed out from the Chair, that
14 there has been no formal public comment period on the
15 revised document.

16 In the spirit of the statute and just even a more
17 conservative interpretation of the statute, I think it would
18 be appropriate if there would be a comment period at this
19 time on the revised document. But there's been, in effect,
20 revisions to the revised document during the course of the
21 meeting, so I think what would work best would be if we were
22 to capture the revisions taken orally today in writing, in
23 very quick order, circulate those for public comment,
24 ending, we think what would be feasible would be July 6th
25 would be the proposed close of the public comment period.

1 We would do responses to comments and then send
2 those to the Panel and envision another meeting at which
3 there were a firm list of five being put to the Panel for
4 its approval for its findings and then formal findings made
5 at that time and then the work on this program matter for
6 this portion of it, this first effort would be done.

7 PANEL MEMBER BLANC: How does that impact the July
8 1st deadline?

9 MS. HECK: It concedes that the date will be
10 missed.

11 PANEL MEMBER GLANTZ: Well, I have a problem with
12 that, because the law says July 1st and I don't see how -- I
13 mean first of all I agree -- well, there's a couple of
14 things you said that I find very problematic.

15 Okay, the first is I don't see how a lawyer can
16 get up here and advise the Panel to ignore a deadline set by
17 the Legislature in law.

18 MR. HECK: I didn't advise the Panel to ignore the
19 deadline.

20 PANEL MEMBER GLANTZ: Okay, well, but that's the
21 effect that it would have.

22 The second thing, which I think is more
23 problematic is the implication that because there are some
24 changes to the report that were going to be made as a result
25 of the discussion at the meeting, that that report should go

1 out for public comment. Because, in fact, every report
2 we've ever dealt with since I've been on this Panel was
3 revised as a result of the final discussion by the SRP at
4 the meeting. And I would hate to establish the precedent
5 that any time we change the document at a meeting where
6 we've made findings it had to then go out for public comment
7 again, because we will then create an infinite loop.

8 Now having said that -- so I take quite strong
9 objection to that. Having said that, I do think it's
10 reasonable to, after integrating all of this stuff, allow
11 public comment on the revised document. Not because of what
12 happened at his meeting, but because of the many changes
13 that have been made as a result of the last few meetings.

14 But what I would propose we do in order to respect
15 what the Legislature has said is I would propose that we do
16 adopt the findings today. And then if there is a procedure
17 that the SRP has for amending these things and if material
18 is produced during this public comment period, which leads
19 the Panel to reconsider the findings, then we would just
20 reconsider them in accordance with our standard procedures.

21 You know, given that these compounds are already
22 all identified as toxic air contaminants and have been
23 kicked around quite a lot, I mean I'd be very surprised if
24 any radical new information becomes available, but I do
25 think people ought to have an opportunity, you know, to look

1 at the document and in what is essentially a final form.

2 But that anyway is the document that we'd be
3 moving forward to the Director of OEHHA. So I'm just very
4 reluctant to just blow off -- I mean I realize that the
5 creation of this deadline, as I said before lunch, has made
6 the process more difficult and has led to more preliminary
7 versions of this document being released and discussed than
8 would normally be the case, but I mean I think we need to
9 take an action consistent with the direction we received
10 from the Legislature.

11 MS. HECK: Let me just briefly respond.

12 I did not mean to imply or set a precedent that
13 every revision triggers another public comment period. I
14 was only suggesting that in the sake of efficiency, since
15 changes were, in effect, generated today, they would be
16 rolled into the revisions that we would otherwise be making,
17 so let me clarify that, and thank you for that
18 clarification.

19 The other thing, I neglected to say and I had
20 taken the liberty of making some proposed edits to your
21 draft findings, that they, in fact, if you chose to go
22 forward today, that you go ahead and do that, but make it
23 very clear that they're subject to change if necessary or
24 appropriate after the comment period, that they be somehow
25 cast as provisional or subject to finalization at a later

1 date. I neglected to mention that.

2 PANEL MEMBER GLANTZ: Okay, I don't have a problem
3 with that.

4 MS. HECK: Then lastly, there would just need to
5 be clear at some point that this is an OEHHA list, as Dr.
6 Froines has mentioned consistently throughout today's
7 proceedings, that the Panel is making findings on as opposed
8 to the, you know, the Panel's product, if you will.

9 So with all of those caveats I think we have a
10 road map that we could follow.

11 CHAIRPERSON FROINES: I need to step in here. I
12 think that these are all highly relevant comments. I would
13 suggest that what the panel does today is to -- let me go
14 back one step.

15 I have a problem with accepting these findings. I
16 will not vote to accept the findings, because I haven't read
17 them yet.

18 PANEL MEMBER FUCALORO: Because, I'm sorry?

19 CHAIRPERSON FROINES: Because I haven't read them.
20 I think it would be inappropriate for me to vote for
21 findings that I haven't read yet. I don't know if Paul or
22 Peter or Tony have read them. I suspect that any reading
23 that's been done has been at least cursory and I think that
24 this Panel on such an important issue should not say we're
25 going to accept these findings at the current state of their

1 preparation.

2 So I'm not ready to do that, Stan. That's not
3 where I'm at, because I haven't read them and I'm not going
4 to put my name on something that I haven't read yet.

5 Secondly, I think, though, that the panel can vote
6 to provisionally accept the documents that we have before
7 us, including Table 1, pending a review by the public and
8 pending comments from OEHHA. And that if there is
9 additional new information then the Panel will reconsider
10 its views of the document. But pending no additional
11 information that changes the views of the Panel that the
12 Panel will then finalize its provisional decision, but the
13 Panel won't reach a final decision until we have actually
14 seen the comments and the responses from the public. But
15 that we can vote, provisionally for the draft as it
16 currently exists in order to -- so that the Director of
17 OEHHA --

18 PANEL MEMBER GLANTZ: Which draft? This draft?

19 CHAIRPERSON FROINES: These drafts that we've
20 received, so that the Director of OEHHA is put on notice
21 that the Panel has provisionally accepted the documents as
22 they're currently written. And then at a later time this
23 month we will finalize and vote on the findings, the final
24 findings and send them forward to Director Denton,
25 accordingly.

1 So that's my current view of how we should
2 proceed. I think this Panel should never adopt, make its
3 final determination on the quality of these documents prior
4 to having public input to them, because will raise important
5 issues and we should -- are obligated to review that.

6 So I think that we can, provisionally, accept them
7 with a final conclusion occurring towards the end of July.

8 PANEL MEMBER BLANC: Can I just modify your
9 comment? I think what we have to be referring to in our
10 preliminary approval would be of the data presented today,
11 not of the documents presented today. Because we have an
12 important asymmetry and this is what I was trying to get at
13 and it's appropriate for counsel to comment on this.

14 I believe that the final document, however it
15 comes out and in whatever timetable, has to treat all of the
16 Tier 2 recommendations equally in terms of a textual
17 approach. I am sensitive to what Stan said about the time
18 demands. Therefore I think within the immediate needs of
19 your document what I would suggest is that for the existing
20 summaries of substances which you are now recommending be in
21 Tier 2, you reduce those summaries to two to three pages
22 only, and that for the approximately five substances for
23 which you do not, at this time, have written summaries, but
24 only the slide presentation text, that you draft two- to
25 three-page summaries of those. And that the document that

1 you circulate to the public have such text summaries for all
2 of the Tier 2 substances, otherwise we would be in the
3 position of having text supporting some of the decision and
4 text being absent for some of the other.

5 PANEL MEMBER FUCALORO: And, Paul, you would
6 suggest that they pay particular attention to the issues
7 earlier today regarding why a particular substance ended up
8 in Tier 2, again, whether they affected children more or --

9 PANEL MEMBER BLANC: I think it would be useful to
10 have a summary paragraph at the end of each of those brief
11 discussions, which says that the evidence appears to be
12 quite strong in terms of childhood susceptibility based on
13 reproducibility, strength of the association and biological
14 plausibility, however exposure data are largely absent,
15 although we do have some concerns about hotspots.

16 Actually I think such a summary at the end of the
17 ones that you also cite would be useful as well.

18 PANEL MEMBER FUCALORO: Really useful for the
19 future I think.

20 CHAIRPERSON FROINES: I think it's important to --

21 MS. HECK: I think those are all very helpful
22 comments.

23 CHAIRPERSON FROINES: I think it's important to
24 also pay close attention to the writing of the manganese
25 document, because the one thing -- it was the one chemical

1 that emerged today for which the evidence, if it wasn't for
2 the exposure data, it would clearly be on the first list of
3 five, and so I think it's important to highlight that.

4 The second thing is I want to disagree just very
5 slightly with one comment, which is I made some comments
6 about PM10, but those changes are almost cosmetic, because
7 they refer to references, although I think I also made a
8 more substantive argument about the nature of the PM10
9 argument.

10 But aside from that, I don't think there were that
11 many changes that were proposed by the Panel today, and so I
12 think that the issue of what goes out for public review is
13 your decision to make. I would try and send out a revised
14 document with the diesel argument changed slightly, but I
15 think that some of the other documents I think are already
16 out in the public.

17 PANEL MEMBER BLANC: But the brief summaries of
18 all of the Tier 2 have to be in there and they don't
19 currently exist.

20 PANEL MEMBER GLANTZ: Yeah, but I'd like to just,
21 you know, I mean I think what you're suggesting, Paul, in
22 terms of symmetry would be ideal.

23 PANEL MEMBER BLANC: I think it's necessary, I
24 don't think it's ideal. I think for it to be a
25 scientifically valid --

1 PANEL MEMBER GLANTZ: I don't see what's to be
2 gained.

3 PANEL MEMBER BLANC: Because how am I supposed to
4 comment on a nonexisting document?

5 PANEL MEMBER GLANTZ: No, no, I'm not, wait, wait,
6 wait. I'm not disagreeing with what you said about the
7 material to be added.

8 PANEL MEMBER BLANC: Yes.

9 PANEL MEMBER GLANTZ: But I don't see where we
10 gain, you know, anything of substance by going through and
11 trying to rewrite to shorten the stuff that's already there
12 just in the interest of esthetics, because then what --
13 first of all the resources available to OEHHA to do all of
14 this are limited by the number of hours in the day.

15 I think it's very important that this be moved
16 expeditiously and if you start whacking stuff out of those
17 sections which people are already comfortable with then you
18 raise the specter of someone getting mad that something was
19 deleted. And I frankly don't see what you lose by providing
20 more rather than less information.

21 PANEL MEMBER BLANC: Then I'll make clear why I
22 think there's something to be -- why it's not just cosmetic.
23 And that is because it gives the impression that you've
24 actually given more attention to some chemicals than you
25 have to others. And I think it's very important that the

1 written record be consistent and my impression from Melanie
2 was that, in fact, such a synopsis or summary of the
3 existing five other longer documents was not a burden that
4 would be excessive.

5 PANEL MEMBER FUCALORO: Well, that's the question.
6 Stan, I recognize your concern, but they're the ones that
7 actually have to do it and they know what personnel they can
8 devote to it. Does this seem to be a very onerous task
9 that's being asked of you?

10 CHAIRPERSON FROINES: Do you think we'll get a
11 single answer here?

12 PANEL MEMBER FUCALORO: Well, take the time you
13 need.

14 PANEL MEMBER BLANC: Della Street is talking to
15 Perry Mason, the question is which is which.

16 (Laughter.)

17 SUPERVISING TOXICOLOGIST MARTY: I'm looking at
18 the length of the Tier 2 summaries that we already have and
19 it's variable. Some of them are pretty short already. And
20 if you say three to four pages, excluding references, we're
21 actually almost there for some of them.

22 PANEL MEMBER BLANC: Okay.

23 SUPERVISING TOXICOLOGIST MARTY: I think in terms
24 of workload, yeah, you know, it's another thing we've got to
25 do before we get the document out the door and George is

1 already mad at me for not giving him enough time to read
2 through this stuff. But, you know, I don't want to spend a
3 whole lot of time hacking at them, but I think we can do it.

4 And also in terms of the chemicals that we talked
5 about today, we actually already had very early drafts on
6 those which we didn't take through the process, so we
7 already have something written for those. We have to fix it
8 a little bit, but I think I can get them all to
9 approximately, you know, plus or minus however much data
10 there is for each one, ten pages.

11 CHAIRPERSON FROINES: The answer we got was yes.
12 Thank you very much. That's great.

13 And George is nodding his head for the --

14 PANEL MEMBER FUCALORO: He's in a coma.

15 (Laughter.)

16 SUPERVISING TOXICOLOGIST MARTY: I'm looking to
17 see if staff is drawing any weapons.

18 (Laughter.)

19 CHAIRPERSON FROINES: Now I think we need a motion
20 to provisionally accept the data. You make the motion since
21 you, I think, stated it better than I did.

22 SUPERVISING TOXICOLOGIST MARTY: John, we were --
23 did you want us to talk more about PAHs or were you guys
24 okay with that?

25 You're okay with that.

1 CHAIRPERSON FROINES: I don't think so. I think
2 that we're prepared to accept the PAHs. If you want to
3 convince us not to, you can do that, but I think you're
4 heading in the wrong direction.

5 SUPERVISING TOXICOLOGIST MARTY: Okay.

6 CHAIRPERSON FROINES: Let me say it more
7 explicitly. I think that the evidence that's been presented
8 on PAHs in the past is sufficient in terms of the Panel's
9 review for the Panel to accept the data that was contained
10 within the documents.

11 PANEL MEMBER GLANTZ: And naphthalene.

12 CHAIRPERSON FROINES: Including naphthalene.

13 PANEL MEMBER BLANC: I would like to move that the
14 Panel provisionally accept the data presented today by OEHHA
15 in text and in slide presentation and specifically that we
16 accept the modified draft Table 1 as presented with the
17 noted deletions of the words "possible candidate" or
18 "probable" and the substitution of the words Tier 2 for Tier
19 1 for formaldehyde.

20 PANEL MEMBER GLANTZ: Second.

21 MS. HECK: Was there any mention of diesel moving
22 from Tier 2 to Tier 1?

23 PANEL MEMBER BLANC: I just covered that. I
24 said -- it was already said possible Tier 1, so by -- it
25 said possible Tier 1 candidate, so by deleting the words

1 possible and candidate it becomes Tier 1.

2 CHAIRPERSON FROINES: Is there any discussion?

3 PANEL MEMBER GLANTZ: Could I just, again to avoid
4 any possible confusion, let me just read what I understand
5 the list to be.

6 For Tier 1 would be lead, acrolein, dioxins,
7 polycyclic aromatic hydrocarbons, and diesel exhaust
8 particulate. And Tier 2 would be formaldehyde, arsenic,
9 benzene, carbon disulfide, glycol ethers, manganese,
10 mercury, methylene chloride, methyl bromide, PCBs and vinyl
11 chloride.

12 CHAIRPERSON FROINES: Nonplanar PCBs.

13 PANEL MEMBER GLANTZ: Okay.

14 PANEL MEMBER BLANC: Yes, as a point of
15 information, that is consistent with the resolution as put
16 forward.

17 PANEL MEMBER GLANTZ: I second the motion.

18 CHAIRPERSON FROINES: All in favor?

19 (Ayes.)

20 CHAIRPERSON FROINES: Opposed?

21 PANEL MEMBER BLANC: The minutes should show that
22 it was unanimous.

23 Now I'd like to make a suggestion in light of the
24 resolution that was just adopted that the Chair work on
25 draft findings, tentatively consistent with the thrust of

1 that resolution and circulate it for comment to the Panel
2 Members so that when the final document has been reviewed we
3 are in a good position to modify that accordingly.

4 PANEL MEMBER FUCALORO: Now these findings will be
5 based upon what we have here, right?

6 PANEL MEMBER BLANC: Yeah, at the Chair's
7 discretion.

8 CHAIRPERSON FROINES: Well, up to now what's
9 happened is Stan has worked with Melanie to generate the
10 first draft of the findings. And as far as I'm concerned I
11 think that the next draft of the findings should come from
12 Stan and Melanie.

13 PANEL MEMBER GLANTZ: Yeah.

14 CHAIRPERSON FROINES: And then I'll take it and
15 review it before --

16 PANEL MEMBER GLANTZ: There was just a screwup,
17 because these should have been circulated to the Panel in
18 advance and I thought Melanie was sending them around and
19 she thought I was sending them around.

20 PANEL MEMBER BLANC: Well, Stan, if you're going
21 to be taking the lead on it, let me just point out a few
22 things then.

23 PANEL MEMBER GLANTZ: Okay, well, you can do it
24 afterwards, unless you want it on the record.

25 PANEL MEMBER BLANC: I think I'd like it on the

1 record. This won't take along.

2 As you've, yourself, alluded I'd like to see a
3 parallel comment to the comment on ETS in terms of
4 pesticides. You've already mentioned that yourself. And
5 I'd like you in the Tier 2 compounds to highlight the ones
6 that the sense of the Panel was that there was the most
7 concern for.

8 And I'd like the polycyclic aromatic hydrocarbon
9 findings to be less weighted towards carcinogenicity and to
10 include the noncarcinogenic effects that were discussed,
11 which had to do with, in particular, that there be specific
12 discussion of naphthalene in the polycyclic.

13 So I'm just reiterating things that were --

14 PANEL MEMBER GLANTZ: Okay. I think that's all
15 fine.

16 PANEL MEMBER BLANC: And I think that also what
17 would be useful would be to follow the same advice for the
18 findings as we suggested that they have that it comment on
19 these two axes in terms of the evidence.

20 PANEL MEMBER GLANTZ: Differential. I see,
21 differential and exposure.

22 PANEL MEMBER BLANC: Right.

23 PANEL MEMBER GLANTZ: That's all fine.

24 PANEL MEMBER FUCALORO: Because all that's in the
25 record now, so I think that makes it very simple.

1 PANEL MEMBER GLANTZ: We don't have a quorum
2 anymore or we could adjourn.

3 PANEL MEMBER BLANC: He's just gone out for a
4 second.

5 So can I clarify then the timeline? The timeline
6 will be that the final -- there'll be a final face-to-face
7 public meeting to comment on the public comments or to
8 address the public comments, is that the intention?

9 MS. HECK: That's the idea.

10 SUPERVISING TOXICOLOGIST MARTY: Hopefully by the
11 end of July.

12 CHAIRPERSON FROINES: I'm sorry.

13 PANEL MEMBER BLANC: I was just clarifying that
14 there is going to be a follow-up face-to-face meeting in
15 which OEHHA will be commenting on the public comments and
16 we'll be responding to OEHHA's comments on the public
17 comments.

18 SUPERVISING TOXICOLOGIST MARTY: You will have had
19 written responses.

20 CHAIRPERSON FROINES: And I'm hoping that that can
21 occur in July, towards the end of July. But does that work,
22 Colleen, in terms of the public having enough time to
23 comment?

24 MS. HECK: We think, given how many iterations
25 there have been and the fact that the stuff can be readily

1 gotten out there fairly quickly that it's about three weeks
2 time that that should be adequate.

3 PANEL MEMBER GLANTZ: You mean from today?

4 MS. HECK: We're envisioning, yeah, that the close
5 of the comment period would be in approximately three weeks.

6 CHAIRPERSON FROINES: So the public has the
7 documents that we have, so if we go until something like
8 July 15th that will be at least three to four weeks of --

9 PANEL MEMBER GLANTZ: Well, I think she had said
10 July 6th.

11 MS. HECK: We were thinking July 6th, because we
12 need to do responses and get them to you and to prepare for
13 the meeting.

14 PANEL MEMBER BLANC: They'd end the public comment
15 period July 6th and then we would meet two weeks later.

16 MS. HECK: Right.

17 PANEL MEMBER BLANC: There has to be a period for
18 OEHHA to respond to the public.

19 MS. HECK: Right, otherwise there would be no time
20 to do the responses and circulate them to the Panel.

21 PANEL MEMBER GLANTZ: Yes, but I think if they can
22 do it, then we should.

23 CHAIRPERSON FROINES: I think this is awfully
24 important and I'd like to make sure the public has
25 sufficient time to comment. So if there could be a few days

1 more I wouldn't -- and I think it would be in our best
2 interest.

3 SUPERVISING TOXICOLOGIST MARTY: That's fine, I
4 think so too.

5 Let's make the Panel Meeting really the last part
6 of July, instead of just kind of the last part of July.

7 PANEL MEMBER GLANTZ: Like the 18th or something?

8 MS. HECK: Closer to the 31st.

9 SUPERVISING TOXICOLOGIST MARTY: 29th, 30th, 31st.
10 I don't have a calendar, so I don't know what days those are
11 in the week.

12 PANEL MEMBER GLANTZ: All right. Well, do we have
13 any other business?

14 CHAIRPERSON FROINES: No, I think -- I hope you
15 agree that having the discussion on administrative matters
16 would be best if everybody were here?

17 PANEL MEMBER GLANTZ: Uh-huh.

18 CHAIRPERSON FROINES: In which then there's
19 nothing more for today. So we'll entertain a motion to
20 close.

21 PANEL MEMBER GLANTZ: I so move.

22 PANEL MEMBER BLANC: Second.

23 CHAIRPERSON FROINES: All in favor?

24 (Ayes.)

25 CHAIRPERSON FROINES: We're done. Thank you.

1 (Thereupon the Air Resources Board
2 Scientific Review Panel Meeting for June
3 15, 2001 was adjourned at 3:20 p.m.)
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CERTIFICATE OF REPORTER

I, JAMES RAMONS, an Electronic Reporter, do hereby certify that I am a disinterested person herein; that I recorded the foregoing Air Resources Board Scientific Review Panel Meeting; that it was thereafter transcribed into typewriting.

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

IN WITNESS WHEREOF, I have hereunto set my hand this 25th day of June, 2001.

JAMES RAMOS

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