MEETING
STATE OF CALIFORNIA
OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT
PROPOSITION 65
CARCINOGEN IDENTIFICATION COMMITTEE

JOE SERNA JR./CALEPA HEADQUARTERS BUILDING
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COASTAL HEARING ROOM
SACRAMENTO, CALIFORNIA

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PETERS SHORTHAND REPORTING CORPORATION (916) 362-2345
APPEARANCES

COMMITTEE MEMBERS
Dr. Thomas M. Mack, Chairperson
Dr. David A. Eastmond
Dr. James S. Felton
Dr. Martin L. Hopp
Dr. Darryl Hunter
Dr. Anna H. Wu

STAFF
Dr. Joan E. Denton, Director
Dr. George Alexeeff, Deputy Director
Ms. Carol Monahan-Cummings, Chief Counsel
Ms. Amy Dunn, Cancer Toxicology and Epidemiology Section
Ms. Fran Kammerer, Staff Counsel
Ms. Cynthia Oshita, Proposition 65 Implementation
Dr. Martha S. Sandy, Chief, Cancer Toxicology & Epidemiology Section
Dr. Rajpal S. Tomar, Cancer Toxicology and Epidemiology Section
Dr. Lauren Zeise, Manager, Reproductive and Cancer Hazard Assessment Branch

ALSO PRESENT
Dr. Dale Gieringer, California National Organization for the Reform of Marijuana Laws

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PETERS SHORTHAND REPORTING CORPORATION (916) 362-2345
CHAIRPERSON MACK: Okay. Let's get started.
We should have a relatively efficient meeting today.

Joan, would you please begin the ceremonies.

DIRECTOR DENTON: Thank you, Dr. Mack.

Unfortunately, Dr. Hamburg and Dr. Landolph will not be able to join us today because of weather down in the south coast. But I would like to introduce the Panel members.

Starting at my far left is Dr. David Eastmond from UC Riverside, Dr. Tom Mack from USC Keck School of Medicine. To my right is Dr. Jim Felton from Lawrence Livermore National laboratory and UC Davis; Dr. Anna Wu, who's a professor at the Department of Preventive Medicine at USC. Next to Dr. Wu is Dr. Hopp, who's President and Chief Executive Officer of the Tower Ear, Nose, and Throat. And then at the far end is Dr. Darryl Hunter, who's a physician with Kaiser Permanente.

So, with that, Dr. Mack, I guess welcome to everybody and I'll turn it back to you.

CHAIRPERSON MACK: All righty. Today we're not actually deciding whether or not compounds should be listed. We're simply evaluating the superficial review of the compounds which showed some evidence of an
epidemiologic relationship with cancer. And we're being asked to decide whether or not we should proceed with the full -- I'm trying to think of a funny word, but I can't think of one -- the full review, anyway, of those three compounds, which would entail a much more detailed search and a much more careful, I'm sure, reading of each of the documents which have come up.

Martha, why don't you begin the formal proceedings.

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: Sure. Thank you, Dr. Mack.

Just to remind everyone, and including many of the members of the Carcinogen Identification Committee who are new, relatively new to the process, back in about 2002 your Committee asked OEHHA to look into streamlining and improving the prioritization process that we were using in order to identify chemicals that seemed to pose a significant hazard to Californians. And so we embarked on this process. We had two members from your Committee, one of whom is Dr. Joseph Landolph, who's unfortunately not here today, and we had some input from members of the DART Identification Committee, and we came up with a revised prioritization process. This is a process whereby we look at the chemicals we're tracking that have some indication or have been nominated as a concern and that we should
look at for possible listing under Proposition 65.

So let me start with the presentation here. I'm going to go over -- give you a brief overview of the prioritization process and then discuss the application of the epidemiology data screen, which we have applied in this round of prioritization.

(Thereupon an overhead presentation was Presented as follows.)

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF SANDY: So as I mentioned, we came up with a revised process. The title of the document is up here on your screen. You should have all had a copy of that. It came out in December of 2004. As I said, it was developed in consultation with your Committee and with the DART Identification Committee.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF SANDY: And the purpose of prioritization is to identify chemicals for evaluation by the Carcinogen Identification Committee.

Our goal of this process is to focus the efforts of your Committee on chemicals that may pose significant hazards to Californians.

And as Dr. Mack has just said, prioritization is a preliminary appraisal of the evidence of hazard. It's a
screening level, relatively quick look at the evidence. The more thorough comprehensive evaluation of the evidence would occur in the hazard identification development phase when we prepare materials for a listing decision. So that would be later.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: So this slide is taken from that prioritization process document. And it shows you the different steps in prioritization. We have a tracking database of chemicals that have been nominated to be looked at for possible listing. And then within that database there's a subset of chemicals we call candidate chemicals. Those are chemicals for which there's a suggestion that there's a potential for exposure to Californians and there's some evidence suggesting a cancer concern.

To that group of candidate chemicals we will be applying different screens to identify chemicals that we should look at in more depth. Those screens will involve focused literature reviews. And in this particular case we've applied an epidemiology data screen.

Then the results of that screening process give us chemicals that are proposed for Committee consideration. And that green asterisk there is meant to
indicate that we release those proposed chemicals to the
public and there's an opportunity for public input on
them. And then we bring them to you, as we are today, for
consultation on these chemicals. And, again, there's an
opportunity for public comment.

The last step here in the process is when OEHHA
takes into account the advice we receive from your
Committee, input from the public, and OEHHA makes a
selection of the chemicals that we will prepare hazard
identification materials on.

So that's the prioritization process.

Then I wanted to show you the hazard
identification process.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF
SANDY: After OEHHA has selected chemicals, we
will issue a data call-in on those chemicals requesting
data submitted to us from the public.

We'll then develop an in-depth comprehensive
review of the evidence of carcinogenicity on those
chemicals. And that is assembled and is referred to here
as hazard identification materials. We'll present those
to the Committee. And there's also an opportunity for
public comment.

And then we'll have a Committee meeting where
you'll review the evidence and make a listing decision.

But right now we're addressing that first process, the prioritization process.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: And just a little more information. The tracking database, chemicals that OEHHA staff find through literature searches, also suggestions from your Committee, other state agencies, the scientific community, and the general public, all those sources of information can result in a chemical being tracked for carcinogenicity.

Among the tracked chemicals we have what we call candidate chemicals. Again, those are chemicals with data suggesting the potential to cause cancer and the potential for exposure in California.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: Now, this schematic is meant to represent the candidate chemicals we have in our cancer tracking database. We have a variety of chemicals with a variety of amounts and types and quality of data suggesting carcinogenicity. And as you can see, some chemicals we may know quite a bit right now, others we may have only been aware of positive short-term studies. But once we look at them a little more carefully, we may find that

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there's more information. And there's more information being generated all the time. So this is a fluid process. But the question we have is: How do we -- we have over 200 chemicals. What technique do we use to reach in and grab a few of them and look at them a little more closely and decide which ones have a higher priority and should be brought to your Committee's attention? And that's the -- the approach that was developed in our process document is to apply a focused literature screen.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: So we've applied this epidemiology data screen. We had 235 candidate chemicals or groups of chemicals for which we did this. And the process involved conducting an online literature database search using TOXLINE and PubMed.

We then were focused on looking for epidemiology data. We identified those chemicals that had epidemiology studies reporting an association between exposure to the chemical and an increased cancer risk.

We gave more weight to analytical studies than to descriptive studies or case reports.

And a single case report was not sufficient to satisfy the epidemiology data screen.

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SANDY: So as a result of this screening process the chemicals that were identified we then looked at in more depth but still in a preliminary fashion. We did what we call a preliminary toxicological evaluation. By doing another literature search to identify animal cancer bioassays, studies on genotoxicity, mechanisms of action, metabolism, pharmacokinetics and other things, we took a look at those studies sometimes looking only at abstracts, sometimes just a quick look at the paper to do a preliminary evaluation of the overall evidence of carcinogenicity.

--o0o--

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: And then through that process we arrived at the chemicals that we're proposing for your consideration. And we have three that were identified through this process: Marijuana smoke, Dimethylformamide, and 2,4,6-Trinitrotoluene.

--o0o--

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: And for each of the proposed chemicals OEHHA compiled key cancer epidemiology studies, animal cancer bioassays, and other relevant data identified
during this preliminary toxicological evaluation. We provided that compilation of studies or abstracts or a combination of those to your Committee. We released them to the public as well for a 60-day comment period.

And any comments that were received were sent to you prior to today's meeting. And we had comments to my knowledge only on one chemical, and that was marijuana smoke.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: So today we're here to allow your Committee to advise OEHHA on the chemicals that you think should undergo the development of hazard identification materials. Again, this would be a much more thorough, comprehensive literature search and evaluation of the studies that we find.

And there's again today an opportunity for additional public comment.

So now I'll turn it over to Amy Dunn, who'll be going over in very brief fashion the information that was compiled on Dimethylformamide and TNT.

(Thereupon an overhead presentation was Presented as follows.)

MS. DUNN: Good morning.

Today I'll be presenting the evidence that we
identified that was available for prioritization of N,N-Dimethylformamide and 2,4,6-Trinitrotoluene. I'll stop after the presentation of Dimethylformamide for the Committee's discussion.

Next slide.

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MS. DUNN: As Martha mentioned the available evidence for consideration included the epidemiological data that was identified during the human data screen as well as animal carcinogenicity data and other relevant data.

Next slide.

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MS. DUNN: N,N-Dimethylformamide is a solvent used in fabric and fiber production, including leather production. It's also used in industrial paint stripping and other solvent applications.

The available human studies include studies of three different types of occupationally exposed groups: Aircraft repairmen, leather tanners, and workers in DMF production and use facilities.

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MS. DUNN: The human studies of Dimethylformamide include investigations of testicular cancer in two different occupationally exposed groups. There's a
cluster investigation of aircraft repairmen of a particular type of aircraft called the F4 that uses Dimethylformamide in a repair process, that was conducted by Ducatman, et al., in 1986.

In addition, within the leather tanners there’s a case series conducted by Levin, et al., in 1987; a case control study in leather tanners conducted by Frumin, et al., in 1989; and a cohort study in leather tanners conducted by Frumin, et al., in 1989 and Calvert, et al., in 1990.

These were all -- all the leather tanner studies were a follow-up on the original cluster that had been identified.

In addition, a range of cancers were investigated in workers in DMF production and use facilities in a case-control study by Walraith, et al., in 1989 and a cohort study by Chen, et al., in 1988.

MS. DUNN: The animal carcinogenicity data includes two-year bioassays conducted by Senoh, et al., in 2004 in rats and mice, as well as 18-month inhalation bioassays conducted by Malley, et al., in 1994 in rats and mice.

MS. DUNN: The other relevant data available on
Dimethylformamide includes genotoxicity evidence reviewed by IARC in 1999 and a review by IARC that same year. However, the review by IARC does not include the two-year inhalation animal bioassay conducted by Senoh, et al., in 2004.

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MS. DUNN: That concludes my presentation of the evidence available on Dimethylformamide.

CHAIRPERSON MACK: First of all, I want to say I think the three chemicals that you allowed to float to the top of the list are very pertinent and I think they're a good start. And the reason they're a good start is because they're in very wide distribution and there are legitimate reasons for concern on the part of the public. Whether or not these are scientifically based or not is to some extent irrelevant in terms of the reasons why we should take a close look at them.

Now, I'm sort of assigned myself the job of looking at Dimethylformamide for beginners. And basically what we've got here is three clusters and they're clusters of the same kind of cancer, testicular or at least germ-cell-type cancers. And they happened in three separate circumstances because they were two of the airplane -- there were two clusters in separate airplane facilities. And that's disturbing, because it's the same
kind of tumor and they happened in clusters three times. Normally one cluster is easy to dismiss because the likelihood of having a cluster in all the people available to report is very, very high. Having a second one in the same occupation with the same outcome is more bothersome. And having a third one with the same outcome in a comparable exposure is also bothersome.

On the other hand, the lack of confirming evidence from animals and the lack of confirming evidence from the more formal analytic studies is of course inconsistent with the potential.

Now, Ducatman wrote a letter to the editor, which I thought was a very thoughtful letter to the editor, pointing out the characteristics of this particular solvent and suggesting the possibility, that I don't think we can exclude, that it may actually offer an opportunity for another carcinogen to get at the cell more efficiently. In other words, it works a little bit like alcohol might work in providing access to carcinogens.

Now, to me, with my definition of what is a carcinogen, anything which you've taken away reduces the risk of cancer is a carcinogen. So the fact that it works that way doesn't make any difference whatsoever to me. But what we're lacking here so far is any real strong evidence in animals that have actually looked at
this possibility, that is to say, the joint exposure to a heavy metal in this case, because both chromium and cadmium are the ones that Ducatman was concerned about. And that means that we to some extent might dismiss or at least not be completely convinced by either the analytic studies where the solvent was looked at alone or the animal studies where it was used only alone.

So I still think there's a possibility that that might be the explanation and that this might be a carcinogen. And on that basis, my inclination is to say, yes, this is worthy of a further look, even though as the evidence that we've been given now I don't think would be able to call it a listable carcinogen.

Now, having said all that, let me turn to everybody else on the Committee and see if anybody --

COMMITTEE MEMBER EASTMOND: I was asked also to look at this compound in a little more detail. So I spent some more time on it. And there are some -- certainly the human data receives a series of clusters or case reports which are suggestive. However, the cohort studies, certainly those from DuPont, don't seem to support the same sorts of associations.

The animal carcinogenicity data is actually a little more messy. There was an early study published in
1994 in mice and rats in which essentially they reported that there was really no increase in tumors, particularly tumors of the liver. Now, the rat study was done up to a concentration -- these were inhalation studies, done up to 400 parts per million exposure in the rats for two years and in the mice for a year and a half. And there was an increase in uterine tumors seen within the rats, but they considered this within historical range. So it was largely considered negative.

However, in 2004, the Japanese Bioassay Research Group repeated these types of studies in both mice and rats of a different strain. And in the rats -- both the rats and the mice, now they went up -- in the rats it was a two-year study again -- they went to a higher concentration. So they went up to 800 parts per million. And in the mice went to the same concentration, but they went from 18 months and they now extended this to a two-year study. And it was a very strong carcinogen in the liver in both males and females and in both the mice and the rats.

Some of these would appear to be almost contradictory studies. The earlier study from 1994 published DuPont by Malley, which superficially doesn't look like they saw anything. Although, as you start teasing this out, what does appear is there are increases
in hepatic foci seen, which is thought by many to be a
preneoplastic lesion. And there looks like kind of a
tendency. So these aren't totally in contradiction. The
obvious differences are strain differences between the two
different strains of mice and rats. But there is some
suggestion at least in the Malley study that some
preneoplastic things might be occurring.

In the Japanese study by Senoh it's a very strong
positive only in the liver. They said they saw no
increase in tumors in any other tissue but the liver. But
they saw both adenomas and carcinomas in the rats. And in
the mouse they saw adenomas, carcinomas, and
hepatoblastoms. So it's unusual in this regard.

With regards to other relevant data,
genotoxicity, this compound has been extensively studied.
It was chosen for a series of test batteries in early
1980s. Largely negative in almost all of them. So it's
essentially negative. There are a couple of positives,
but the general overall pattern is very consistently
negative for genotoxicity.

So we have kind of a strange situation here where
you have agents got conflicting results in rodent studies.
One's a very strong positive in both mice and rats in both
males and females. The other one's largely negative. But
those two may be not so inconsistent when you start
looking at them in a little more detail. And then you've
got mixed pattern within the epidemiological data.

So I mean I think, given Tom's comments, the
animal data in itself is probably suggestive additional
examination is probably merited. It would be useful to
ty and figure out what type of mechanisms it might be
acting by, because what appears to be happening in the
liver is certainly not through -- it does not appear to be
through a standard sort of mutagenic mode of action. So
it would be useful to know -- have other information on
how this might be acting.

CHAIRPERSON MACK: Thank you, David.

Now, let's go to survey the other people.

Why don't we start with Darryl.

No, wait a minute. No, Jim has a comment first.

Go ahead.

COMMITTEE MEMBER FELTON: I just have two
concerns. One is the at-risk epidemiology populations are
just being exposed to so many chemicals besides this one.
So we've got that on one side where we've got all these
different exposures. And maybe like Tom says, this thing
is potentiating those other compounds, because both
leather workers and engine maintenance people are getting
a big dose of various carcinogens.

And then on the other hand we do the animal
studies and we throw away all the other compounds and we
look at this one alone. So, again, if Tom's right, we
don't do animal cancer studies the way you'd really like
to see them, is take a few of those other exposed -- or
chemicals the individuals are exposed to and give them
together with this N,N-Dimethylformamide.

So it seems to me we're not doing things quite
right, but we've got what we got.

CHAIRPERSON MACK: Okay. Darryl, do you have any
comments?

COMMITTEE MEMBER HUNTER: No.

CHAIRPERSON MACK: None?

COMMITTEE MEMBER HUNTER: Not on this point.

CHAIRPERSON MACK: Okay. Martin.

COMMITTEE MEMBER HOPP: Well, I agree the bigger
problem is these cohorts of groups of patients who had --
I think the identified patient groups that had what I'd
consider very significant tumors that are unusual would
warrant evaluation, and I think it still warrants
evaluation.

The problem is that the individual chemical, as
you say, one by itself may not be causing it but multiple
exposures. It's very disturbing to see these groups of
patients and their tumors.

And I was also very disturbed at this higher
exposure. There's very clearly a higher exposure. This
was carcinogenic. But all in all the data just seems to
be still very weak but very disturbing.

CHAIRPERSON MACK: So we may wind up still not
being able to say that this is a listable chemical. But I
think, in my opinion, it probably behooves us to think
seriously about looking.

Now, I have a more formal way to go through this,
as we usually do. So I'm going to read a preamble. And
then I'm -- Oh, I'm sorry. I always forget the public.
I apologize to the public.

(Laughter.)
CHAIRPERSON MACK: Does anybody wish to say
things?
My God.
(Laughter.)
CHAIRPERSON MACK: Jay, you're unconscionably
quiet.
(Laughter.)
CHAIRPERSON MACK: Okay. Thank you.
Now, I will read a preamble and then I will say,
"Do you advise OEHHA to begin preparation of the hazard
identification materials for N,N-Dimethylformamide?" And
you will respond "yes" with your hands or "no" with your
hands.
So here we go.

"The Carcinogen Identification Committee is being asked whether OEHHA should prepare hazard identification materials for any of the chemicals presented today and be brought back to the Committee at a future meeting for our consideration making a listing decision."

"We are not making any listing decisions at today's meeting. With this in mind, I will poll the Committee members for their advice to OEHHA concerning these chemicals.

"Do you advise OEHHA to begin preparation of the hazard identification materials for N,N-Methylformamide? All those advising against, please raise your hand."

Oh, yes, yes.

(Hands raised.)

CHAIRPERSON MACK: I will read it again, because apparently some members of the Committee have lost it.

"Do you advise OEHHA to begin preparation of the hazard identification materials for N,N-Methylformamide?"

All those advising "yes" please raise your hand.

(Hands raised.)

CHAIRPERSON MACK: Do you advise them to do this?

(Hands raised.)

CHAIRPERSON MACK: Jim is having trouble?

No? Okay.
So we have 1, 2, 3, 4, 5 yea's.
All those advising "no" please raise their hand.
(Hand raised.)
CHAIRPERSON MACK:  One.
So the vote is 5 to 1.
And I presume that means that the motion probably
passes.
All right. Dr. Dunn, the field is yours again.
MS. DUNN:  Next slide.
--o0o--
MS. DUNN:  2,4,6-Trinitrotoluene, also known as
TNT, is a well known explosive used in military and
industrial applications. Exposure to TNT may occur during
production, during the manufacture and loading of
munitions, during blasting operations, and from water or
soil that has been contaminated by discarded munitions or
manufacturing waste.
The available human studies of TNT include two
types of exposed groups: Individuals living in an area
that has contaminated soil and water; and factory workers
making ordnance.
--o0o--
MS. DUNN:  The human studies of TNT in
individuals residentially exposed have focused on leukemia
incidents. A case-control study was conducted by Kilian,
et al., in 2001. A descriptive study of the same population was conducted by Kolb, et al., in 1993.

In addition, hematological abnormalities were studied in ordnance workers in a case-control study by West and Stafford in 1997.

--o0o--

MS. DUNN: The animal carcinogenicity data available on TNT includes two-year bioassays of TNT in the diet of rats and mice conducted by the Army in 1984.

--o0o--

MS. DUNN: In addition, other relevant data on TNT include genotoxicity evidence summarized by IARC in 1996, of which there were many different types of positive studies. Examples are listed here and include frameshift mutation in Salmonella strain TA-98 and TA-100, mouse lymphoma gene mutation assay, Chinese hamster ovary cell mutation assay, and chromosomal aberrations in exposed workers carrying the NAT1 rapid acetylator genotype.

--o0o--

MS. DUNN: In addition, hemoglobin adducts in exposed workers has been studied in association with health effects not including cancer, and as biomarkers of exposure to TNT.

Reviews of the carcinogenicity evidence of TNT include a recent review by Bolt, et al., published in 2006.
as well as IARC's 1996 review.

IARC's review did not include the case-control study of leukemia by Kilian, et al., in 2001; the case-control study of hematological abnormalities conducted by West and Stafford in 1997; nor the two-year animal bioassays conducted by the Army in 1984, because the result of those studies were not published in the peer-reviewed literature.

--o0o--

MS. DUNN: That concludes my presentation of the evidence for 2,4,6-Trinitrotoluene.

CHAIRPERSON MACK: We're in a little bit of -- we have a little bit of difficulty today because the two people who were going to look at this chemical, as you did, in as much detail as they could -- and the information is fairly sparse -- are both sitting in Burbank airport.

So those of us who did not have it as a primary assignment have to fall in. And I now am eagerly awaiting somebody to take the initiative and tell us what they think.

And David looks really excited.

(Laughter.)

COMMITTEE MEMBER EASTMOND: Well, I did look over the data, so I'll give you my comment on it.
The first study, which was by Kolb, is essentially a descriptive study in which they had a high incidence of leukemia in an area and then they looked around and thought -- and they realize this area was contaminated with trinitrotoluene, TNT. And so it was kind of a cluster and TNT was suspected.

In the follow-up study, it was largely negative when they did the case control. So that's not too informative.

The other one in which they looked at the case control for hematological abnormalities by West and Stafford, they saw somewhat elevated risk, odds ratio of 1.8 I believe, but it was not statistically significant.

However, TNT is well known to cause hematological effects, including aplastic anemia in humans. And that's why they were focusing on the leukemia as the myeloid leukemias.

With respect to the two-year animal bioassays, what we saw were more or less excerpts of I assume much larger documents that we got the key portions of this. But in the rats there was a significant increase in bladder cancer seen in the female rats. This include both hyperplasia, which would be a preneoplastic type of change, increasing adenomas and carcinomas seen in the female rats.
And in the B6C3F1 mice, there was a dose related increase in splenic lymph -- leukemic and lymphomas is what it's called. And I assume these are T-celled leukemias and lymphomas. But some of the detail wasn't provided.

The mutagenicity data -- this compound is clearly mutagenic in a variety of different short-term tests. I will say though that the Sabbioni work from 2007 I found the chromosome abberation data pretty marginal at best. In fact, in one of the other papers they kind of indicated there was no increase seen in the TNT exposed workers.

But, anyway, there's a series of adducts formed. From my point of view, there is -- we have -- essentially in two different species we have significant dose-related increases in cancer in our rodent models. So that that would -- for me would argue that we should go forward and look into some more detail. I think the human data is much weaker. Certainly epi data is quite weak anyway.

CHAIRPERSON MACK: Thank you, David.

Anybody else?

Jim.

COMMITTEE MEMBER FELTON: We've looked -- being in a national lab, we've looked at a lot of these things that go boom --

(Laughter.)
COMMITTEE MEMBER FELTON: -- things I wasn't even
able to learn the name of. But they're all very similar
in structure. And to the compound, when you put these
nitro groups on these compounds, they're very mutagenic,
and some are a lot more mutagenic than this. So these are
really good mutagens.

So the question is -- you know, maybe comparing
the metabolism between the animals, metabolism among
individuals, they saw the paper that was on the
N-Acetyltransferase. You know, there may be individuals
that have various polymorphisms from that activation step
that would be more or less susceptible. So to me this is
a very interesting compound to look at. And I think
there's a lot of questions both about susceptibility in
humans versus one another and...

The real question though comes -- you know, we
talked earlier about: Are the people in California
exposed to large amounts of this? And there's the real
question. Is this really just a processing plant problem
or is this really an environmental problem? And I think
that's worth understanding too. So I'm really in favor of
going forward with this.

CHAIRPERSON MACK: Okay. Anybody else have
comments?

Darryl, Martin, and Anna?
All negative?

Okay. Well I actually did read it and I actually quasi-dismissed the epidemiology for much the same reason that David did, because this is a single cluster, and the cluster was inevitably going to contaminate anything that was not a subject -- or in the same population. So I think it just confirmed that there was a cluster and nothing more.

But I did get -- I did actually miss one of the species. But that bladder cancer caught my eye and that's what disturbed me. And the presumption -- and also the analogy to the other compounds are a very similar nature, which are in fact, as Jim said, very mutagenic. So I tend to fall on the side of pursuing this one as well.

Yes, Martin.

COMMITTEE MEMBER HOPP: Sorry I didn't mention earlier. When I looked through this, I was very impressed with of course the chemical data an animals. But what impressed me more was the lack of epidemiological findings, because this compound has been around for a long time with a lot of exposure and a lot of workers with a lot of intense exposure and in huge -- you know, it's very widespread and you would -- I would expect much more demonstrable epidemiological studies if this was a significant carcinogen.
So of all the things that disturbed me about this
relative to it not being carcinogenic in humans, although
laboratory data may show that, is the vast use of this and
the vast lack of any data proven in humans to show
carcinogenicity.

CHAIRPERSON MACK: I think that's a really good
point. The difficulty always though is what were the
difficulties in trying to study it? Were the people
exposed to it also exposed to lots of other things?
Usually if there isn't one cancer that stands out, you
wind up not getting a good study done because people don't
have the initiative of doing it.

But that's certainly a valid point.

COMMITTEE MEMBER HOPP: Yeah, it often disturbs
me -- going to come up later on also -- is that we may in
fact find chemicals that are carcinogenic in animals but
we have really no data that it's carcinogenic in humans.
And the question that this Committee always comes up to is
that -- what we're charged with is, is this chemical a
risk to humans? And this is an ongoing problem that we
keep having. But what disturbed me particularly was the
lack of epidemiological data because it's so widespread.

CHAIRPERSON MACK: We've had occasion in the
Committee to talk about this issue of humans versus
animals. And I come down on one side. And that if you
look at the way the initiative is worded, our job is to find chemicals that cause cancer, and not cancer in humans but cancer. So obviously the reason is to be concerned about cancer in humans. And that of course underlies our concerns about things. But the actual wording doesn't actually specify humans. It only specifies cancer. Now, fortunately insects don't get cancer. But at least mammals do. And some of the things that we've listed previously there wasn't good human evidence. But everybody on the Committee was very convinced that the animal evidence suggested that humans were in fact at risk.

So I don't think we should dismiss it because we don't think it causes cancer in humans. I think your first point though was a really good one, namely, that maybe we should have expected some good epidemiology in this particular compound.

Anybody else have any comments?

David.

COMMITTEE MEMBER EASTMOND: Yeah. Just the comment -- what's interesting to me is the two animal studies that we have access -- referring to that are kind of driving this are really not -- have not been published in the general literature. OEHHA's been able to get access to them. But when IARC reviewed the data, these
were not available to IARC. So in some respects by getting this out, the information that there are these animal data that show that this has been associated with cancer, may prompt people to do additional studies on TNT.

CHAIRPERSON MACK: Joan.

DIRECTOR DENTON: I wanted to have Carol just clarify just back to the point that you were talking about, Dr. Mack.

CHIEF COUNSEL MONAHAN-CUMMINGS: For Dr. Hopp -- and, Dr. Mack, you did a good job of describing the statute. But in terms of the findings that this Committee has to make when they do list a chemical, which you're not doing today, you're actually finding whether or not the chemical has been clearly shown through scientifically valid testing according to generally accepted principles to cause cancer. And so it doesn't say to cause human cancer. And in fact there was some early litigation in the Prop 65 area where it was determined by the courts that it was appropriate and indeed required that chemicals be listed based on animal evidence alone, unless there was some indication that a human would not react the same way, you know, mechanistically or whatever, to that chemical. So I understand what you're saying about, you know, human cancer. But this Committee, as Dr. Mack mentioned, is really looking at: Does the chemical cause
cancer? It doesn't have to be shown to cause cancer in humans.

Does that help?

CHAIRPERSON MACK: Jim.

COMMITTEE MEMBER FELTON: One other thing I think we need with respect to your comments is -- there's a big difference between negative epidemiology studies and no studies. And, you know, we have to consider that all the time. There may be really no good reason why this population hasn't been studied except that it was never anybody's priority to do so. It doesn't mean that there aren't cancers there, but nobody's studied it. So it's a problem for us when we do the evaluations. But there's a big difference between negative and not done at all.

CHAIRPERSON MACK: Okay. Are we ready to -- oh, Darryl.

COMMITTEE MEMBER HUNTER: No, I just had a question. You'd made reference that there were a couple animal studies that weren't published but that we were able to get access to. I was curious --

CHAIRPERSON MACK: That's what David said. And the fact is that if we were to vote that this should be looked at in detail, they're not limited by the rules that apply to IARC. They can get whatever evidence is available whether or not it's been published. So you can
be sure that the most available information will be looked at if we vote yes.

COMMITTEE MEMBER HUNTER: I was just curious as to why they weren't published. Was it just opinions --

COMMITTEE MEMBER EASTMOND: Essentially they're paid for by the Army to do a study for the Army. These were contract labs. They did their study. They provided the report. But Army had no reason to publish it. So these are -- you know, certainly the technical documents are available. We have excerpts of them in our handouts. But they just didn't have any motivation to publish it.

So it's not been published. I think that's a fair --

CHAIRPERSON MACK: The Army does not have the residents of California as their primary concern.

(Laughter.)

CHAIRPERSON MACK: Are we ready to vote?

CHIEF COUNSEL MONAHAN-CUMMINGS: No, You need to have public comment first.

CHAIRPERSON MACK: Oh, public comment.

All right. Is there an overwhelming surge of public people who want to comment?

This is really a record.

All right. Here we go again.

I'm not going to read -- do I have to read the paragraph again?
DIRECTOR DENTON: No.

CHAIRPERSON MACK: No, I don't have to read the paragraph again.

Do you advise OEHHA to begin preparation of the hazard identification materials for 2,4,6-trinitrotoluene?

All those advising "yes," please raise their hands.

(Hands raised.)

And that covers the waterfront. Nobody is left to vote "no". So they had 6 to 0.

All right. And now we turn to Dr. Tomar.

(Thereupon an overhead presentation was Presented as follows.)

DR. TOMAR: Marijuana, the botanical name for the plant is cannabis sativa and the candidate chemical is marijuana smoke.

DR. TOMAR: It's a commonly used drug by young adults. The exposure is through inhalation of burning flowers, leaves, stem or resin of marijuana plant. It is used for recreational as well as medicinal purposes. And the combustion products contain carcinogenic and procarcinogenic compounds.

DR. TOMAR: The available evidence identified in the prioritization process for consideration include
epidemiological data identified during human data screen, animal carcinogenicity data. And other relevant data include on genotoxicity, carcinogenic constituents of marijuana smoke, immunosuppression, and endocrine effects.

DR. TOMAR: The epidemiological literature includes studies on cancer related to individuals with exposure to both marijuana and tobacco smoke. This suggests that some individuals who use marijuana may also use tobacco.

This is a compilation of a study that appears to have a reasonably well-defined measure of exposure to marijuana smoke alone. Both positive and negative studies are included here.

We have a series of case-control studies with the tumor of head and neck cancer, a study of lung and upper aerodigestive tract cancer, a study of the oral squamous cell carcinoma, another case control study of transitional cell carcinoma of the bladder. We have one large retrospective cohort study dealing with all sites cancers. And we also have case series study dealing with the respiratory tract cancer as well as couple of case reports which deals with the transitional cell carcinoma.
gestation is associated with acute nonlymphoblastic leukemia as well as childhood rhabdosarcoma.

DR. TOMAR: The animal carcinogenicity studies include marijuana smoke condensate with a classical skin painting study as well as initiation and promotional study by Hoffman, et al., 1975.

We have another study in rat with the subcutaneous administration by Repetto, et al., in 1979.

There's one study by inhalation on marijuana smoke by Murthy, et al., 1985.

DR. TOMAR: The other relevant data include the genotoxicity, which is -- we have a positive study on somatic cell mutation in mothers and their newborns.

We also have another important study on DNA adducts in the lungs of exposed monkeys. But unfortunately the study was conducted seven months after the exposure I just talked.

DR. TOMAR: Marijuana smoke contains many of the same carcinogen and procarcinogen found in tobacco smoke, such as acetaldehyde, benz[a]anthracene, benzo[a]pyrene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, dibenzo[a,e]pyrene.
DR. TOMAR: We have other -- quite a bit of data on respiratory and immunological effects studies; as well as endocrine-like effects, one single report; and one report on molecular mechanism, mostly it is for cannabionoids rather than tobacco smoke alone.

We have a series of reports recently on epidemiological data which have been reviewed in a number of studies.

DR. TOMAR: We have only one comment from Dr. Gieringer, Director of the National Organization for the reform of Marijuana Laws.

This concludes my overview of studies compiled in the prioritization process. And I leave it to the Committee now for their further discussion.

Thank you.

CHAIRPERSON MACK: I only have one correction, Dr. Tomar. This is a material which is used by people who used to be young adults.

(Laughter.)

COMMITTEE MEMBER WU: Well, in reviewing the epidemiologic studies, I -- there were really 16 studies that I examined. Twelve of them were in adults and four of them were in children.
In the adult studies, as has previously been covered, it covered a range of cancer sites including lung, head and neck lymphomas; anal and penile cancers; as well as bladder cancers. And even though there were a few early studies in adult studies -- in adult cancer, suggesting an increase risk, more recent studies that were larger that were able to look at both dose response as well as duration response relationships did not find a positive association.

And I think probably the largest study was one that was published last year by Hashibe. And that particular study included both -- included really five sites.

So I would say that adult studies covering a range of exposure -- a range of cancer sites, and some of them were able to adjust for potential confounders, did not find an association.

Now, there were two small studies of -- two earlier studies of lung cancer that suggested an increased risk with exposure. But both of those studies were unable to really tease out the effect of tobacco use. So I think those two studies, the results are very difficult to interpret.

There were four studies looking at various childhood cancers. All four of those studies in fact
found an increased risk associated with maternal use of marijuana during gestation. In at least two of those studies they also had information on father's use of marijuana, and they both also showed an increased risk, although it was a little bit lower than what they found in terms of mother's exposure during the pregnancy.

There are a variety of issues that one can go into in terms of maybe explaining the inconsistent findings in the various epidemiology studies, which I can go into if we want to. But maybe I should stop and see if there are other comments and then we can continue the discussion.

CHAIRPERSON MACK: Martin.

COMMITTEE MEMBER HOPP: I also looked at this fairly carefully. And I appreciate your quick review here. It saves me a lot of time.

I do want to bring up a couple things that I think are confounding and make this issue a little bit smoky.

(Laughter.)

COMMITTEE MEMBER HOPP: The issue we have in front of us is marijuana smoke, not cannabis. And I think that the confusing of the two is a problem in the literature relative to its effect on humans as well as the discussion here. Because cigarette smoke -- excuse me --
marijuana smoke contains a huge amount of chemicals besides THC and cannabis. And the discussion here we really have to focus on is the effect on marijuana smoke. And when you really eliminate the argument of whether or not THC or cannabis itself has its carcinogenic effect, the same issue has to be made for the other chemicals that are within marijuana smoke. So you may eliminate THC or add it. But we're still looking at the entire process of marijuana smoke and whether or not that is a risk to a carcinogenesis.

If you look at the individual chemicals and things contained within cigarette -- excuse me -- within marijuana smoke, you find a whole list of chemicals that are clearly unambiguously carcinogenic: Benzanthracene, benzopyrene, benzofluoranthene. These are things that are clearly carcinogenic, long established to be carcinogenic in animals and humans, and have been on our bad, bad list for a long time.

There's also additional data to show in these multitude of studies that the aqueous portion of cigarettes -- excuse me -- the aqueous portion of marijuana smoke, the tar portion, is the most carcinogenic. Unfortunately the aqueous portion of marijuana smoke is probably the portion that is absorbed most in humans.
So I think the first point I want to make is that we really have to separate out THC from the carcinogens and the carcinogenicity of marijuana smoke.

The second thing I wanted to bring out was the epidemiological data -- and I think -- well, Dr. Wu had mentioned Hashibe's study, which I think is spectacular. I think that we also have to recognize that our chairman here, Thomas Mack, was part of that study and is undoubtedly -- I'd like to hear his comments relative to the data that was absorbed there.

As an epidemiologic study, it's probably more classically -- the highest level type of studies we have because they actually interviewed the patients and the controls at a very rigorous manner. The other studies that are shown epidemiologically are focus studies with what I would consider poor case controls and small numbers.

And, lastly, what I wanted to bring up was the immunomodulation effect of THC in the confounding biologic data that we see in these studies. There's -- as we talked about is some of the other discussions, when you have a whole multitude of chemicals present in the exposure to human, you could have carcinogenic and non -- as well as protective and procarcinogenic activities all at the same time. And identifying individual activities...
of each one of these is particularly difficult in
marijuana smoke, because of the lack of control of the
substance, its quality, quantity and volume.

So that while individually we may see multiple
chemicals, we have to look at the seriousness of these
chemicals I think and understand them as they relate to
the whole.

End of comment.

Would you like to comment a little bit more about
your --

CHAIRPERSON MACK: Actually I don't think it's
appropriate. I played a relatively small part in that
study. The study was published, and I think everybody has
seen it.

Any epidemiologic study -- no epidemiologic study
is perfect. This was a relatively large study. The
people who ran it tried really hard to make the controls
comparable to the cases. So for what it's worth, they
were unable to show any association.

But, you know, whenever we look at human studies
or animal studies, for that matter, there's always a limit
in representativeness of all exposures and all studies.
And particularly in this case when you have a lot of known
carcinogens, as you rightly point out, in the smoke, one
presumes, if one has any sense I think, that it's a matter
of dose. If people got enough dose to marijuana smoke, it's likely that it probably would produce the same cancer that the same chemicals do when they're given in the same dose in other formats. But that's not our job to speculate about.

So I think that the likelihood is that this probably could cause cancer under some circumstances because those chemicals are known to. But I can't rule out the possibility that there's something in marijuana smoke that counteracts all of those carcinogens and provides a safety net. So we just look at the empiric evidence as it stands.

So, anyway, now I'll shut up.

Jim.

COMMITTEE MEMBER FELTON: Just a question for Dr. Tomar.

The list of chemicals that we have in front of us are -- where did they come from? Because these are all PAH type compounds. Yet if you look in cigarette smoke, I mean there's people out there that say that PAHs aren't even important, it's really the nicotine derivatives, NNK, it's amino-a-carboline, there's aminobiphenyl. I mean there's just so many good carcinogens in cigarette smoke, I would expect some of those to be in marijuana but they aren't listed here.
DR. TOMAR: I didn't get the question. Sorry.

COMMITTEE MEMBER FELTON: So this list -- you have seven compounds on your list that you gave us.

DR. TOMAR: Yes.

COMMITTEE MEMBER FELTON: And they're all basically PAH-type compounds.

My question is -- in cigarette smoke there are many other compounds that we know are carcinogenic. Have those been looked at in marijuana or is this just what somebody looked for?

DR. TOMAR: No, marijuana constituents are compared with the tobacco smoke. And all these are well-known identified carcinogens. So I did want you to put something which I was ambiguous about or which I cannot prove that it is a carcinogen by one way or another.

But, yes -- and as far as individual chemical is concerned, I'm more concerned about, as the comments made, the total smoke is the candidate chemical and not the unusual chemical, because we know that some of the cannabanoids will very highly immuno suppress you, especially for -- immunity.

So I didn't want you to bring those things to the Committee.
SANDY: Dr. Felton, just to follow up.

We did not do a thorough review of all the papers that have measured the constituents in marijuana smoke. We had a couple papers that were fairly old. And we just were able to note that the several that we've put in that table that we've presented that are both in marijuana smoke and they happen to be in tobacco smoke and they're known carcinogens. But this is not meant to be a thorough review of everything that's carcinogenic in marijuana smoke.

COMMITTEE MEMBER FELTON: That's what I wanted to ask.

CHAIRPERSON MACK: David.

COMMITTEE MEMBER EASTMOND: Yeah. And this is more of a question for some of the epidemiologists. But as I look through this in kind of a cursory fashion, there were a moderate number of studies that I thought had intermediate-level relative risk for odds ratio, in the range of 3.1, 3.4, et cetera. And even the Hashibe study, all the crude odds -- many of the crude odds ratios were significantly increased. And it wasn't till they did three different models of adjusting -- and even under model 1 I think all three of those continued to be significantly increased. It wasn't till they got to adding more and more variables in, and then they lost --
the significance disappeared.

And the fact that they presented all three models means there was a reason for it. And I tried to -- like
to get some insights into what you think when you see this
sort of situation where you have a crude odds ratio of
significant, use one model, there's still significant, use
another model. Two of the three are no longer
significant. And third model, all three drop out.

How would you interpret that sort of data?

COMMITTEE MEMBER WU: Well, I'll take a stab at
it first.

I think one of the concerns with the previous
studies and I think the reason why they probably presented
the data in that fashion was that it was always suggested
that because marijuana and tobacco smoke as well as
alcohol use are very important and -- very strong
potential confounders for all of those sites that were
examined, that it is really important to be able to look
at the association, carefully adjust it really for the
other exposures. Now, of course if you can get a clean
group, which is never smokers -- never drinkers who are
using marijuana, that's the best, right?

(Laughter.)

COMMITTEE MEMBER WU: But of course those numbers
are tiny in the studies. So I think they did their best
to really try to at least demonstrate what type of
information was there, and that in fact it is extremely
difficult in these situations to really rule out the
potential confounders. And I think it's really a balance
of looking at, first of all, what is the nonexposed group,
what -- you know, and I think one thing they didn't do is,
you know, if you actually look at how -- when they
carefully adjust it for tobacco smoke, you know, what kind
of reduction in risk.

And I think if they have -- so I think you have
to sort of decide, you know, whether in fact this is
really -- unless you actually have a clean group, you
know, is there a marijuana smoke effect? Or is it really
confounded by active smoking and alcohol use, which have
all very, very strong established risk factors for these
other cancers that they're looking at?

CHAIRPERSON MACK: I agree completely.

I'd like to just add one thing. And, that is,
when you do these adjustments with additional models which
enter additional confounders, you never can be sure that
you're not eliminating a true association. But you wind
up having to conclude, like we do in many instances,
saying we don't have any evidence that there is a true
association. Doesn't mean it isn't there. It means that
this particular study doesn't really find evidence that
it's there, partly because it's masked by the other exposures which we know are important.

So I think you're getting some concern from the fact that the crude association was strong. But you're left still not being able to say that this is the marijuana smoke.

COMMITTEE MEMBER EASTMOND: Well, that was the one example I used. But, you know, you've got -- and for those where you think alcohol might contribute. But I mean evidence like the carcinoma of the bladder like Chacko, et al., had odds ratio, now that's a tobacco-related target site. So -- you know, maybe it's tobacco related. And usually, I would imagine, there's a lot of confounding between the two. But, you know, you go through this and I wasn't sure what to think.

The biggest issue for me was is there was so much inconsistency across these studies. You would think that if it was positive at least in one type of tumor, you'd see a similar thing in others. And there's just no consistency from what I can tell.

CHAIRPERSON MACK: I don't know what Anna would say, but I guess I would begin by saying if you expect epidemiologic studies to be consistent --

(Laughter.)

CHAIRPERSON MACK: -- you're in the wrong
ballpark.

(Laughter.)

CHAIRPERSON MACK: Because all kinds of people --
and especially when you're talking about an exposure that
is very difficult to measure. If I ask you how much
marijuana you smoked when you were a given age -- I'm not
going to --

(Laughter.)

CHAIRPERSON MACK: -- you may not be able to
remember. In fact, you may not have been able to remember
ten minutes later.

(Laughter.)

CHAIRPERSON MACK: I think you have to be
concerned about the quality of the exposure information
always, and especially if there's a potential problem in
terms of the implications for society. So I'm not so
surprised that they're inconsistent.

I think the studies that I was most interested
in -- and I don't know how to evaluate them, and let me
ask Anna maybe to elaborate a little bit -- are the
children's studies. The Rhabdomyosarcoma is done by a
good epidemiologist. And, in fact, both of them were done
by good epidemiologists, but -- and they were strongly
positive. But you still are left not feeling very
comfortable about it.
COMMITTEE MEMBER WU: Yes, I have to say I -- I thought that the children studies were of concern, partly because they were really careful in trying to assess misclassification, especially because in the one study where there was a -- the risk was 11 -- the odds ratio was 11, they only had very low -- low prevalence of exposure among the control group. And so they actually tried to check the other children's cancer study that they had access to to try to see whether in fact this was really due solely to underreporting among the control's mother or overreporting among the case mother.

And I think they concluded at the end of all that that it really was not simply because of both overreporting among case mother and underreporting among control mother, but that this effect is real, it may not be 11-fold increase risk. And certainly the other children's study also suggests that.

And so I'm left with thinking that it's not something that we could ignore, and probably should try to see, you know, what other studies are out there in children. And I'm certain that there are -- I mean I don't know this literature, but whether there are actually studies -- because all of these studies except for the few studies done in Africa were all done in western U.S. populations. And I think the exposure to marijuana is

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act actually quite substantial in other western populations. And try to see, you know, whether there are studies out there.

Certainly there is a large European childhood cancer consortium. I don't know whether those studies ask the question. So I think it's worthy of exploring these large children studies that I know are out there.

CHAIRPERSON MACK: Okay. Are there any other comments from -- yeah, Jim.

COMMITTEE MEMBER FELTON: Well, I know it talked about the epidemiology a lot here. But and then coming back to the comment on the last chemical. I mean when you have such mixed complex exposures, you've got to go to the rodent and then see what you get there, because you've got to put the two together, because you can't do it all with the human studies.

CHAIRPERSON MACK: David.

COMMITTEE MEMBER EASTMOND: My concern was is the animal studies were really poor and very few of them.

So --

COMMITTEE MEMBER FELTON: Then you might end up --

COMMITTEE MEMBER EASTMOND: Yeah, I mean it was just -- it was an idea of saying -- usually you look at the animal studies to help give some clarity when you have
this sort of mixed exposure, a variable sort of outcome.
And the animal studies in this case are really quite poor.
At least that's my interpretation.

CHAIRPERSON MACK: Any more comments from the
members of the Board?

And now --

COMMITTEE MEMBER HOPP: Yeah. Again, I'd like to
respond to that a little bit, because this is again my
concern. Again, I'm not -- I don't know every article
about marijuana smoke and its -- in the studies in animal
because I think that there's a general tendency for
skipping animal studies in things like marijuana smoke due
to the prevalence in the vast data available for regular
cigarette smoke containing the same carcinogens. So
there's no doubt that it's not necessarily the same, but I
think that there's also a lack of general -- I would
suspect there's a lack of a huge industry looking at this
in the animal.

CHAIRPERSON MACK: Okay. Any other comments?

And now third time is the charm.

Is there any comments from members of the public.

DR. GIERINGER: Sure. I'm Dale Gieringer with
California NORML, a national organization for reforming
marijuana laws, who submitted testimony.

But there were just a couple of points that I
wanted to highlight that I didn't hear mentioned in the
discussion. One was the -- I didn't hear anyone mention
the relatively new data -- studies that have come out
showing an anticarcinogenic effect from THC and
cannabinoids in particular, which are the peculiar
ingredients in marijuana smoke. In particular, just this
year, the study by Preet, showing
Delta-9-tetrahydrocannabinol inhibits epithelial growth
factor-induced lung cancer cell migration in vitro as well
as growth and metastasis in vivo. And there are a couple
more recent studies showing protective effects from the
cannabinoids in particular.

But getting along to the larger question about
the smoke, I hope everybody here is aware of the very poor
quality of the data we have about the consistency of
marijuana smoke. Most of those studies are old studies,
and they all use a particular -- a particular source of
marijuana. In fact, it's the government itself, the
famous marijuana farm in Mississippi, that is actually the
only legal source of marijuana. And except for your
epidemiological studies, any study that's done on animals
or in labs uses this marijuana. And this particular
marijuana is sort of egregious for its low quality, its
low cannabinoid content. It's very leafy. It's more like
tobacco, I would say, than anything else. There are -- it
certainly does not meet the current standard for the kind of cannabis that's used most frequently, especially in medical practice, where the bud is -- the bud of the plant is smoked rather than the leaf. The government's substance consists of leaf, which has usually been freeze-dried actually before the study, is my understanding.

It has been impossible to do any studies of sinsemilla or the many, many different varieties of cannabis that are currently out there and being smoked, because the government just doesn't let people do studies on that. And you can -- I mean I've had trouble with that myself, because we've been trying to do studies on marijuana smoke vaporization and they won't let us get any realistic marijuana for the studies.

So I hope if you do take a further look at this, you will somehow try to find some information about the constituents of the actual smoke that is out there rather than the smoke that comes from burning NIDA's Mississippi ditch weed, as it's called, by aficionados, of which there aren't any.

(Laughter.)

CHAIRPERSON MACK: Thank you very much. Actually that was a really good point. And I wonder if -- I suspect the OEHHA staff would greatly appreciate it if you
could find documentation of that fact basically of the
Mississippi source of most of the animal studies.

DR. GIERINGER: Certainly.

CHAIRPERSON MACK: Are there any other comments
from the public?

COMMITTEE MEMBER HOPP: I have a comment.

CHAIRPERSON MACK: Martin is a public person too.

(Laughter.)

COMMITTEE MEMBER HOPP: I'd like to just review a
position paper by the Thoracic Society of Australia and
New Zealand that's in our pile here and their conclusions
about relationships of this. And they talk about the
histologic effects -- histopathologic effects of cannabis
smoking in humans, including changes consistent with acute
and chronic bronchitis. Cellular dysplasia has been
observed, suggesting that, like cigarette smoke, cannabis
exposure has the potential to cause malignancy. These
features are consistent with clinical presentation of
symptoms of cough, early morning sputum production in
young individuals who smoke cannabis alone.

It states here that "almost all studies indicate
the effects of cannabis and tobacco smoking are addictive
and independent. Public health education should dispel
the myth that cannabis smoking is relatively safe by
highlighting that the adverse respiratory effects of
smoking cannabis are similar to those of smoking tobacco, even though it remains to be confirmed that smoking cannabis alone leads to the development of chronic lung disease," which is exactly the point that I think we were discussing.

CHAIRPERSON MACK: Thank you, Martin.

So now I guess we're ready to take the vote again.

COMMITTEE MEMBER FELTON: I just have one further comment.

CHAIRPERSON MACK: Oh, it's always going to be one more thing.

Go ahead, Jim.

COMMITTEE MEMBER FELTON: It seems to me that -- I think our public comment brings up a real problem with the studies here, because when you're studying tobacco smoke, you can go get a commercially produced Camel or Marlboro, or whatever you've got, or some government manufactured standard cigarette. But here -- I mean the source of this particular product that we're going to study is so varied, that I'm not sure how we're going to make any conclusion except maybe based on what the government farm has. But whoever's growing this in Sacramento in their backyard, I can't -- when we get the data, we can't say whether that falls into the general
category or not.

So it just worries me that we're dealing with a very inconsistent source of the chemicals rather than what you get out of a commercial product.

CHAIRPERSON MACK: You suggest that every effort ought to be made to find European studies, which presumably are not limited by the American Government's decision -- and the Australian and New Zealand studies and places where there might be availability of other forms. Anyway, you're just reinforcing the point that the public member made, and that was a very good point.

David.

COMMITTEE MEMBER EASTMOND: One item. The long-term -- the study by Murthy, et al., that is the inhalation study in rats is actually out of Jamaica. So I'm not sure the U.S. Government, you know, source is really relevant in that case.

CHAIRPERSON MACK: So we have one ganja study.

(Laughter.)

CHAIRPERSON MACK: All right. Are we ready for a vote?

Do you advise OEHHA to begin preparation -- oh, I'm sorry.

Do you advise OEHHA to begin preparation of the hazard identification materials for marijuana smoke?
All of those advising "yes," please raise their hand.

(Hands raised.)

CHAIRPERSON MACK: 1, 2, 3, 4.

All those advising "no," please raise their hand.

(Hands raised.)

CHAIRPERSON MACK: Two.

4 to 2.

All right. That's the vote. And I think that concludes our official -- well, there's an open item which says that if anybody else has a chemical that they wish to propose for Committee consideration -- anybody on the Committee has a chemical that they wish to propose for consideration, then a separate polling should be taken.

Does anybody else have -- does anybody have such an additional chemical?

And I guess the answer to that is no.

Now, having finished the formal business, I wish to say one thing. And you may say something also.

CHIEF COUNSEL MONAHAN-CUMMINGS: Dr. Mack, you might ask the public here whether they have any chemicals they'd like the Committee to consider.

CHAIRPERSON MACK: Oh, but I don't think they have that option, do they?

CHIEF COUNSEL MONAHAN-CUMMINGS: Yeah, they do.
CHAIRPERSON MACK: They do?
All right. Public, please present your chemicals?
Thank you for keeping me honest.
Now, I will continue.
Dr. Felton is leaving the Committee as of today.
And that is a great loss. As you can see, he pipes up quite frequently. And his expertise has been extremely valuable. In fact, I don't know what I would have done in the last -- how many years has it been?
COMMITTEE MEMBER FELTON: More than ten.
CHAIRPERSON MACK: More than ten.
I don't know what I would have done without him. And I'm going to miss him terribly. I'm thinking seriously of going with him if he really is serious about it.
So thank you, Jim.
COMMITTEE MEMBER FELTON: You're welcome.
DIRECTOR DENTON: Dr. Felton resigned -- was it last month? -- several weeks ago. And the reasons that he gave was that he has retired from Lawrence Livermore and also that he's doing more with his duties with UC Davis.
So from OEHHA -- we'd like to say how much we appreciate the service that you've given on the Committee.
And I do have a couple of resolutions that I
would like to give to you.

Is there anything that you would like to say officially before we do that?

COMMITTEE MEMBER FELTON: Just if I could say a few things. You know, being on a committee for all these years -- and the staff that we had to work with here in OEHHA is just amazing. I mean they come up with the information. We look it over. But our job -- if we had to start from scratch, it would be almost impossible. So these guys really make the job being on this Committee quite easy.

The other thing is that from a bench scientist -- I'm not a physician or an epidemiologist -- to participation in something like this where the public is actually interested in the science and the decisions is really a real treat. And it's so different than the rest of my life. And I remember hearing some of those same comments from other bench scientists that have been on this Committee. This is just a different way of looking at science and information. And so it's been a great experience for me.

Thank you.

Oh, my gosh.

DIRECTOR DENTON: We're prepared.

So this is a commemoration from -- actually
signed by the Governor. So I'd like to read it to you,
Dr. Felton.

"November 9th. Dr. James Felton.

"Allow me to convey my

congratulations to you as you retire

from the Science Advisory Board's

Carcinogen Identification Committee.

"I deeply appreciate your hard work
to protect the health of all
California. Among many
accomplishments, your insightful
evaluation of chemicals to determine if
they cause cancer will undoubtedly save
lives. I applaud your tremendous
contributions to the Committee and your
remarkable career that has seen you
serve in many pivotal positions.

"It was a pleasure to appoint you to
the Science Advisory Board. And you can
take pride in the many hours you devoted
to improving public health in the
environment. Whether as a board member,
professor, or researcher, you have done
much to enhance our state, and your
expertise will be missed.
"On behalf of all Californians,
please accept my gratitude and best
wishes for every future success.

"Sincerely, Arnold Schwarzenegger."

(Applause.)

COMMITTEE MEMBER FELTON: Pretty nice.

DIRECTOR DENTON: I do have one more. And it's quite long so I won't read it all. But it's a recognition of service to Dr. Felton. And it talks about Dr. Felton having served on the Science Advisory Board, inaugural Carcinogen Identification Committee, three gubernatorial administrations, almost 15 years of public service, participated in the evaluation of complex data, provided insightful guidance in decisions made by the CIC. The CIC has benefited the people of California by helping to ensure they receive clear and reasonable notice of exposure. Consistently provided keen scientific analyses of genetic toxicity in the area of cancer identification. And an invaluable advisor to the state.

He's a world-renowned researcher in the role of dietary heterocyclic amines formed during cooking. And during his professional career has shared his expertise in cancer causation and prevention as they relate to food.

He served as the deputy, associate director, and division leader the Biology and Biotechnology Program of
Lawrence Livermore National Laboratory, adjunct professor at UC Davis.

Recognized for his expertise. And among his accomplishments has served on the National Cancer Institute's Board of Scientific Counselors Division of Cancer Etiology, past President of the U.S. Environmental Mutagen Society, and is a member of the Steering Committee of the American Association for Cancer Research.

"Therefore, I, Linda S. Adams, the Agency Secretary for Environmental Protection, do hereby recognize Dr. Felton for his years of outstanding public service to the people of California."

And that signed by Linda S. Adams, the Agency Secretary.

(Appause.)

DIRECTOR DENTON: So now we want them prominently displayed in your new office.

CHAIRPERSON MACK: Well, that's the fun part of the meeting. Now we have to turn back to work.

And so, Martha.

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: Thank you.

(Thereupon an overhead presentation was Presented as follows.)

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF
SANDY: Okay. So the next item is discussion of our next prioritization data screen that we'll be performing. We have to come up with some other way of sifting through that varied mix of chemicals with varied types of data available to choose a group of chemicals and then look at them a little more carefully.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: So as described in the prioritization document that we referred to, we will reapply the epidemiology data screen, and we may get a few chemicals. A few years have passed. There may be more studies. But then we're going to need to apply one or more animal data screens. And then we would conduct a preliminary toxicological evaluation of those chemicals we identified through the use of those screens.

And then we would bring that to your Committee as chemicals proposed for Committee consideration.

So really the question here is: What should those data screens look like, those animal data screens?

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: So we're looking for input from you all. And we have a few ideas that I'd like to take a little time to go through.
SANDY: In thinking about how to structure these screens, there's many different approaches. We did consider in the back of our minds your Committee's criteria for identifying chemicals for listing as known to the state to cause cancer.

We also considered the Preamble to the International Agency for Research on Cancer Monographs on the Evaluation of Carcinogenic Risks to Humans. At your last meeting last year we had Dr. Vincent Cogliano from IARC come and give a presentation. And part of his presentation was discussion of how the preamble had been revised, recognizing that there are fewer and fewer animal cancer bioassays that are published now and we expect fewer to be published and more use of mechanistic data.

So keeping all of that in mind, thinking of what type of a screen we would apply now to reach into that mixed bag of chemicals and choose which ones to look at more carefully.

SANDY: One screen would be perhaps to look for chemicals with two or more positive animal cancer bioassays or one positive bioassay with either malignant
tumors occurring to an unusual degree with regard to incidence, site or type of tumor or age of onset; could be combined malignant and benign tumors; or a positive -- one positive bioassay with findings of tumors at multiple sites. So it's one possibility.

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CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF

SANDY: A second screen might be looking at chemicals with one positive animal cancer bioassay; and then in addition to that, structure activity comparisons with a known carcinogen or evidence from a second animal cancer study of benign tumors known to progress to malignancy; or evidence that the chemical operates by a mechanism known to be involved in human carcinogenesis such as genotoxicity or altered gene expression or immune suppression or hormone disruption.

So those are two possible screens to start your discussion.

Thank you.

CHAIRPERSON MACK: All right. I'm going to start with David.

Are you alert and awake? And you watch those two screens.

COMMITTEE MEMBER EASTMOND: Yeah, I mean I -- my impression is that you have to go forward using some sort
of systematic approach. And I think you've taken a
reasonable approach to go forward. So I don't -- I mean I
would probably support the approach you're taking.
I don't have a lot of other comments.

CHAIRPERSON MACK: Do you have a preference as to
the two screens?

COMMITTEE MEMBER EASTMOND: Let me go look at
them again. Just a second.

COMMITTEE MEMBER HOPP: Martha, can I ask you,
when you put those two together, what do you see as the
significant difference between the two in how chemicals
are going to come up?

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF
SANDY: Well, the way we structured this, there's
sequential screens. The first screen would be one cut
where you get two or more positive studies, or one with
the particular concern. And then if we didn't get enough
chemicals using that screen, we would go to the second
screen, which goes down a level lower. But, as I said,
there's many ways to cut this, and we're just looking --
here's our proposal and looking for comments and ideas
from you.

COMMITTEE MEMBER EASTMOND: I mean that's the way
I kind of interpreted it. I didn't see it as one versus
the other. It was just here's the first cut, then the
second, which seems to make sense from my point of view just going forward that way. I mean I think one of the issues is going to be -- and I don't know how. It has to do with some of these newer types of animal bioassays, the short-term cancer bioassays, and how we interpret those, either in the -- such as the P53 heterozygotes or even the newborn mouse assay, in looking at some of those. And I don't know how -- I think the Committee's going to have to try and figure out how we interpret that data. Because, As you said, the number of studies that are being done in sort of conventional two-year chronic rodent bioassays is dropping quite dramatically, but there are increases in other types of tests. And so this is something I think we'll have to be discussing as a group later.

CHAIRPERSON MACK: Jim.

COMMITTEE MEMBER FELTON: Well, I agree with David. I think if I was designing this myself, I probably wouldn't do anything different than what you guys have already done. I think this is well thought out and probably the right way to do it. Sometimes though some of these other criteria that you haven't screened too can be quite important. So maybe my only difference would be maybe not so much screen 1 and screen 2 having such big differences. But if you
see something that's really dramatic as far as the mechanism or the structure activity goes, that it would be a call-out as well. But I don't think I'd design it any different. Very good.

CHAIRPERSON MACK: Martin?
Anybody else?

I guess the question that Jim is asking is, couldn't you put the two of them together in a single screen? Or maybe having them run consecutively is the same thing as that, but maybe it is more work.

But I agree. I think that -- and I think the only question was which -- is it clear that screen 1 is going to be picking up lower hanging fruit than screen 2. And I'm not sure it's true anymore.

So to try and put them together might be a good idea. But if you can't put them together, then putting them in consecutively is giving you the same thing, so I don't think anybody is going to disagree with that.

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF SANDY: Okay.

CHAIRPERSON MACK: Does anybody else --

COMMITTEE MEMBER HOPP: Well, the difference really is in the one positive animal cancer assay. You know, if you have two positives, then it goes through. But if you have only one positive, what else in addition
to that is significant relative to bringing it to the top?
And basically there's five different ways of additional
assays besides one animal assay.

CHAIRPERSON MACK: Anybody else?
COMMITTEE MEMBER HOPP: Seems good. I mean I
think all these things are pertinent to cancer production
and I think those are the right things to look at.
CHAIRPERSON MACK: Okay. Does anybody in the
public have any comments on this issue?
I guess not.
So I think that's as much feedback as you're
going to be able to get from us.

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF
SANDY: Well, thank you.
CHAIRPERSON MACK: You're way ahead of us.
So now we come to the next to final item, which
is staff updates.

Sorry. Come to the next to the last item, which
is staff updates.
Martha.

CANCER TOXICOLOGY & EPIDEMIOLOGY SECTION CHIEF
SANDY: And I think that's Cynthia Oshita.
CHAIRPERSON MACK: She's going to do it. Okay.
MS. OSHITA: Good morning.
OEHHA has administratively added three chemicals
to the Prop 65 list. Two chemicals as known to cause cancer. They were iprovalicarb and anthraquinone. And one chemical as known to cause reproductive toxicity. And that was di-isodecyl phthalate.

In addition to these three chemicals, isosafrole, 5-nitro-ortho-anisidine, tris(aziridinyl)-para-benzoquinone were removed from the Proposition 65 list. These chemicals were added to the list in October 1989 by operation of law based on the Labor Code Sections 6382(b)(1) and (d) that incorporates by reference chemicals that require the inclusion of substances listed as human or animal carcinogens by the International Agency for Research on Cancer and requires the inclusion of chemicals within the scope of the federal Hazard Communication Standard 29 CFR 1910.1200 which establishes that a chemical is a carcinogen or potential carcinogen for hazard communication purposes if it's identified as such by IARC or the National Toxicology Program.

The classification of isosafrole and tris(aziridinyl)-para-benzoquinone as Group 3 by IARC and the removal of 5-nitro-ortho-anisidine from designation as such by NTP required that these chemicals be removed from the Proposition 65 list.

A summary sheet of these latest changes to the

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Prop 65 list along with their effective dates are provided in your binders behind the "Staff Updates" tab. In addition to these listings and delistings, there are several chemicals under consideration for administrative listing now. They include gallium arsenide, as a chemical known to the state to cause cancer, and hexafluoroacetone, nitrous oxide, vinyl cyclohexene dioxide, and methanol as chemicals known to the state to cause reproductive toxicity. Comments were received on these chemicals and they are under review.

Also included in your binders is a summary sheet of the safe harbor levels that were adopted during the last year. There were three Maximum Allowable Dose Levels (MADLs) that were adopted effective September 30th, 2007. They are ethylene glycol monoethyl ether, ethylene glycol monoethyl ether acetate, and potassium dimethyldithiocarbamate. And in June 2007, OEHHA issued a Notice of Proposed Rule-Making announcing the proposed MADL for di-n-butyl phthalate. Written comments were received which we are reviewing and will respond to as part of the rule-making process.

Thank you.

CHAIRPERSON MACK: Thank you, Cindy. You've shown yourself able to pronounce those words much better than any of us.
There's one case that I think is still of interest to this Committee that's still in litigation and, that is, the cases that were filed in regard to exposures to acrylamide from french fries and potato chips. I think I've mentioned this to the Committee before, that there were some private cases that had been brought against Burger King and McDonald's. And then about a year ago, maybe longer than that now, the Attorney General's Office also filed some cases against a number of different defendants for the same kinds of exposures to acrylamide from potato chips and french fries. And they're basically -- it's litigation about whether or not there should be a warning for those foods -- exposures to acrylamide in those foods.

The cases have been proceeding along. Discovery is almost complete. It included the deposing of 20 expert witnesses on both sides of the case.

The trial is currently set for January the 15th in Los Angeles. Three defendants have settled and agreed to provide warnings, those being Burger King, Wendy's, and KFC. You may see those warnings popping up in their restaurants.

The remaining defendants include Frito-Lay,
Proctor & Gamble, McDonald's, Lance Heinz and Pepsico. And it's always possible that some of those will settle before the trial date. And it's also possible the trial date will change.

But that's the update on that case.

CHAIRPERSON MACK: I'll bet whoever took those depositions had a good time.

Thank you very much.

Dr. Denton.

DIRECTOR DENTON: This is the last item of the agenda, and that's the Summary of Committee Advice and Consultation.

So just to summarize the last I guess a little over an hour and a half, the Committee advice to OEHHA regarding the three chemicals brought before us, in all three cases basically the Committee did -- a majority Committee members thought that we ought to pursue the preparation of hazard identification materials on these three chemicals. Although in the case of TNT, it was unanimous; in the case of N,N-Dimethylformamide, it was 5 yes, 1 no; and then in the case of marijuana smoke, it was 4 yes and 2 no. So that's essentially it.

And regarding the next prioritization data screen, there was basically an endorsement of the proposal brought forward as far as using the screen 1 and screen 2,
basically screen 2, if you have one positive animal cancer bioassay. So kind of a combination of both.

Before I go on, Carol, did you want to say something?

CHIEF COUNSEL MONAHAN-CUMMINGS: I just wanted to mention on marijuana smoke, technically speaking that's not a majority of the Committee. The 4-2 would not be a majority because, at least under the rules for this Committee, we'd need at least five to vote positively. But it does give us some advice in terms of whether or not we'd proceed. It just it wouldn't be -- you know, if they were listing, for example, that wouldn't be sufficient.

DIRECTOR DENTON: Legally well put.

Thank you, Carol.

CHAIRPERSON MACK: Who wants to change his vote?

COMMITTEE MEMBER EASTMOND: I'm willing to change my vote to -- I'd like to go forward.

DIRECTOR DENTON: Okay. On marijuana smoke?

COMMITTEE MEMBER EASTMOND: Yes.

DIRECTOR DENTON: So we now have a majority of Committee members recommending that we go forward with marijuana smoke. So it's 5 yes and 1 no.

I only have a couple of other things that I wanted to mention.

I wanted to introduce our new staff counsel in

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the very beginning and I forget it. But at this point I
would like to introduce her. She's in the back. That's
Fran Kammerer.

STAFF COUNSEL KAMMERER: Kammerer.

DIRECTOR DENTON: And so Fran is going to be
working on Prop 65. And so the Committee will be seeing
her in future meetings.

The second thing that I wanted to mention is how
much again we appreciate Dr. Felton's service on the
Committee and his insight. It's really been very helpful,
and we will miss you.

And then I guess the second to the last thing
that I wanted to mention is that as an administrator of
the Proposition 65 process, this prioritization process I
think really makes logical sense to bring these chemicals
to you to have a preliminary discussion to see whether or
not more information should be developed. And so sitting
here is really kind of a reinforcement of that
prioritization process that we did adopt in 2004. This is
the first time that we've brought chemicals for your
advice and I think that it really makes a lot of sense.

And then I guess the last thing, and maybe Dr.
Mack will turn it back over to you, but I really do wish
that all of you have a happy holiday and we really
appreciate your service.
CHAIRPERSON MACK: Yeah, I think you better hold your fire until we see what happens when some of the next ones come through, because the epidemiology ones were relatively easy. It's the next batch that will be hard. Happy holidays, everybody.

Thereupon the Carcinogen Identification Committee adjourned at 11:55 a.m.)
CERTIFICATE OF REPORTER

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

That I am a disinterested person herein; that the foregoing California Office of Environmental Health Hazard Assessment, Carcinogen Identification Committee was reported in shorthand by me, James F. Peters, a Certified Shorthand Reporter of the State of California, and thereafter transcribed into typewriting.

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

IN WITNESS WHEREOF, I have hereunto set my hand this 28th day of November, 2007.

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