

MEETING
STATE OF CALIFORNIA
AIR RESOURCES BOARD
AIR QUALITY ADVISORY COMMITTEE MEETING

RADISSON HOTEL CONFERENCE ROOM
200 MARINA BOULEVARD
BERKELEY, CALIFORNIA

WEDNESDAY, JANUARY 23, 2002

10:30 A.M.

Reported by:
James Ramos

APPEARANCES:

For the Air Quality Advisory Committee:

Michael Kleinman, Ph.D., Chairman, UC-Irvine

John Balmes, M.D., Chief, UCSF

Russell Sherwin, M.D., UCSF

Sheldon K. Friedlander, Parsons Professor, UCLA

Constantinos Sioutas, D.Sc., Associate Professor, USC

Michael P. Sherman, M.D., Professor, UC-Davis

Dean Sheppard, M.D., Professor, UCSF

Ira B. Tager, M.D., M.P.H., Professor, UC-Berkeley

Gerry Cropp, M.D., Ph.D., Professor Emeritus, UCSF

George Thurston, Associate Professor, NYU

Also Present:

Richard Bode, Chief, ARB Health & Assessment Branch

Bart Ostro, Ph.D., OEHHA

David Mazzer, Ph.D., ARB Research Division

Michael Lipsett, M.D., J.D., OEHHA

Jeff Cook, Chief, Quality Management, ARB

Sue Wyman, Air Resources Board

George Alexeeff, Ph.D., DABT, Deputy Director, OEHHA

Margaret (Peggy) Jenkins, ARB

P R O C E E D I N G S

1
2 DR. ALEXEEFF: I would like to get underway
3 and welcome you all here. My name is George Alexeeff.
4 I'm deputy director for Scientific Affairs of the
5 Office of Environmental Health Hazard Assessment. And
6 we, with the Air Resources Board, have prepared this
7 report and the recommendations are from our office.

8 And I want to thank all of the -- I'll step
9 over here -- thank all of the members of the Peer
10 Review Committee that has been assembled, sort of our
11 Air Quality Advisory Committee Plus. And about 15
12 months ago or so we met here and discussed the
13 privatization process for a Children's Health Bill
14 that was passed with regards to air pollution and air
15 toxics, and we prioritized particulate matter into
16 tier one. And I'm very pleased that 15 months later
17 we actually have a report to the Peer Review on
18 particulate matter.

19 And I think that we're looking forward to
20 your comments on this document to improve it, and to
21 get any advice you have on the recommendation that
22 we're attesting to the Air Resources Board for
23 particulate matter standards.

24 So, with that, I'll go ahead and pass it over
25 to Richard Bode with the Air Resources Board.

1 CHIEF BODE: Thanks, George.

2 Good morning, Dr. Kleinman and members of the
3 Air Quality Advisory Committee. Actually, I can say,
4 the microphones we have here the court reporter tells
5 me are actually for his recording, they don't really
6 amplify our sound. We'll try our own voices this
7 morning and if we have any problems and we want
8 amplification, we can ask the hotel to do that.

9 UNIDENTIFIED SPEAKER: Richard, it is hard to
10 hear already.

11 CHIEF BODE: Is it? That's a bad sign.

12 UNIDENTIFIED SPEAKER: You'd better act now.

13 CHIEF BODE: Sue, maybe we ought to look into
14 getting some amplification.

15 MS. WYMAN: Okay.

16 CHIEF BODE: Part of my problem is I'm
17 talking to the Committee and so my voice isn't going
18 backwards too, so I'll try and speak a little louder
19 until we get some amplification in here.

20 Basically, I wanted to start off by saying
21 that this committee has a longstanding history of
22 providing scientific review and advice to both the Air
23 Resources Board, the Office of Environmental Health
24 Hazard Assessment, and before that, the Department of
25 Health Services, that today's meeting actually takes

1 special prominence for this committee since changes in
2 state law have now required that the California
3 Environmental Protection Agencies, when they're making
4 changes to either regulatory actions or policies that
5 they actually -- regulatory actions -- that their
6 scientific justifications for those actions go under a
7 scientific peer review. And that committee is to be
8 appointed by the president's office of the University
9 of California.

10 So this committee that's meeting here today
11 was specifically appointed to review the draft
12 document that's before you today. This document was
13 prepared jointly by the Air Resources Board and the
14 Office of Environmental Health Hazard Assessment, and
15 contains our draft recommendations. And I'll stand --

16 UNIDENTIFIED SPEAKER: Stand in the front.

17 CHIEF BODE: As long as I don't have to
18 dance.

19 [Laughter]

20 CHIEF BODE: I've got to make sure the court
21 reporter can still hear me.

22 So basically, before you is our draft, draft
23 joint report which contains our draft recommendations
24 for modifying and actually revising the California air
25 quality standards for particulate matter and sulfates.

1 I want to thank you, as George already has,
2 for taking time out of your already busy professional
3 lives and giving us your advice in reviewing this
4 document, and we look forward to the discussions that
5 we have today.

6 Just to kind of begin, I thought what we'd do
7 is -- what I thought I'd actually do is I'd actually
8 allow the Advisory Committee to maybe introduce
9 themselves, starting with Dr. Kleinman. Kind of
10 introduce yourselves, your specialty and maybe your
11 affiliation for everyone in the room.

12 CHAIRMAN KLEINMAN: I'm Michael Kleinman and
13 I serve as the chair on the Air Quality Advisory
14 Committee. I'm a professor at the University of
15 California at Irvine in the department of community
16 and environmental medicine. And my primary research
17 is in the field of inhalation toxicology.

18 I also want to introduce, to my right,
19 Dr. George Thurston, who is here as an advisor to
20 OEHHA and to ARB, and, although he's sitting at this
21 table, that's only because there was no chair next to
22 Bart.

23 PROFESSOR THURSTON: I wanted to sit next to
24 Bart, really.

25 [Laughter]

1 CHAIRMAN KLEINMAN: So, George, why don't you
2 introduce yourself and we'll go around the table.

3 PROFESSOR THURSTON: Yeah. Again, my name is
4 George Thurston. I'm on the faculty at the NYU School
5 of Medicine and I'm associate professor there, and I
6 do research into the health effects of air pollution
7 there. And, as was stated, I'm here in, you know, an
8 advisory capacity to the Office of Environmental
9 Health Hazard Assessment.

10 PROFESSOR SHEPPARD: I'm Dean Sheppard. I'm
11 a professor of medicine at University of California,
12 San Francisco where I direct the Lung Biology Research
13 Center and the UCSF Sandler Center for Basic Research
14 in Asthma.

15 PROFESSOR SHERMAN: I'm Michael Sherman. I'm
16 a professor of pediatrics at UC, Davis. I'm the chief
17 of neonatology at that institution, and my field of
18 research is pulmonary immunobiology.

19 DR. BALMES: I'm Dr. John Balmes. I'm a
20 professor of medicine at University of California, San
21 Francisco. I have a laboratory in the Lung Biology
22 Center where Dean is the director where I do
23 controlled human exposure studies of air pollutants,
24 looking at respiratory health effects. And I also
25 collaborate on several other epidemiologic studies

1 with Dr. Tager, whom you'll hear about in a second.

2 PROFESSOR FRIEDLANDER: I'm Sheldon
3 Friedlander. I'm a professor of chemical engineering
4 at UCLA, and I'm the director of the aerosol
5 technology laboratory. Aerosol is the small particles
6 in gases, so that is my research interest. I was the
7 first chair of EPA's Clean Air Scientific Advisory
8 Committee, which established the early federal
9 standards for particulate matter.

10 PROFESSOR CROPP: I'm Gerry Cropp, I'm
11 professor emeritus of clinical pediatrics at the
12 University of California, San Francisco. I just
13 stepped down as the chief of the pediatric pulmonary
14 group at UCSF, and I'm also the editor-in-chief of a
15 journal called Pediatric Pulmonology, which devotes
16 itself to all aspects of lung health in children.

17 PROFESSOR TAGER: I'm Ira Tager. I'm a
18 professor of epidemiology at the School of Public
19 Health at UC, Berkeley, and my research interests are
20 primarily epidemiologic studies of the health effects
21 of the environment on the human lung, particularly in
22 children.

23 PROFESSOR SIOUTAS: I'm Costas Sioutas. I'm
24 an associate professor at the department of civil
25 environmental engineering at the University of

1 Southern California. I'm also the deputy director of
2 the Southern California Particulate Matter Center and
3 Supersite, a very large research center that we do
4 everything from exposure measurements to epidemiology
5 to toxicology. My specific field of expertise is
6 particles, aerosols and development of monitoring
7 technologies or technologies that now some of my
8 colleagues in this room are using to expose animals or
9 humans to determine health effects of particulate
10 matter.

11 DR. SHERWIN: I'm Russell Sherwin at the
12 University of Southern California, department of
13 pathology. My special area is environmental lung
14 pathology. I deal with mostly humans, though we do
15 have experimental programs as well.

16 CHIEF BODE: Great. Thank you very much.

17 CHAIRMAN KLEINMAN: Thank you. Before we
18 turn the floor over to Richard Bode, I wanted to
19 mention that we're going to have a minor modification
20 of the agenda. I've asked Bart Ostro to -- if you'll
21 notice, at 10:50 there will be a discussion of health
22 effects, and what I've asked is that we hold off on
23 the recommendations until the second on monitoring
24 methods is presented, and then to present the
25 recommendations and the way in which the

1 recommendations were arrived at at the end so we have
2 a total picture.

3 So, with that I'll turn it over to you,
4 Richard.

5 CHIEF BODE: Okay, thank you. I'd also like
6 to introduce Sue Wyman. If you've got any questions,
7 anything with the room, Sue put together all of the
8 logistics for this meeting, and thank you, Sue, for
9 doing that.

10 MS. WYMAN: You're welcome. We have two mics
11 running as we speak, so there shouldn't be a problem,
12 as well as unfortunately, for those of you who haven't
13 heard, this hotel decided to refurbish all women's
14 bathrooms at the same time.

15 [Laughter]

16 MS. WYMAN: For us of the female persuasion,
17 we have to either run downstairs, or I have two keys
18 to two hotel rooms. And what I did, instead of
19 everybody walking up here so that everybody knows you
20 have to use the bathroom, I put them on the chair by
21 the door, as well as, gentlemen, if you don't want to
22 go downstairs either -- Apparently, this floor doesn't
23 have a bathroom -- you can use a hotel room as well.
24 It's basically just down and around the corner in a
25 hotel room.

1 The only condition is that if you're missing
2 more than ten minutes, I'm coming after you, because
3 you're probably taking a nap or you decided to watch
4 TV or something --

5 [Laughter]

6 CHIEF BODE: Good. I'm glad you introduced
7 that, I'd hate to offend anyone, so --

8 Just some simple logistics too, for the
9 committee members, in your packets we put the basic
10 committee roster, the agenda, executive summary to the
11 report. Also, for the public outside, we've got those
12 materials on the table outside. So if you didn't get
13 a copy, feel free to grab one.

14 We've also got copies of reports out there.
15 If we run out of copies, you might either leave Sue a
16 copy of your business card and we'll mail you a copy
17 when we get back, we'll make some more of those, and
18 we've also got copies of the technical support
19 document, the appendices. And actually, we didn't
20 bring any copies of those, we didn't get them back
21 from reproduction. So if you want copies of those,
22 again, leave Sue your business card and we'll mail
23 those off to you.

24 We have two sign-up sheets outside, and I'd
25 ask everybody to sign in on the attendance sheet,

1 please sign in. We have also got a sign-in for those
2 wishing to make oral comments in the committee
3 tomorrow morning, and please sign in to that. What
4 we'll do is pick that up at the end of the day today
5 and make it available to the chairman, and we'll be
6 using that to decide how to divide up the speaking
7 time.

8 Basically, that public comment period,
9 according to the chairman's wishes, is really to
10 summarize written comments that have been submitted,
11 rather than presenting original arguments so that we
12 have enough time for everyone really to make an oral
13 presentation if they wish to.

14 One last thing: We do have a court reporter,
15 and the microphones, like I said, are for the court
16 reporter. So anything you say this morning will be on
17 the record and we'll be able to review that. With
18 that, I think I've covered all of the logistics.

19 Any questions up to this point?

20 Okay. The agenda for the meeting,
21 Dr. Kleinman already mentioned that he's going to
22 change the agenda a bit. Also, the times on the
23 agenda are approximate that we'll see how the
24 discussions go and they -- you know, the chairman may
25 wish to change some of the discussion times, based on

1 what happens this morning.

2 Finally, I'm just going to make a little
3 introduction of the staff making presentations this
4 morning. We will begin with a presentation by
5 Dr. David Mazzera from the Air Resources Board, who
6 will basically overview the process of putting
7 together the draft review document that you have, the
8 little bit of the purpose and the reason that we're
9 coming here today.

10 He'll be followed by Dr. Bart Ostro from the
11 Office of Environmental Health Hazard Assessment, who
12 will explain the health effects data that's been
13 reviewed and the interpretation of that data.

14 And finally, Jeff Cook from our monitoring
15 laboratory division will discuss the monitoring
16 methods that are being recommended to support the
17 standards.

18 And, with that, I'll let David move ahead.

19 DR. MAZZERA: Thank you, Richard, and thanks
20 everyone for taking time out of their busy schedule to
21 participate in this meeting, we appreciate that.

22 My name is Dave Mazzera. I'm with the Air
23 Resources Board Research Division, and in order to put
24 the standard review process into some type of
25 perspective, I'll be covering the following, and I'll

1 try to do this as timely as possible: the ambient air
2 quality standard reviews, why we do them. I'll
3 briefly review the PM and Sulfate Review document, so
4 we have an understanding of what the contents of that
5 document are. That will be followed by a description
6 of the standard-setting process. And finally, I'll
7 cover the time line for this first review of the PM
8 and Sulfate standards.

9 Why do we review standards -- in this case,
10 for particulate matter? We review them primarily to
11 determine if, based on recent findings, the standard
12 adequately protects public health; however, in
13 addition and of equal importance, we desire also to
14 address the mandated requirements of the Children's
15 Environmental Health Protection Act, also known as
16 SB 25.

17 Now, the Children's Environmental Health
18 Protection Act has many important requirements;
19 however, the most immediate importance for the
20 standards are listed on the slide. The ARB, in
21 consultation with OEHHA, has been mandated to
22 determine which of the standards are not protective of
23 public health, with particular consideration given to
24 children and infants.

25 Now, out of all of those standards that then

1 need to be reviewed because they were determined
2 inadequate, it must be prioritized based on the
3 potential risk to public health. So we take all of
4 the standards and we prioritize them.

5 We must then revise the highest priority
6 standard by the end of this year, so by the end of
7 2002, with a revision of the remaining standards to
8 occur at the rate of one per year. From this initial
9 review of all of the standards, it was determined that
10 most, actually all did not adequately protect the
11 health of the public, including infants and children.

12 The list of standards found inadequate were
13 then prioritized into the following: PM 10 including
14 sulfates, under the first priority, PM 10 including
15 sulfates, ozone, and nitrogen dioxide. Under the
16 second priority, lead, hydrogen sulfide, sulfur
17 dioxide and carbon monoxide. First priority standards
18 represent those with the greatest potential risk to
19 public health. Particulate matter, at the level of
20 the current standard, was determined to pose the
21 greatest risk to public health and is, therefore,
22 being reviewed first.

23 In addition, we are mandated to complete this
24 first detailed review and revise the first standard by
25 the end of this year, like I mentioned; however, the

1 chairman of the Air Resources Board emphasizes concern
2 of the public health impacts of PM, and would,
3 therefore, like to expedite the review to ensure
4 public health protection, with a target date of May
5 2002.

6 Now, in January of 2001, staff from the ARB
7 and the Office of Environmental Health Hazard
8 Assessment formed the PM Standards working group. The
9 working group diligently compiled a large body of
10 information related to particulate matter, and
11 ultimately generated a staff report for the Air
12 Quality Advisory Committee with the major topics
13 listed here: Physics and Chemistry, Sources and
14 Emission, Measurements, Exposure, Welfare Effects,
15 Health Effects, Controls or Direction Strategies, and
16 Quantifying Adverse Health Effects. These are the
17 major components of the document of which you're well
18 aware of.

19 The draft document was sent to the committee
20 on November 30th, 2001, which then opened the public
21 comment period originally scheduled to be closed by
22 December 30th but was later extended to January 11th
23 of 2002. On January 14th those comments were
24 forwarded to Air Quality Advisory Committee for their
25 consideration for this process.

1 Next let's take a brief look at the ambient
2 air quality standards. California Health and Safety
3 Code authorizes the Air Resources Board to adopt
4 standards for ambient air quality in consideration of
5 public health, safety and welfare, including but not
6 limited to health, illness, irritation to the senses,
7 aesthetic value, interference with visibility, and
8 effects on the economy.

9 Ambient air quality standards represent the
10 legal definition of clean air. The legal definitions
11 are defined in the key elements required for each
12 standard. Most important, a standard specifies the
13 concentrations and durations of exposure to air
14 pollutants that reflect the relationship between the
15 intensities and composition of air pollution and
16 undesirable effects.

17 Ultimately, an objective of an ambient air
18 quality standard is to provide a basis for preventing
19 or abating health and welfare effects of air
20 pollution; however, it is important to note that a
21 standard does not define what control measures must be
22 taken. That occurs through another process but is not
23 part of the standard-setting process.

24 Now, what are the key elements of an ambient
25 air quality standard? It includes a definition of the

1 pollutant; for example, PM 10 in this case,
2 particulate matter of an aerodynamic diameter of ten
3 microns or less; an averaging time, for example, an
4 annual average or 24-hour average; a concentration,
5 which is a level of the defined pollutant or a
6 specified adjutant time not to be exceeded; and
7 finally, a standard must have an identified monitoring
8 method or methods.

9 Now, as part of the standard review process
10 the Health and Safety Code defines the role of the Air
11 Quality Advisory Committee. The Health and Safety
12 Code requires that the scientific basis of the methods
13 used in making the recommendations for an air quality
14 standard be peer reviewed by the Air Quality Advisory
15 Committee. Specifically, the committee is required to
16 review the recommendations in the document and, in
17 doing so, may consider both written and oral public
18 comments, and must then prepare a written evaluation
19 staff report describing the scientific basis for the
20 proposed ambient air quality standards.

21 Now, briefly, let's take a look at the time
22 line for this review process. In November of 2001,
23 the report was released to the Air Quality Advisory
24 Committee and the public. Then in December of 2001 we
25 had a series of public workshops throughout the state.

1 Now in January we're going through this process with
2 Air Quality Advisory Committee.

3 In March of 2002 a draft report will be
4 released that will begin a 45-day public comment
5 period prior to the board meeting. In April of 2002
6 we'll have another series of public workshops. And in
7 May of 2002 the goal is to bring the final
8 recommendations and to present them at the board
9 hearing.

10 Now, the current -- Okay, next, just as a
11 refresher, let's take a look at the current PM
12 standards. California currently has three PM
13 standards to protect public health: a filter-based
14 high-volume site-selective inlet method, with an
15 average of 30 micrograms per cubic meter calculated as
16 a geometric mean, which is intended to protect against
17 long-term health effects; and a 24-hour average of 50
18 micrograms per cubic meter, which is intended to
19 protect against short-term health effects.

20 California also has a total suspended particulate
21 method for sulfates, with a 24-hour average standard
22 of 25 micrograms per cubic meter. So these are the
23 current standards.

24 Now, the proposed recommendations for
25 updating the standards include the following: For PM

1 10, it is proposed to reduce the annual average from
2 30 micrograms per cubic meter to 20 micrograms per
3 cubic meter pertaining to the 24-hour standard of 50
4 micrograms per cubic meter. For PM 2.5, at an annual
5 average standard of 12 micrograms per cubic meter.
6 For sulfates, pertaining to the 24-hour average
7 standard of 25 micrograms per cubic meter, and propose
8 an alternate monitoring method for that standard.

9 Now, as far as monitoring methods, which
10 we'll talk about in more detail in a little bit, it is
11 proposed to adopt the existing federal reference
12 methods for PM 10, adopt an existing federal
13 recommended method for the new PM 2.5 proposed
14 standard, use ARB method 007 for PM 10 sulfate, so
15 that will be the updated monitoring method. And, in
16 addition, possibly designate continuous methods as
17 acceptable for PM 10 and for PM 2.5, as well as retain
18 provisions for identification of other methods that
19 are acceptable to ARB.

20 Now, if I briefly can go back to these, I
21 just want to mention a couple of things. This plot is
22 showing trend data in annual PM 2.5 concentrations for
23 several sample locations -- I'm sorry. This plot is
24 showing trends in annual PM 10 concentrations for
25 several basins in California from 1990 to 2000.

1 Concentrations are presented as annual geometric
2 means.

3 Now, it's typical from this plot, this trend
4 plot to describe an overall trends to data; however,
5 it is important to point out that the annual
6 concentrations for several of the air basins
7 consistently exceed the current standard of 30
8 micrograms per cubic meter. And similarly, this plot
9 shows trends in annual PM 2.5 concentrations for
10 sample locations from the PM 2.5 dichot network in
11 California from 1988 to 1999. While data from the
12 dichot network is no longer being collected, data for
13 the PM 2.5 based on the federal reference method have
14 been collected only for the past several years.

15 So the point of this trend graph is really to
16 show that PM 2.5 concentrations around the state have
17 remained relatively consistent over time. The graph
18 shows annual PM 2.5 concentrations from the dichot
19 samples ranging from approximately 10 to 25 micrograms
20 per cubic meter from various monitoring locations
21 around the state. So basically, I just wanted to show
22 these two graphs so you have an idea of what
23 concentrations are over time for PM 10 and PM 2.5.

24 Next I want to introduce Dr. Bart Ostro from
25 the Office of Environmental Health Hazard Assessment,

1 who will present OEHHA's proposed recommendations
2 along with the rationale and scientific basis for
3 these recommendations. He will then be followed by
4 Jeff Cook from ARB's Monitoring Laboratory Division,
5 who will discuss proposed monitoring methods in
6 support of the proposed standards.

7 DR. OSTRO: So we were responsible for
8 chapter seven, the health effects, and I'd like to
9 acknowledge my co-authors on the chapter, Dr. Lipsett,
10 who is sitting with me here, and Dr. Broadwin, who is
11 in the first row over there. And any errors, of
12 course, are theirs, not mine.

13 [Laughter]

14 DR. OSTRO: There were four basic questions
15 that we wanted to address in this chapter. First, is
16 there evidence of health effects at or below the
17 current standards; second, what is the general weight
18 of evidence; third, is the evidence consistent and
19 coherent; and fourth, is there evidence of causality.

20 As Dave mentioned, we had a meeting in
21 October 2000 where the pre-existing members of AQAC,
22 the Air Quality Advisory Committee endorsed the
23 recommendations of OEHHA and ARB that the PM standards
24 were not necessarily protective of public health,
25 including that of infants of children, with a margin

1 of safety. And in December, those recommendations
2 were brought to the Air Resources Board, the political
3 appointees of the board, and they accepted these
4 recommendations and asked for a formal and expedited
5 review of the standards, which is where we are now.

6 Now, the second question is what is the
7 weight of evidence. There are hundreds of
8 epidemiologic, toxicological and clinical studies
9 examining the effects of particulate matter in
10 different forms and health. An overwhelming number of
11 these studies indicate strong and significant
12 association between particles measured typically as PM
13 10 and a whole range of health effects. We emphasize
14 in our chapter a lot of the epidemiology because of
15 the nature of particles. Since it's so heterogeneous
16 regarding particle sizes and chemistry that until
17 recently there hasn't been a lot of talks and clinical
18 evidence to draw on, so epidemiology -- unlike many of
19 our other standards, but this standard epidemiology
20 carries a lot of the weight, a lot of the evidence.

21 Now, the recent reviews of the epidemiology
22 have been used by several organizations -- US EPA and
23 the World Health Organization, the European Union, the
24 UK and Canada, to name a few -- as a basis for
25 standard setting. And we thought that we would try to

1 highlight the major studies, not do an exhaustive
2 review of all of the studies which really, the US EPA
3 does in their criteria document -- We don't really
4 have the resources, the time or the inclination to
5 replicate that -- but rather, we really wanted to try
6 to highlight the major studies.

7 Now, this is a chart just to show the AQAC
8 members where our standards are and our proposals are
9 relative to what other people are doing or have done.
10 First, regarding the PM 10 standards, you can see that
11 US EPA's is at 50, our previous is at 30, and for the
12 next couple of years, the European Union has a
13 standard of 40, and that's going to be converted to an
14 annual average standard of 20 for the year 2010. So
15 our proposal of 20 is somewhat in keeping with the
16 European Union's proposal, or actual promulgated
17 standard for annual average.

18 For PM 2.5 annual average, US EPA has
19 proposed a 15 and actually tried to promulgate it, and
20 as probably most of you know, that's been in court, a
21 legal situation since then. When they went out with
22 their proposal of 15 they said they would consider a
23 range of 12.5 to 20 for their annual average. Our
24 proposal is 12 micrograms per cubic meter for PM 2.5.

25 Regarding the 24-hour average, again US EPA

1 is at 150. Our current standard and proposed
2 continued standard would be 50 micrograms. The
3 European Union has also a standard of 50 with 35
4 exceedences for the year 2005, to be dropped to seven
5 exceedences in the year 2010. And then finally for PM
6 2.5, the 24-hour standard, the US EPA proposed 65 with
7 a range of 20 to 65, and Canada has a PM 2.5 24-hour
8 standard of 30 to be met by the year 2010. So that's
9 an overview of where we fit in the international
10 picture.

11 Regarding the weight of evidence, as I'd
12 indicated, a lot of the evidence is dependent on the
13 epidemiologic studies, and I wanted to just start with
14 a little intro on what are some of the pros and cons
15 and advantages and disadvantages of using some of
16 these studies. And basically, they've real-world
17 exposures, short- and long-term can be looked at over
18 a wide range of conditions; that is, different housing
19 conditions, different behavior patterns, different
20 base line health status and so on. There's no need to
21 extrapolate to different doses or to different
22 species, and we can also consider a wide range of
23 health responses.

24 We can also examine lots of different
25 segments of the population. We can look at elderly

1 people, people with asthma, people with heart disease,
2 children and infants, and there are studies on all
3 these different elements where you would be able to
4 study them, of course in clinical settings.

5 On the down side, of course, we have less
6 precise measurement of exposure. Typically these
7 studies use central site fixed monitors or an average
8 of monitors. Sometimes they use monitors closer to a
9 designated community when we're doing a targeted panel
10 study. And also, of course, we need to worry about
11 potential confounders, other factors that might
12 explain the health outcome. Besides air pollution, we
13 want to make sure that those things are controlled or
14 taken care of in the studies.

15 So most of the analysis that we review in our
16 document relies on multiple regression techniques, and
17 in multiple regression techniques, typically we have
18 health as a function of wide range or independent
19 explanatory variables, one of which is pollution.
20 There might be other pollutants in the model as well.
21 We would probably control for weather, seasonality,
22 things like the time of the study over the years, we
23 want to control for just time. There's day-of-week
24 effects on certain health outcomes.

25 And also, depending upon the type of study,

1 of course, we want to control for individual risk
2 factors. So these things all go into a multiple
3 regression. And the beauty of the technique allows
4 you to isolate the effects of pollution while
5 controlling or netting out the influence of the other
6 potential risk factors.

7 Recent developments in statistical techniques
8 and software provide, over the last couple of years,
9 an amazing new range of methods to look at some of
10 these models and to look at some of the evidence.

11 There are smoothing techniques that I'll talk about
12 briefly and simulation techniques that now can be done
13 on PC's in a minute where it used to take an hour or a
14 day to do it years ago; methods to address potential
15 confounders, autocorrelation in the data, and also,
16 ways to aggregate the data in meta-analytical
17 approaches. So there is a lot of new software that is
18 being used and new statistical approaches that have
19 been used over the last couple of years that have
20 become almost a requirement for publishing some of
21 these studies.

22 Regarding the weight of evidence, we have
23 results from now five continents regarding linking a
24 particular matter to a wide range of outcomes,
25 everything from premature mortality due to both short-

1 and long-term exposure, cardiovascular and respiratory
2 hospitalization, emergency room visits, and urgent
3 care visits in general, doctor visits; worsening of
4 asthma, bronchitis in children due to longer-term
5 exposure, usually of a year or so. And more minor
6 outcomes, like work loss and school absenteeism,
7 respiratory symptoms and decrements in lung function.
8 So the weight of evidence shows really a coherent
9 pattern from relatively minor and transient outcomes
10 to very, very severe outcomes.

11 Now, when these studies were conducted, they
12 have been reporting ranges over a wide range of
13 different factors. And it's important to recognize
14 that because these studies have been repeated in so
15 many different parts of the world that you get a true
16 mix of the underlying conditions. So if only one
17 study or three studies were done in Oakland and you
18 had those findings and those were the only findings,
19 that would certainly tell you one thing about the
20 potential uncertainty and the potential causality of
21 the findings.

22 When you repeat it over and over again in
23 many different environments, physical and social and
24 economic environments, that adds a lot of power to the
25 conclusion that you can draw from the epidemiologic

1 studies. In fact, it's one of the strongest things
2 you can do is repeat the same study using roughly the
3 same data in an entirely different situation.

4 And that's what's happened here. The studies
5 have been conducted over a wide range of climates and
6 seasonal patterns, a wide range of PM 10
7 concentrations and mixtures of particles and other
8 pollutants, a wide range of different weather co-
9 variants, where sometimes you might be concerned about
10 humidity and temperature and other times you might be
11 concerned about dryness and other things; a wide range
12 of population characteristics, age and smoking status,
13 and ranges of occupational exposure; and also, things
14 like housing stock, which might affect the penetration
15 from pollution outside inside.

16 So the studies have been conducted over a
17 wide range of these different factors. And again,
18 consistently report associations between PM 10, other
19 forms of particles, and some of these health effects
20 that I've referred to.

21 Now, more specifically, the evidence in our
22 document has been divided into results from relatively
23 short-term exposures, what we call acute exposures,
24 and relatively long-term exposures, although the
25 differences between those two types of exposures are

1 sometimes a little hazy.

2 As I indicated, the studies have been
3 conducted throughout the world. Regarding short-term
4 exposures, by that we mean a day or multi-day
5 exposure, sometimes five to seven days of exposure,
6 there's over 200 cities now that have reported
7 associations between air pollution, PM 10 and
8 mortality; typically, these studies have used many
9 years of data for a city, often three to four years,
10 sometimes seven to ten years.

11 And regarding mortality, they've examined the
12 relationship between daily concentrations of PM 10 or,
13 as I said, multi-day, two-or-three-day averages
14 sometimes, and the daily counts of mortality; that is,
15 the total number of deaths that occur in that city.
16 So it's not looking at an individual, per se, looking
17 at the counts of mortality that occur on a given day,
18 and seeing if there's a correlation between those two,
19 after accounting for other factors that might explain
20 the daily changes; for example, weather factors,
21 seasonal factors, and, as I indicated, day of week and
22 other types of factors, anything else that might
23 explain daily changes in mortality.

24 PROFESSOR FRIEDLANDER: And when you --
25 Mortality refers to respiratory-associated deaths?

1 DR. OSTRO: In this case we're talking about
2 lots of different forms. Sometimes all-cause
3 mortality, mortality from any cause. Net of accidents
4 and homicides are usually taken out of the data, so
5 it's so-called natural mortality. Also,
6 cardiovascular-specific mortality, and --

7 PROFESSOR FRIEDLANDER: Are taken out or
8 incorporated?

9 DR. OSTRO: The accidents and homicides are
10 taken out --

11 PROFESSOR FRIEDLANDER: And cardiovascular?

12 DR. OSTRO: -- and then cardiovascular as a
13 separate category are looked at. And then sometimes
14 subcomponents of cardiovascular, and then respiratory
15 mortality as well. So usually, all three of those,
16 and then sometimes people look at mortality for older
17 age groups, like people above age 50 or above 65,
18 because those tend to be the more sensitive regarding
19 mortality.

20 The important thing to remember about these
21 studies is that the individual level factors are
22 basically constant on a day-to-day basis. So age and
23 smoking status and other things that typically might
24 affect mortality when you do these time-series studies
25 are basically constant. So you only have to worry

1 about things that change on a daily basis, like
2 weather, day of week, other pollutants, and particles.
3 So it's a nice way of looking at the data.

4 So, Dr. Friedlander, in answer to your
5 question, the studies have, as I indicated, looked at
6 many different types of outcome for mortality.
7 Typically we see stronger effects for the
8 cardiovascular mortality as you might expect, and
9 stronger effects for the older age groups than we do
10 for all-cause mortality.

11 Our document cites 64 single-city studies
12 using PM 10, so it's important to know that other PM
13 metrics, like PM 2.5, coarse particles, black smoke
14 called fission of haze, and extinction coefficient
15 have all been used and have all shown associations
16 with daily changes in mortality, but we didn't want to
17 have to worry about how to extrapolate from those or
18 translate from other measures of PM 10 to PM 10.

19 So we focused our document in table one on
20 studies using PM 10. So there are 64 plus or minus a
21 few. We might have lost some or missed some among the
22 literature. And we tended to de-emphasize some of the
23 other metrics that were used, and we focused on the
24 single-city studies, because in those studies
25 researchers tried to really develop models very, very

1 specific to those cities.

2 What we found when we reviewed all of those
3 time-series studies, these studies of daily mortality,
4 that there was a consistent association between PM 10
5 and mortality, and a very common range of effects of
6 between .5- and 1.5-percent increases in daily
7 mortality per 10 micrograms of PM.

8 I don't know if you can see this clearly,
9 it's also in the report, but this is a list of our 64
10 or so studies that specifically use PM 10. I mean,
11 absent here is the percent increase in daily mortality
12 and PM 10 concentrations here, and our assessment was
13 that a lot of the studies fall into the range of 1.5
14 to .5, within this range here. You get a lot of the
15 studies in there. But, of course, you will get
16 studies higher, showing higher and lower effects. So
17 the dominant effects appear to be in the .5 to 1.5
18 range. That's 1.5 percent change in daily mortality
19 for ten micrograms of PM 10.

20 CHAIRMAN KLEINMAN: By daily mortality you
21 mean all-cause?

22 DR. OSTRO: In this case it would be all-
23 cause mortality. For cardiovascular mortality, you
24 know, it might be a little higher, and for elderly
25 groups it might be a little higher as well.

1 PROFESSOR SHEPPARD: This is the X axis daily
2 or annually?

3 DR. OSTRO: This is the long-term average of
4 the study, so it's looking at -- All the studies use
5 24-hour average PM 10, and then this would be the
6 average of the three or four years value as a PM 10.
7 So it's really a long-term average of the 24-hour
8 average.

9 CHAIRMAN KLEINMAN: Bart, I don't know if
10 it's part of the presentation, but for reference, you
11 mentioned that you had 64 studies that did daily PM 10
12 measurements. By comparison, how many studies had
13 daily PM 2.5 measurements?

14 DR. OSTRO: Okay, we do have that in the
15 report, and I think there are nine studies that use PM
16 2.5, plus or minus two studies, but I think about nine
17 studies that actually specifically measured PM 2.5.
18 There are other studies that again approximated PM 2.5
19 using extension coefficients or relating PM 2.5 to
20 other measures, but those that have specifically
21 measured it I think are about eight or nine.

22 CHAIRMAN KLEINMAN: Thank you.

23 DR. OSTRO: The other thing to note about the
24 studies is that it appears that there is a slightly
25 greater uncertainty about the association for those

1 studies conducted at lower concentrations.

2 Again, this figure is also in the document.
3 And I don't know if this is a -- I mean, this
4 certainly is a debatable issue here about statistical
5 significance and all, but when you go down to the
6 lower concentrations, what we have now is the 95-
7 percent confidence that will go around the central
8 estimate from the studies.

9 And our assessments showed that as you got
10 into lower levels, say below 25 or so, you tended to
11 see a lot more studies where the confidence interval
12 included zero, based on a 95-percent level. And
13 again, there are problems with that interpretation
14 that is using a strict coherence, adherence to a .05
15 as a projector, except -- but certainly we can see
16 that there's greater uncertainty at these lower
17 levels. There might be lots of reasons besides the
18 fact that it's this lower PM 10 that would explain
19 that, but at least it is an observation that as you go
20 to the lower levels, we did, in fact, find slightly
21 greater uncertainty or a greater likelihood that the
22 confidence interval would include zero.

23 DR. LIPSETT: For those of you who are
24 looking for this in your document, it's on page 122.

25 PROFESSOR CROPP: And are these mortalities

1 over age 65 or what mortality?

2 DR. OSTRO: This is all-cause mortality.

3 PROFESSOR CROPP: All-cause mortality.

4 DR. OSTRO: If we looked at those over 65,
5 probably everything would be higher, and it would be
6 less likely to overlap the zero.

7 PROFESSOR CROPP: But wouldn't you expect
8 there to be a greater scatter, as indicated by the
9 greater variations in the means at lower
10 concentrations and, consequently, the noise at that
11 level, at the lower concentrations is much greater.
12 And, consequently, there may be considerable
13 statistically significant evidence but, you know, it's
14 a bit blurred by the noise.

15 DR. OSTRO: Right, I think so. That might be
16 the total explanation for it.

17 PROFESSOR CROPP: Because at the higher
18 concentration there's very little noise.

19 DR. OSTRO: The signal is stronger, right.
20 So it might not be that we're really less certain of
21 the effect, it just might be a little harder to
22 distinguish it statistically.

23 Now, besides these 64 or 67 single-city
24 studies that we talked about, there have been several
25 multi-city studies; that is, where a group of

1 researchers look at many cities at once. And among
2 the most recent and largest was a series of studies, a
3 study funded by the Health Effects Institute. For
4 those of you who don't know about HEI, it's an
5 independent institution jointly funded by the US EPA
6 and the auto industry. It has a very serious and
7 significant review process and review panel as part of
8 it.

9 And, in order to address the issue of series
10 times studies and mortality, they funded John Samet,
11 who is the chairman of the epidemiology department at
12 Johns Hopkins, and Scott Zeger, who is the chairman of
13 the biostatistics department at Johns Hopkins, to
14 conduct a study of what ultimately turned out to be
15 the 88 largest cities or counties in the United
16 States. And they also did a subanalysis of the 20
17 largest cities in the US.

18 And the whole concept was, rather than having
19 separate authors do separate cities and all that, to
20 try to use a consistent approach over all the cities,
21 use relatively similar years, similar data, similar
22 models and conduct a wide range of sensitivity
23 analyses of the results to see if there was an
24 association between PM 10 and mortality. Also, to
25 address the issue of publication bias, and also to add

1 statistical power; that is, almost to address your
2 question that you might not see an effect in one
3 smaller city, but you can look at the weight of
4 evidence and combine the results across all the cities
5 together. So it adds a lot of statistical power to
6 the overall project.

7 And what they found in their analysis was
8 statistically significant association between PM 10
9 and several of the mortality indicators, again all-
10 cause mortality and cardiovascular and I believe
11 respiratory mortality again as well. And they
12 indicated that the results were consistent with the
13 previous single-city studies that had been conducted
14 to date. And they also found in their analysis of the
15 88 cities that the strongest effects appeared to be in
16 the Northeast and the Southern California area, but in
17 their document they say that there doesn't seem to be
18 strong heterogeneity across the cities; that is, the
19 results across the cities appear to be pretty
20 consistent.

21 Specifically, to quote Dr. Samet in the New
22 England Journal article that was published, "We found
23 consistent evidence that the level of PM was
24 associated with the rates of death for all causes and
25 from cardiovascular and respiratory causes. The

1 association of PM 10 was not affected by the inclusion
2 of other pollutants," and this I'll get into later.
3 "Our findings strongly support the findings of prior
4 studies of PM and mortality."

5 And then their independent review panel
6 concurred and said, also concluded that the evidence,
7 the effect on both deaths and hospitalizations, which
8 was another component of this HEI study, looking at
9 hospitalizations and PM, tend to be regarded as
10 compelling and consistent. So the findings of this
11 very large study seem to very strongly support the
12 findings of the individual city studies.

13 Also, it's important to note there have been
14 several other multi-city studies that have been
15 conducted over the last couple of years. There has
16 been a long tradition of studies, what's called the
17 Harvard Six Studies, a project started in the '70's.
18 Schwartz recently published several articles looking
19 at ten US cities together. Burnett published a study
20 of the eight largest Canadian cities. And Klea
21 Katsouyanni published several studies on results of 29
22 European cities, using both PM 10 and black smoke.

23 And all of these studies together clearly
24 show associations between particulate matter --
25 specifically, PM 10 -- and daily mortality. So there

1 is a wide range of evidence from both the single-city
2 and from the multi-city studies looking at daily
3 changes in concentrations of PM 10, or sometimes
4 several-day averages of PM 10.

5 A related issue to this is what about
6 coherence when we're talking about the weight of
7 evidence. Do you just find one set of outcomes and
8 nothing else. If something is going on, you should
9 find a whole continuum of effects. And, in fact,
10 often using similar techniques to these time-series
11 studies that I've indicated, there have been a wide
12 range of outcomes that have also been found.

13 And, for example, with these hospitalization
14 studies and urgent care visits, they often use similar
15 techniques to what I was talking about with mortality,
16 where you look at total counts of the number of people
17 who go in for, who are admitted to the hospital for
18 cardiac disease or for respiratory disease, and see if
19 that's associated with daily or multi-day averages of
20 PM 10, after controlling for many other factors that
21 might also relate to hospital admissions.

22 So much for the short-term exposure studies.
23 Now on to the long-term exposure studies. Several
24 studies have looked at the effects of long-term
25 average, and here this could mean anything from one or

1 two years of average PM 10 exposure to seven to ten
2 years of PM 10 exposure. The different studies use
3 different amounts, and it considered PM 10 sulfates
4 and PM 2.5 in these analyses, and there's four studies
5 to date.

6 There's the Harvard Six Studies that was
7 published in '83 -- I think that was in the New
8 England Journal of Medicine, the first of the articles
9 to show relationships between long-term exposure to
10 particles and the likelihood of survival. Then there
11 is the cohort study using the American Cancer Society,
12 cohort, by Polk, et al., that looked at 500,000 people
13 in 150 cities throughout the US, following those
14 people for up to seven years. The Icelag study and
15 the Adventist Health Study of Smog, which is a study
16 of Seventh Day Adventists, I think mostly are all in
17 California. And then recently there has been a study
18 sponsored by the Electric Power Research Institute of
19 a Veterans cohort of 30 centers with about 20,000
20 people in the United States.

21 And these so-called perspective cohort
22 studies, unlike the previous studies I was describing,
23 follow people, follow individuals, as opposed to
24 looking at total death rates in a city. So they're
25 very powerful in that they can control for individual

1 level factors like smoking and weight and alcohol,
2 occupational exposure, gender and age, a whole host of
3 other individual level factors. And then, after all
4 the individual other risk factors are taken into
5 account, can look at whether differences in long-term
6 air pollution affect survival rates and survivability
7 in these cohorts.

8 People are followed from seven to 15 years in
9 these studies, and, as I mentioned, the largest one
10 uses 500,000 individuals that were followed for the
11 seven years in over 150 cities. Then several of these
12 studies report associations between longer-term
13 exposure to PM 10, PM 2.5 and/or sulfates, and either
14 life expectancy or survival, depending on how you look
15 at it. As opposed to the roughly one-percent change
16 in mortality per ten micrograms that you get from the
17 short-term studies, these studies show much stronger
18 effects of roughly a four-to-seven-percent change in
19 mortality per ten micrograms. As you might expect, it
20 is indeed a longer-term exposure process, possibly
21 pushing people into the risk pool but certainly
22 pushing people from the risk pool into earlier
23 mortality than otherwise.

24 And the studies, the ACS study, the Cancer
25 Society cohort study show that if you compare the

1 least and the most polluted cities and their cohort,
2 which is around a 25-microgram-per-cubic-meter
3 difference in PM 2.5, or roughly 50 micrograms of PM
4 10, the difference in the life expectancy for the
5 entire communities, after controlling for individual
6 factors, was somewhere between one and one and a half
7 years. If you apply the results to a life table and
8 do the analysis, look at the relative risks and then
9 crank it out for what it means in terms of survival,
10 the community as a whole in the more polluted area
11 would have about a one-to-one-and-a-half-year less
12 life expectancy. So these are effects that are quite
13 significant.

14 Now, since that original Harvard Six-City
15 Study and the American Cancer Society cohort studies
16 were published in the mid-'90's, again the Health
17 Effects Institute sponsored a multi-million-dollar
18 reanalysis and validation of those two studies, since
19 the implications of those two studies were so
20 important. So they basically had researchers start
21 from scratch, reconstructing the entire database,
22 reconstructing the analysis, and then going ahead and
23 doing a whole range of very detailed sensitivity
24 analyses.

25 And initially they totally replicated the

1 initial results, so the initial results held up under
2 a whole new data collection paradigm, and the
3 sensitivity analysis that they conducted, and there's
4 about a 350-page, four-columns or two-columns-per-page
5 document with very small print that you can go blind
6 trying to read. After conducting a wide range of very
7 detailed sensitivity analyses, the associations
8 between different measures of PM and mortality were
9 confirmed.

10 And I don't want to go through all of these
11 in detail, but just to give you an idea of some of the
12 things that were considered in these long-term
13 exposure studies was alternative statistical models, a
14 whole range of individual level variables, things like
15 physical activity and looking at smoking in lots of
16 different ways, and marital status and so on, a wide
17 range of individual level variables.

18 They also looked at ecological -- that is,
19 city-wide variables that might be not captured by some
20 of the individual level factors; things like weather
21 patterns and income in the area, number of hospitals
22 and water hardness, population growth, a whole range
23 of other ecological variables. Looked at non-linear
24 specifications in the data to try to see if there was
25 a threshold in the data, which they did not find.

1 Looked at a wide range of co-pollutants, different PM
2 metrics using different years. Measures to
3 incorporate underlying variation from city to city,
4 and also measures to look at potential for spatial
5 clustering of the cities. So there was a wide range
6 of sensitivity analyses that were connected in this
7 test. And again, the basic conclusions were upheld.

8 And finally, regarding the long-term studies,
9 recently at the International Society of Environmental
10 Epidemiology meetings in Germany, Arden Pope, who was
11 the lead author on the previous study, presented their
12 most recent results, and these are non-published, so I
13 normally wouldn't cover them, we didn't really cover
14 non-published articles in our document. But given the
15 significance of these studies, I thought it would be
16 interesting to at least see what the updated analysis
17 showed.

18 This analysis doubled the follow-up period,
19 from seven years to 16 years, so they had another
20 bunch of years to look at additional mortality in the
21 different cohorts. They also added extensive controls
22 for smoking, occupational exposure. They went back
23 and incorporated dietary factors and looked at fat
24 intake and a whole range of other factors. And they
25 found that the results were generally similar to the

1 results of the previous analysis.

2 And they also, in a subsequent paper by
3 Burnett, who has developed this new technique to look
4 at spatial correlations -- that is, that there might
5 be clustering of cities that might have separate
6 effects and that cities that are in different regions
7 might have different effects, and after controlling
8 for a lot of those types of spatial relationships, the
9 associations were also apparent.

10 So the results seem to be very robust in
11 different sensitivity analyses, and to basically doing
12 the study all over again, and then again expanding the
13 study to 16 years of follow-up.

14 Other notable results for both the short- and
15 long-term, and I'm going to just briefly go through
16 these and this afternoon I'm sure there will be
17 comments on some of these things and we can discuss
18 them in greater detail, but I at least wanted to point
19 out there as part of the weight of evidence, there are
20 I think nine available time-series studies that look
21 at fine particles, particles below 2.5 microns, and,
22 of course, particles that are between 2.5 and 10
23 microns.

24 And typically, one measure or the other is
25 statistically significant. Sometimes both are

1 statistically significant or show associations with
2 mortality. But our evaluation, and I think I have a
3 table subsequent to this, show mixed results. That
4 is, sometimes the PM 2.5 showed stronger effect,
5 sometimes the coarse particle showed stronger effect,
6 sometimes the effects were equally strong, so it was
7 really hard to say that one constituent was a lot more
8 toxic than the other. But certainly, there are some
9 short-term studies showing effects specifically from
10 PM 2.5 and from coarse particles.

11 A second point is that most of the analyses,
12 when they've looked at trying to detect a threshold in
13 the response, have failed to detect such -- that is, a
14 no-effects level. It's not been found in these
15 studies.

16 And we thought of three different ways, at
17 least, that people could infer or not infer
18 thresholds. One is if you conduct studies at very low
19 levels and you find effects, that indicates certainly
20 that effects are going down to those low levels. And
21 in our report, in the 64 or 67 cities, we did include
22 a lot of cities that had very low concentrations.
23 Most of those cities are in Northern Europe; Sydney,
24 Australia; Vancouver. And many of those studies do
25 find effects and associations.

1 Another way to infer something about the
2 threshold is using statistical approaches using
3 flexible models, really allowing the data to drive the
4 shape of the just response or concentration response
5 function. And typically, those approaches indicate a
6 lack of the threshold of continuum of responses.

7 And sometimes smoothing approaches have been
8 used, where you basically look at weighted averages of
9 the data and see what the shapes are of that relating
10 to daily mortality. And again, both for the short-
11 term exposures and for the long-term exposures,
12 there's no clear threshold that's been indicated from
13 these studies. There have been one or two studies
14 published that do seem to suggest or have found a
15 threshold using their analyses. There was a paper
16 done in Phoenix that showed a threshold at around 20
17 micrograms at PM 2.5, but most others who have looked
18 at the data have failed to find one.

19 Here is a chart that's also in the document,
20 the finding of coarse particle associations. The
21 coarse particles are the bigger symbols here, and I
22 don't know how anybody can read this, but confidence
23 interval around each -- this is I think table 7.4 of
24 the document. Take a look at it more carefully there,
25 if you want. What we're trying to show here is that

1 associations were found for both, but again, sometimes
2 the fine particle shows stronger association and
3 actually sometimes the coarse particle shows a
4 stronger association.

5 So we've indicated that for the short-term
6 mortality studies, it wasn't easy to pick out a
7 specific fine particle effect. Both types of particle
8 sizes seemed to have associations. That seems to be
9 in contradiction to the long-term studies. Most of
10 the long-term studies appear to show stronger effects
11 from the PM 2.5 relative to PM 10. In the ACS cohort,
12 the effects are dominantly from fine particle
13 exposures.

14 PROFESSOR FRIEDLANDER: So what did you
15 conclude from that last figure?

16 DR. OSTRO: Okay. What we concluded is
17 that --

18 PROFESSOR FRIEDLANDER: I mean, is the fine
19 more frequently or is it equal?

20 DR. OSTRO: We thought it was about equal. I
21 mean, we couldn't say that you would -- from the
22 available data, that you would only be worried about
23 fine particles. We thought there was enough evidence
24 for a coarse particle effect, even in areas where you
25 didn't think coarse particles would dominate, that we

1 would rule out a coarse particle effect, but that
2 there was enough concern for some fine particle effect
3 as well.

4 So when you look at it in general, it does
5 seem on a per-microgram basis, the fine particles are
6 more toxic, and you might expect that because fine
7 particles will penetrate more easily into the lung,
8 into deep parts of the lung, and also penetrate more
9 easily into homes. So on a per-microgram basis, it
10 does look like fine particles in general are a little
11 bit more important than coarse. But we wouldn't rule
12 out a coarse-particle effect.

13 CHAIRMAN KLEINMAN: Bart, these fine particle
14 studies, are these studies that specifically measured
15 PM 2.5 or are surrogate measures in there?

16 DR. OSTRO: These are ones that specifically
17 measured 2.5. I'm looking at them quickly. I think
18 they all measured specifically 2.5, and they measured
19 coarse particles just by differencing. No one had
20 specific coarse particle --

21 PROFESSOR SIOUTAS: Well, that was part of my
22 next point I was going to ask you, thanks for
23 answering, so the measure of coarse concentration here
24 is by differencing.

25 DR. OSTRO: Just by differencing.

1 PROFESSOR SIOUTAS: So it could be subjected
2 to pretty large --

3 DR. OSTRO: It could be, depending upon the
4 monitors that were used, right.

5 This is just an example of a threshold
6 analysis using a smooth. This is from Schwartz &
7 Zanobetti, looking from ten US cities where they
8 looked at the results across all the ten cities and
9 then allowed the data to tell them what did the dose
10 response look like over the particle concentration.
11 And again, you don't see much of a threshold in this
12 type of study.

13 Likewise, these are from the long-term cohort
14 studies. This is from the Krewski reanalysis of the
15 American Cancer Society. This is the HEI-sponsored
16 study, and for fine particles, I think they only have
17 about 50 cities that actually had fine-particle data,
18 and the dots are in the data points of fine particles,
19 and a residualized measure of mortality. This is
20 mortality after other factors have been factored out.
21 All the individual level factors have been taken care
22 of.

23 And again, they show a dose response, and
24 when they did a test for linearity, what they said was
25 it was basically fairly near linear in their response.

1 This is cardiovascular, cardiopulmonary, and this is
2 all-cause mortality.

3 But what we wanted to highlight as well was
4 not only a near-linear association, but that at the
5 lower level, around 12 or 13, the scatter gets greater
6 and the uncertainty gets greater. So the
7 extrapolation down to the lower levels of coarse gets
8 a little bit more uncertain.

9 PROFESSOR THURSTON: Can I just, as long as
10 you have this up, just make one comment? Because
11 there were in the written comments a couple of people
12 who pointed to this figure, which I guess this is from
13 figure six in the Krewski report, and said that this
14 showed a poor fit of the model, because I think that
15 they were assuming that PM was already in this model.
16 But these are the residuals, not including PM, and
17 then seeing how much PM explains of the remaining
18 variation.

19 So I think there was a misunderstanding by
20 the public from the Krewski paper. So they thought
21 the PM was already in the model, and that gee, look,
22 it's tilted, it's not a flat line, so it's a poor fit.
23 But actually, what it's showing is, is that after
24 controlling for everything else, there still is,
25 there's a slope there, there's an association between

1 remaining unexplained mortality and pollution.

2 DR. OSTRO: Yeah. Well, we'll wait a little
3 bit to get into that and everything later on this
4 afternoon.

5 PROFESSOR THURSTON: Okay.

6 DR. OSTRO: Okay. Other notable results, the
7 mortality displacement or what some people call
8 harvesting appears minor. By that we mean there's an
9 issue of are these people who are affected by
10 particles, is the prematurity only a matter of a day
11 or two, or is it of more significant importance, more
12 significant days? And the studies that have been
13 conducted to date on looking at this phenomenon of
14 mortality displacement indicate that for the
15 cardiovascular mortality, the reduction, the amount of
16 prematurity appears significant.

17 One study on the cardiovascular mortality and
18 points showed that it's at least two months and
19 probably longer, in terms of how many days brought
20 forward. And the chronic studies indicate, as I've
21 indicated a much greater effect, in terms of life
22 shortening. So mortality displacement -- that is, the
23 short-term consequence in terms of prematurity --
24 appears minor. The cases appear to be bringing out
25 lots of reduction.

1 The second is that there have been only a few
2 but there are some composition-specific studies. So
3 rather than looking at a specific component like PM
4 2.5 or PM 10 or sulfates, a couple of studies have
5 tried to look at markers and use principal components
6 to try to say what are the effects of sources in
7 general. So they've looked at mobile sources or
8 combustion sources or stationary sources and crustal
9 sources and so on. And the preliminary efforts to
10 date suggest that the combustion-related particles are
11 the most toxic, based on the few studies that have
12 been done.

13 And at PM 10 might be a broader surrogate for
14 combustion particles in general. So when you're
15 controlling some of these sources you'll be
16 controlling PM and be controlling, reducing or having
17 effects on some of these health outcomes that we're
18 talking about, but you also might be controlling some
19 other factors that might have additional effects on
20 health.

21 Another important thing is that there has
22 been very careful control for weather and for other
23 potential confounders in these models. And just to
24 give one example, and I don't want to spend too much
25 time on this, we might discuss this later, but things

1 like linear or smooth variables have been included,
2 looking at temperature, humidity and dewpoint. More
3 importantly, in a nice paper that actually George did
4 publish this year, I guess, or last year, extremes in
5 the weather sometimes are far more important than
6 looking at weather on a day-to-day basis.

7 But it's really the very hot and very cold
8 days that really relate to mortality and
9 hospitalization. And it's more important to look at
10 the extremes, so a lot of studies will look at that.
11 And, of course, the influence of longer-term seasonal
12 cycles need to be taken into account, and most of
13 these studies do that.

14 Okay, we'll stop here. Based on the
15 suggestions of the chair, we'll now go to the
16 monitoring and then we'll come back I think to the
17 rationale.

18 MR. COOK: Thank you. Dr. Kleinman, esteemed
19 members of the Air Quality Advisory Committee, we're
20 pleased to bring before you for your review the
21 methods portion of the proposed PM standard. My name
22 is Jeff Cook and I'm the chief of the Quality
23 Management branch at the Air Resources Board. And I'd
24 like to also introduce my colleague, Cliff Popejoy,
25 and he and I together are responsible for the

1 monitoring portion of the staff report for the PM
2 standard review, and the recommendations contained in
3 that.

4 We're here today to provide you with some
5 history of the current statement that's for measuring
6 ambient PM, how it relates to federal methods, and to
7 review our proposal for updating a statement that's in
8 a rather significant way.

9 As you are all aware, state regulators are
10 keenly interested in the measurement method that is
11 used to determine ambient concentrations for
12 comparison to ambient air quality standards. In the
13 case of PM, the choice of the sampler type can
14 influence the particulate that is measured, and
15 ultimately, the definition of the parameter itself.
16 We are proposing a suite of standardized methods for
17 the state ambient air quality standards for PM that
18 will ensure consistency and accuracy in regulatory
19 monitoring that becomes the basis for annual
20 determinations made by the board regarding what areas
21 attain and what areas don't attain the ambient air
22 quality standards.

23 In doing so, we need to establish networks
24 that use reliable measurement techniques. This is
25 going to be an interesting task, given the significant

1 number of PM 10 and PM 2.5 samplers already in
2 California networks. At the same time, we're most
3 interested in opening the door for other instruments,
4 particularly continuous PM instruments that provide
5 accurate real-time data in short averaging time.

6 It will come as no surprise, I'm sure, to
7 anyone on this panel that the perfect instrument does
8 simply not exist, or, if it did, it's simply not
9 practical for large-scale network deployment.

10 Fortunately, PM methods have been evaluated in a
11 number of studies in California over the years, and we
12 have become familiar with the pluses and minuses of
13 some of the available instruments. We're also
14 fortunate to have several PM supersites in California
15 that are working on identifying instruments for this
16 very same potential use.

17 Staff had a very limited selection of sampler
18 types to consider as the official state method when
19 the first PM standard method was adopted in 1986. The
20 high volume PM 10 sampler methodology approved by the
21 board wasn't even mature at that time, and went
22 through at least two subsequent iterations to arrive
23 at today's workhorse sampler that we refer to as the
24 SSI. Part of the method approved by the board allowed
25 the executive officer to approve new generation

1 samplers that yielded equivalent results to the state
2 method.

3 The TSP network in place for particulate
4 matter measurements prior to the adoption of the PM 10
5 standard was greatly de-emphasized at that time,
6 leaving the sulfate and lead standards as the sole
7 reasons for maintaining the TSP sampling network. For
8 a short while we did use the TSP samplers for some
9 aspect of our air toxics network; however, that has
10 been shifted to another sampler.

11 One of the advances with the new PM high-
12 volume samplers over the TSP devices was the
13 requirement to use low-alkalinity filters. Because
14 there would no longer be a TSP standard and there is
15 no federal ambient standard for sulfate, there was
16 little reason to address sulfate on the TSP filters.

17 At the present time, the SSI sampler filters
18 are analyzed principally for mass, but are also
19 analyzed at selected sites for the dominant cations
20 (phonetic) and anions (phonetic) that make PM 10. The
21 low alkalinity filters in use for the PM 10
22 measurements virtually eliminated the possible sulfate
23 artifact that existed prior.

24 Nitrate loss is still an issue, however.

25 Activities that reduce the time to analysis have been

1 incorporated into the Air Resources Board's network.
2 Extractions are made soon after filters arrive, and
3 there has been an effort to reduce the time from the
4 conclusion of sampling until the filter is included in
5 the equilibration chamber.

6 Handling extremely heavily loaded filters
7 from dusty windy environments is another challenge for
8 any integrated filter-based measurement. Beyond that,
9 the labor-intensive nature of filter-based networks
10 affects directly the sampling schedule state and local
11 agencies are able to maintain. So there are some
12 clear disadvantages to the method and the single
13 method that we have for the state PM method.

14 We're addressing these shortcomings in part
15 by incorporating more samplers that work in California
16 with an eye again to including those that minimize the
17 loss of important PM constituents and reduce the labor
18 required per mass determination.

19 The staff's proposal is to incorporate by
20 reference the current federal reference methods as
21 acceptable samplers for the state ambient air quality
22 standards for PM. Quite a few federally recognized
23 but non-state-approved samplers exist in the state now
24 to satisfy federal SIP planning and monitoring
25 requirements. And because of the federal program,

1 most of the attention has been drawn towards federally
2 approved samplers. The federal samplers have
3 consistency in measurement principals, and have been
4 evaluated in extensive field and bench tests. These
5 include capture efficiencies, filter composition, pore
6 size, inlet design and flow characteristics, to
7 mention a few.

8 The samplers are capable of maintaining
9 constant and accurate flow, an invaluable aspect of
10 the sampler; record the time of operation and the flow
11 rates. As federal methods, they're governed by
12 detailed monitoring regulations and guidelines for
13 sampler and laboratory operation.

14 What we are not proposing to adopt, however,
15 are federal equivalent methods carte blanche. This is
16 where you find the federally approved continuous
17 samplers. Our primary reason for this is that
18 equivalent samplers were approved on the basis of
19 field performance and few were tested in California
20 before they were granted that designation. Some have
21 proven to be troublesome for the state.

22 To begin with, we're proposing to adopt the
23 PM 10 and PM 2.5 filter-based federal reference
24 methods as acceptable for all regulatory purposes.
25 The PM 10 devices are both high-volume and low-volume

1 samplers. The PM 2.5 is low-volume only. Any
2 federally recognized sample inlet will be likewise
3 approved with its appropriate sampler.

4 An important point I'd like to stress at this
5 time, for reasons mentioned a moment ago, is that
6 federally approved continuous samplers will not be
7 similarly incorporated by reference. Without further
8 evaluation, they must be demonstrated to work in
9 California.

10 We're planning to include continuous samplers
11 in the final report, however, but that action will be
12 based on the results of a study that is underway and
13 wrapping up in Bakersfield at the end of this month.
14 We're proposed to retain the equivalency provision in
15 the regulation to encourage the continued development
16 of new samplers. And lastly, to shift from TSP to PM
17 10, the sampler of choice for sulfate.

18 We anticipate this action will be welcomed by
19 state and local regulatory agencies who must now
20 grapple with two sets of requirements when making
21 sampler purchases, and knowing that to virtually tag
22 some samplers as good for some purposes and others for
23 other uses. We hope to not only close the gap between
24 the approved state and federal sampler technologies,
25 but to approve continuous PM 10 and PM 2.5 samplers

1 for compliance purposes.

2 At the present time, the US EPA has not
3 approved a continuous PM 2.5 sampler. We're hopeful
4 that they will see our proposal as an endorsement of
5 the samplers that work in California, and move to
6 accept our continuous PM 2.5 samplers in their current
7 deliberations.

8 A few words about the study that is drawing
9 to a close in Bakersfield. First, we're evaluating
10 four instruments that are in general use and that hold
11 promise in California. Some include very recent
12 upgrades. The instruments are being watched carefully
13 by our staff, who operate the devices according to the
14 vendors' desires. We are approaching this study with
15 the idea to optimize the chances for success within
16 the expectations of normal operations.

17 Instrument vendors were allowed time to set
18 up their instruments and to train our staff and be
19 fully satisfied before they left the station. They
20 have been able to fix serious problems when they
21 occur, and only one of those has occurred, and no
22 vendor has had access to the station or any of the
23 samplers' data since the study began in October 2001.
24 Calibrations and audits have been performed in
25 accordance with the protocol drafted by ARB staff and

1 agreed to by the vendors. By the way, we are very
2 pleased with the way this study is proceeding.

3 Bakersfield in the winter, as you probably
4 know, poses difficult environmental conditions for a
5 sampler. This area was selected for that reason, in
6 that it represents well the atmospheric soup in areas
7 of California that have the most persistent and
8 difficult PM problems. Lastly, all of the vendors
9 have agreed to equip their samplers with the sharp cut
10 and lead cyclone.

11 We're focusing on the latest generation
12 instruments or software upgrades to familiar systems.
13 The samplers we're evaluating are all paired. They
14 produce hourly average PM measurements directly in
15 micrograms per cubic meter. They are, for PM 10, the
16 federal equivalent method, Met One Beta Attenuation
17 Unit; the federal equivalent method, Anderson Beta
18 Attenuation Unit that operates on a slightly different
19 principle; the Rupprecht-Patashnik TEOM with the
20 Sample Equilibration System and Filter Dynamic
21 Measurement System. This is a new add-on to their
22 TEOM that we're looking at now.

23 And lastly, for PM 10, the Anderson
24 Continuous Ambient Monitoring device referred to as
25 the CAM. These will be compared against both the

1 Anderson SSI, the high-volume sampler, and the
2 Rupprecht-Patashnik Partisol, a low-volume PM 10
3 sampler, both of which are also paired.

4 The PM 2.5 samplers include virtually the
5 same types of designs, the same instruments with only
6 the PM 2.5 head: the Met One Beta Attenuation Unit,
7 the Anderson Beta Attenuation Unit, the Rupprecht-
8 Patashnik TEOM, the SES, FDMS, and the Anderson CAM
9 unit. Those will be compared against paired PM 2.5
10 FRM samplers. Those data will be available in late
11 February.

12 The two reasons regulatory agencies have for
13 operating TSP samplers are for compliance with the
14 state sulfate ambient air quality standards, and the
15 state federal ambient air quality standard. With
16 adoption of the size-segregated PM standard, the
17 decrease in sulfate concentrations, and the dramatic
18 decrease in sulfur dioxide concentrations, and the
19 shift from community-oriented to source-oriented
20 monitoring for lead, the number of TSP samplers has
21 markedly decreased over the years.

22 The sulfate network in California exists
23 primarily in the South Coast air basin. Replacing the
24 TSP samplers with the SSI for sulfate measurements
25 would not only permit further downsizing of the TSP

1 network, but at the same time incorporate a much
2 larger PM 10 SSI network as the potential devices for
3 sulfate monitoring.

4 There are several issues that we wanted to
5 talk about just briefly that actually represents more
6 or less a work in progress, and that is, in looking
7 back at the historic sampling device for sulfate, the
8 TSP sampler, that it has a known artifact to it.
9 Depending on the alkalinity of the filter, it can
10 range from one to eight micrograms per cubic meter.
11 The factors that have been cited to affect this
12 include things like the sulfur dioxide concentrations,
13 the relative humidity, the flow rate of the sampler,
14 as well as the alkalinity of the filter.

15 The SSI sampler, which uses a quartz filter,
16 has controlled alkalinity on the filter which is
17 controlled by regulation, and that should reduce and
18 eliminate the artifact. So what we're grappling with
19 now is looking back at the information that was relied
20 upon to set the original standard and is that
21 information specific enough that we should be
22 considering modifying the level to account for that
23 artifact, or is that basically within the noise of the
24 decision.

25 PROFESSOR FRIEDLANDER: But are we going to

1 stay with the filter system, are you verifying that?
2 Because I've always thought that there's, particularly
3 in an area like Southern California where we have very
4 strongly oxidizing atmospheres and we have hydrogen
5 peroxide, that the peroxide should react with the SO₂
6 in the aerosol that's deposited in the filter and lead
7 to a positive artifact.

8 MR. COOK: On the quartz fiber filter, as
9 well as the --

10 PROFESSOR FRIEDLANDER: Excuse me?

11 MR. COOK: On the quarter fiber filter, as
12 well as the glass filter?

13 PROFESSOR FRIEDLANDER: If you have sulfates
14 already deposited, I think there will be water there,
15 and peroxide and SO₂ can dissolve in it, yeah.

16 MR. COOK: I think one of the bigger
17 questions -- I agree with what you're saying, I think
18 the big question we're grappling with is, is the
19 information that was presented to the staff at the
20 time they made that nominal determination of 25
21 micrograms per cubic meter, and how specific was that,
22 and is it sensitive enough that this artifact needs to
23 be looked at closer and possibly accounted for in that
24 determination, or in the determination to go to PM 10
25 as the filter of choice.

1 PROFESSOR FRIEDLANDER: Mr. Chairman, will we
2 have time to revisit this this afternoon, these --

3 CHAIRMAN KLEINMAN: Well, I think this is an
4 important issue, especially since there is a
5 recommendation on the table for a PM -- rather, a
6 sulfate standard.

7 Jeff, can you give us sort of a ballpark idea
8 of what you think the magnitude of this artifact might
9 be? Are we talking a few micrograms, are we talking
10 more than that?

11 MR. COOK: The data that I've looked at for
12 California over the years is rather interesting,
13 because the artifact is not consistent throughout the
14 state. As a matter of fact, the agreement between PM
15 10 and TSP sulfate in some portions of the South Coast
16 air basin is actually very good. When you go out
17 towards the western portion of the basin, from the
18 sites that we have available to us -- For example, out
19 in Hawthorne -- is where you start to see some of the
20 disparity grow. And we're looking at numbers on the
21 order of maybe four micrograms per cubic meter.

22 The other places that we've seen the
23 disparity is in the Bay Area itself, and in places
24 like San Francisco, Richmond, San Jose, where we see
25 that disparity about the same order of magnitude,

1 about four micrograms. There has been a lot of
2 discussion about where that comes from, is that
3 actually an artifact or is that actually a larger
4 sulfate particle generated either by the sea spray or
5 by some other -- we just, we're not -- But it's about
6 four, something on that order, three to four.

7 PROFESSOR FRIEDLANDER: Yeah, Professor
8 Sioutas just pointed out that in San Francisco, where
9 you have relatively high humidity, that that would be
10 a possible place where you would expect a high water
11 content in these filters, and they're possible sites
12 for the sulfate reactions as a result of SO₂ and
13 peroxide from the gas phase, dissolving and reacting.

14 MR. COOK: And would those tend to form on
15 that, on the glass fiber filter more readily than the
16 others, because --

17 PROFESSOR FRIEDLANDER: If you have water
18 there.

19 MR. COOK: -- we're seeing a difference in
20 this area, which is the one thing that's a little
21 interesting to us.

22 The other interesting thing is --

23 PROFESSOR FRIEDLANDER: I mentioned this in
24 the comments that I sent to Dave Mazzer.

25 Is he here? I guess he's out.

1 DR. MAZZERA: Yes, I'm here.

2 PROFESSOR FRIEDLANDER: Yeah, when you --
3 This is discussed in the comments that I sent to you.

4 CHAIRMAN KLEINMAN: Yeah, I think we'll go
5 into this in more detail this afternoon.

6 DR. BALMES: Could I add one element to this?
7 This is actually, the issue of the artifact is
8 actually something that was kind of raised by
9 monitoring people after the first recommendations were
10 put out in the draft report. We went back and we
11 looked at the data that was used to set the original
12 sulfate standard of 25 micrograms back in 1976. At
13 that time they weren't aware of the artifact in that
14 original 1976 document, that that standard was set on
15 the basis of -- basically, respiratory irritation from
16 studies seen in animals, from sulfate aerosols.

17 It was set looking at actual occupational
18 settings of human exposures at about 350 micrograms
19 per cubic meter studies. And then using a safety
20 factor to get down to the 25-microgram level. And
21 then it was also used, epidemiological data that
22 basically had seen some effects in that same kind of
23 area.

24 The Air Board, at the time the Public Health
25 Services and at that time the Air Quality Advisory

1 Committee had recommended the 25-microgram level, and
2 at that point it was set so that the artifact actually
3 was not, did not enter into the original setting of
4 that 25-microgram standard. And in subsequent reviews
5 in 1977, they again reviewed the sulfate standard,
6 retained the standard at the current 25-microgram
7 level, and at that time they had had their first study
8 of monitoring methods and had identified that there
9 was an artifact problem that depended on sulfate
10 concentration and the alkalinity of the glass filters.
11 But they still retained the 25-microgram level of the
12 standard, so just some background.

13 So basically, in our recommendations, we
14 believe that moving to the PM 10 sulfate standard
15 actually gets back to what the original data was, and
16 that was to look at basically sub-micron-sized
17 particles in aerosols that was the basis for that
18 original standard. Moving to the PM 10 monitoring
19 method also increases the areas that are covered and
20 will give a much better understanding of sulfate
21 concentrations around the state.

22 PROFESSOR SIOUTAS: I also want to point out
23 that there is now a lot of published and commercially
24 available continuous sulfate monitors that our
25 supersite has shown excellent agreement with the

1 teflon-based sulfate. So this is something you may
2 want to consider. And these continuous monitors would
3 not suffer from those artifact problems, so I implore
4 you to consider those too.

5 CHAIRMAN KLEINMAN: Okay. Is that
6 information available, Costas, so we can --

7 PROFESSOR SIOUTAS: Yes.

8 CHAIRMAN KLEINMAN: It's a published report?

9 PROFESSOR SIOUTAS: And I will provide it,
10 sure.

11 CHAIRMAN KLEINMAN: Okay. That's good to
12 have.

13 MR. COOK: It's evident that the number of PM
14 samplers available today has grown substantially since
15 the board adopted the ambient air quality standard and
16 the method back in 1986. We're confident all would be
17 well served simply by eliminating as much confusion as
18 possible between the samplers that are usable for
19 federal versus state activities.

20 We would also like to adopt continuous
21 methods that I've mentioned before. In so doing, we'd
22 be making a statement about some of the federally
23 approved samplers that do not work in California.
24 We're optimistic that the latest version of the
25 samplers we are evaluating will give us the ability to

1 do just that. As mentioned earlier, we will work with
2 the US EPA in hopes it will accept the results of this
3 study as it moves ahead in its desire to approve
4 continuous PM 2.5 samplers.

5 The state's proposal fills several needs.
6 One is the long-overdue action to recognize federal
7 reference PM 10 methods for use within the state and
8 for the state standards. Another is the timely
9 decision to recognize the federal reference method for
10 PM 2.5. And lastly, the step forward to adopt
11 continuous samplers for work in California.

12 We look forward to sharing the results of the
13 Bakersfield study with you and the rest of those
14 interested in continuous ambient PM 10 monitoring.

15 PROFESSOR FRIEDLANDER: When you refer to the
16 continuous monitor, do you mean the impactor-based
17 standards, where you collect the -- flash vaporization
18 technique, or what method are you referring to for the
19 continuous?

20 MR. COOK: The methods that we're looking at
21 now are basically beta attenuation, two different
22 versions of beta attenuation. One uses, one collects
23 samples for 50 minutes and does its analysis and then
24 advances the tape. The other collects it over a 24-
25 hour period, making continuous measurements.

1 PROFESSOR FRIEDLANDER: Are they both filter-
2 based?

3 MR. COOK: Those are both, they are both
4 filter-based. It's a roll of tape. It actually looks
5 like a modern version of the old AISI tape sampler
6 with -- using beta rays rather than using light
7 transmission.

8 The other is the CAM unit, which operates on
9 a principle of pressure drop as particles build up
10 again on a filter. I think that's a teflon filter.
11 And then the last unit is an elaborate unit that
12 Rupprecht-Patashnik has adapted from their TEOM, which
13 is the -- which probably you're familiar with, it's
14 the oscillating microbalance that uses a small flow at
15 about three liters per minute. And the change in the
16 oscillation is attributed to the increase in mass on
17 that filter.

18 The problems with that sampler in California,
19 and that's been seen in study after study, is that the
20 temperature that they operate that microbalance on, in
21 order to eliminate the effect of moisture gain on that
22 oscillation tends to drive off the volatiles. And
23 they recognize that and they've tried in a number of
24 instances to modify that, decreasing the temperature.

25 And now what they've done is they've added

1 both a dryer system that they actually pass the air
2 through several times, and kind of a switching system
3 where they can sequentially measure heated and non-
4 heated in order to try to preserve the volatiles. So
5 that's the latest instrument that we're looking at.

6 The one point I would like to make, however,
7 is that we do open -- we are preserving this operation
8 called equivalency. And it exists in the state
9 regulations now and it will exist in the future. And
10 that's the door that we want to leave wide open to
11 samplers that we are not looking at now, samplers that
12 are at supersites. We're just blessed to have these
13 supersites in the state, and I think those will
14 provide very valuable information to supplement what
15 it is that we'll be proposing. Thank you.

16 DR. OSTRO: Okay. So on to the
17 recommendations part. So the general rationale for
18 the new recommendations, new standards is that our PM
19 10 standard was introduced in 1983, and, as we've
20 indicated, since that time there have been hundreds of
21 published studies confirming associations between PM
22 10 and PM 2.5 with mortality and many other adverse
23 outcomes. And recent studies also suggest effects
24 from both fine and coarse particles.

25 So now to talk specifically about the annual

1 average standards, when we looked at the magnitude of
2 the effects from the short-term exposure studies and
3 the long-term exposure studies, we found that, as I
4 reported, that the greatest impact is from the long-
5 term exposure studies. Depending upon which studies
6 you use, the impact could be two to four times higher
7 from the long-term exposure. So clearly, the longer-
8 term averages play an important role.

9 Our primary focus is on reducing the entire
10 PM distribution and the long-term exposure by lowering
11 the annual PM 10 average and the annual PM 2.5
12 average. In lowering the annual average, of course,
13 the entire distribution will fall, and the likelihood
14 of extreme concentrations would also fall.

15 We also thought it was important to have an
16 annual average for PM 2.5, as we discussed in the
17 document, and because of the long-term studies that
18 have been specifically shown to be associated with PM
19 2.5. That has different sources, different lung
20 deposition and different penetration from the outside
21 indoors. And again, quite significant effects,
22 specifically from long-term averages for PM 2.5

23 Now, in terms of how we developed the annual
24 average of 20 as a recommendation, we looked at all
25 the different types of studies that have implications

1 for long-term exposures. Specifically, for example,
2 if we looked at the ACS cohort which has a range in
3 terms of PM 10 from about 18 to 60, and the Harvard
4 Six-City Study which had a range of 18 to 46, the
5 averages for those studies are around 28 micrograms
6 per cubic meter. As we indicated, though, there
7 certainly is some likelihood of effect below those
8 levels.

9 Then we also talked about when you look at
10 the long-term average, the long-term means of those
11 daily exposure studies, relatively acute studies, the
12 daily means range from 15 all the way up from 70.
13 But, as we indicated in the document, things get a
14 little bit more uncertain lower than 25. And equally
15 important, most of the studies conducted at the lower
16 end may be less similar to situations in California,
17 conditions like in Northern Europe where these studies
18 have been undertaken, and they don't typically have
19 the same types of setups that we have here.

20 So we're maybe slightly less confident in
21 extrapolating from those studies to situations in
22 California. But the means of those studies, the
23 ranges that we were really concerned about were the 25
24 to 35 micrograms per cubic meter.

25 And also, we have a few studies on the

1 effects of long-term exposure on morbidity.
2 Specifically, there were some studies published using
3 the Harvard Six-City Study, some Harvard 24-City
4 Studies, that show effects of one-year averages of
5 particles on various chronic outcomes in children;
6 things like bronchitis and chronic cough. And also in
7 California, the ARB has funded a long series of
8 studies on children's health in Southern California,
9 and those two are -- the range of those studies are
10 around 21 to 35. Children's health studies were also
11 finding effects on symptoms and a little bit on lung
12 function as well on the studies reported to date.

13 So when we put all these studies together, we
14 thought an annual average of 20 was providing a good
15 deal of health protection for the population in
16 California.

17 Likewise for PM 2.5, we looked at studies
18 that have implications for long-term averages. Here,
19 the studies go from 9 to 35 with a mean of around 18,
20 and likewise for the Six-City Harvard Study, a mean of
21 around 18. Again, no absolute clear threshold reduced
22 the effects probably down below those levels. And, as
23 I indicate, once you get to those lower levels,
24 alternatively become greater and fewer points down
25 there.

1 And also we looked at the studies of daily
2 exposures by looking at the long-term average of those
3 studies. Because you want to basically be below the
4 means of what those studies are showing, you want to
5 drop down below those distributions. And the range of
6 those studies, the means are around 13 to 18.

7 So we thought moving to a 12-micrograms-per-
8 cubic-meter for PM 2.5 would afford sufficient
9 protection for the public health. We're not claiming
10 it's a zero risk, and point out that there is some
11 possibility of effects lower than that, but we thought
12 that the scientific evidence pointed to a number as
13 this 12 micrograms per cubic meter for PM 2.5

14 Regarding the 24-hour standards, there was a
15 concern that some areas that will be attaining the
16 annual average or averages still might have episodic
17 elevations on a short-term basis. So, for that
18 reason, we decided to recommend retaining the current
19 24-hour average standard for PM 10 of 50 micrograms
20 per cubic meter with the belief that that, coupled
21 with a 20-microgram-per-cubic-meter annual average
22 would afford protection for both long-term exposure as
23 well as short-term exposures.

24 We do discuss in the document, though, that
25 it is difficult to disentangle the effects of long-

1 term exposures from short-term exposures. I mean, the
2 studies might be measured in 24 hours or many years,
3 but, in fact, people are exposed all the time. So
4 even though you're measuring short-term exposures and
5 you think there are effects from those short-term
6 exposures, but those are all superimposed on the
7 underlying amount of long-term exposures.

8 So there are no studies in which we have
9 background exposures and then all of a sudden we have
10 a 24-hour exposure and then background again, and then
11 we elicit an effect off all of the studies that we're
12 forced to rely on by nature to incorporate chronic
13 exposures that makes this more difficult to invoke a
14 short-term standard for PM 2.5.

15 So, consequently, because of the difficulty
16 in the bright line, even though we do think there are
17 effects from short-term exposures, in fact, well-
18 documented effects, at this time we do not recommend a
19 24-hour average. We think the combination of a 20-
20 microgram PM 10, 12 microgram 2.5 annual average and
21 the 50 average for PM 10 for 24 hours will drop down
22 the whole distribution and afford a good deal of
23 protection for both the PM 10 and the PM 2.5.

24 For sulfates, our assessment of the sulfate
25 evidence was that it's less consistent than that for

1 PM 10 and PM 2.5. When you review the studies as a
2 whole, some studies where you expect to see sulfate
3 effects stronger than PM 2.5 effects, and you don't
4 see them but then you see the reverse in some cases
5 where you wouldn't think sulfates were necessarily a
6 problem, you'd see a stronger sulfate effect.

7 We didn't feel that there was enough evidence
8 to really change what we have right now, and we
9 coupled that with information that sulfate
10 concentrations in California are far lower than the
11 current standard, and the strongly acidic sulfates
12 associated with health effects are relatively uncommon
13 in California. We've reduced a lot of our major SO2
14 sources a decade ago, so we don't have a lot of SO2
15 sources to worry about at this point.

16 Two final points. One is we were planning to
17 talk a little bit about mechanisms, but in the
18 interests of time we'll wait until this afternoon.
19 And Dr. Lipsett will then lead a discussion and hear
20 the comments from AQAC on mechanisms. So we won't
21 cover that this morning.

22 But we did want to mention that our risk
23 analysis of the data showed that there would be
24 significant health benefits from attaining the newer
25 standards, and just as an example, the details are in

1 the document, but as an example, suppose the change
2 from current levels to our 20-microgram-per-cubic-
3 meter annual average, if you apply the epidemiologic
4 evidence and look at that change from current levels
5 to 20 micrograms, it ultimately would result in about
6 6500 deaths per year, premature deaths per year that
7 then would be saved; 3100 cardiovascular
8 hospitalizations for those above age 65; and a
9 significant number of lower respiratory symptoms that
10 could be reduced.

11 And we also looked at other outcomes, like
12 asthma exacerbation and I forget the other ones, but a
13 couple of respiratory hospitalizations and so on. So
14 there's a whole range of benefits that we expect,
15 significant benefits that will be improved by
16 attaining a standard of 20 micrograms.

17 DR. SHERMAN: I know this is a long time ago,
18 in 1983, but do you have any information about all-
19 cause daily mortality prior to '83 and the institution
20 of the PM 10 30-microgram standard, and how that
21 affected all-cause mortality between time points
22 before and after?

23 DR. OSTRO: Unfortunately we don't have a lot
24 of data. Up until 1983 there had been relatively few
25 of these time-series studies. The primary source of

1 data at that time was a London data set that had
2 existed for 14 years. And that was measuring black
3 smoke. The studies at that time, if you try to make
4 some rough comparisons between black smoke in London
5 in the '70's and PM 10, you can -- some analysis has
6 shown that the effects are along the same continuum;
7 that is, the effects per microgram equivalent on
8 mortality appear to be about the same. So you're
9 looking at basically one long concentration of
10 response function.

11 But there wasn't a lot of studies on PM 10,
12 and I think there was actually only one study of PM 10
13 when we actually set the standard and even US EPA set
14 the standard. There was very little studies actually
15 using PM 10. It was based on TSP, plus our knowledge
16 of the lung and the likelihood that the smaller
17 particles were more important than total particles.

18 PROFESSOR SHERMAN: Is there any plan?
19 You've given us some predictions of reduction in all-
20 cause mortality with the new 20-microgram versus 30-
21 microgram standard. Have you made plans to try and
22 see that your predictions will actually hold true?

23 DR. OSTRO: Well, it's a hard thing to do,
24 because there are so many other factors, of course,
25 that affect mortality and morbidity. Actually, the

1 Health Effects Institute I think is going to be
2 funding some studies that they call accountability
3 studies, which are aimed at specifically doing that.

4 We're trying to look at natural experiments
5 in a way to see what happens pre- and post-. For
6 example, they're looking at, or they're thinking about
7 looking at New York City, which is going to move
8 towards getting rid of diesel buses and maybe certain
9 cars that maybe threaten the whole city, and then they
10 want to try to look at health effects beforehand and
11 afterwards, as a way of looking at that.

12 DR. BALMES: And the Air Resources Board is
13 also strongly considering funding such a study. I
14 don't know if it's finally been approved or not.

15 CHIEF BODE: We are actually, we have an RFP
16 we hope to get out here within the next week or two
17 that does that. Actually, it's asking for a proposal
18 to do that. We'll look at basically changes in the
19 air quality over the last 20 years and identifying any
20 health benefits, which will be difficult --

21 DR. OSTRO: It's a very difficult thing to
22 do.

23 CHIEF BODE: Yeah, very difficult.

24 DR. OSTRO: Because when you're looking at
25 long-term changes, lots of other things change. And

1 again, if you're looking at an outcome like asthma or
2 hospital admissions or so many other factors that, of
3 course, have changed over the last decade, that make
4 it very difficult, but I guess people will make an
5 attempt to do that.

6 PROFESSOR SHEPPARD: But there would be
7 little point in evaluating the effect of setting a
8 standard. Really, the health effect impact would be
9 complying with the standard. And, as you showed,
10 there really hasn't been much change in the overall
11 levels of particles over the last ten years.

12 So despite having set the standard in 1983,
13 really the health effects are going to be seen by
14 enforcement of the standards.

15 DR. OSTRO: Correct, yes.

16 CHIEF BODE: Now, there was a fair amount of
17 improvement in the '80's with regard to particulate
18 matter exposure, where there has been less improvement
19 in the '90's.

20 DR. OSTRO: Well, and also, things could have
21 gotten worse. You know, things staying relatively
22 constant might be a gain in a way, because that might
23 be some of the standards. Things might have gotten
24 even worse. And some of the other pollutants have
25 shown significant changes, significant improvements.

1 In the LA area, for example, where ozone has dropped
2 really dramatically and some of the other pollutants
3 have dropped. Lead and others have dropped really
4 dramatically over the last ten or twenty years.

5 PROFESSOR CROPP: But I think in regard to
6 children, you have to concentrate a great deal on
7 morbidity. Because I think children have a long time
8 to live, children are in general healthier than
9 adults. And we have to try to look at minor morbidity
10 effects in this population compared to just mortality
11 in the older population.

12 And the mortality effects or expected life
13 expectancy is perhaps one parameter to also look at,
14 because I think if you just concentrate on the effects
15 of the older population we miss a great deal.

16 DR. OSTRO: I have to say I'm glad you
17 reminded me, I didn't -- I gave short shrift to some
18 of the morbidity outcomes because a lot of the tension
19 and action is on the mortality. But in the document
20 we have outlined that there have been many studies now
21 in children, healthy children and asthmatic children.
22 There have also been some studies trying to look at
23 infant mortality and other outcomes for infants.

24 So we do review some of those studies. And
25 again, there does seem to be fairly consistent effects

1 relating PM 10 to exacerbation of asthma and
2 respiratory symptoms in general among children, so
3 those studies are out there and definitely have
4 concern as well.

5 CHAIRMAN KLEINMAN: Okay. Bart, thank you.

6 We're going to -- I think we're due to take a
7 break for lunch. After lunch Mike Lipsett is going to
8 take a few minutes and go over the mechanisms.

9 DR. LIPSETT: Only a few.

10 CHAIRMAN KLEINMAN: Only a few. And I was
11 wondering, because it goes back quite a ways, if not
12 today maybe tomorrow we could get a very brief review
13 of the kinds of data that went into the original
14 setting of the PM -- not the PM, but the sulfate
15 standard, way back when. Because I think that plays
16 into considerations of how important is the artifact
17 and the monitoring methods, in terms of, you know,
18 whether there are problems or not, in that particular
19 standard.

20 DR. LIPSETT: I hope we can comply with that,
21 Mike. None of us were working at the Health
22 Department when that was set, and we've actually
23 looked for that documentation and I don't think either
24 one of us has it in our files.

25 CHIEF BODE: I do.

1 DR. LIPSETT: Oh, Richard does, excellent.

2 CHIEF BODE: I have back to '76, '77.

3 DR. LIPSETT: Okay, good. You can summarize
4 it tonight, then.

5 [Laughter]

6 CHIEF BODE: That will teach me a lesson.

7 CHAIRMAN KLEINMAN: Okay, then, we're
8 adjourned until 1:30?

9 CHIEF BODE: Why don't you plan about 2:00
10 o'clock?

11 DR. OSTRO: Or about 1:45, how about a
12 compromise.

13 CHIEF BODE: Okay, 1:45.

14 (Thereupon, the luncheon
15 recess was held off the
16 record.)

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A F T E R N O O N S E S S I O N

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2 CHAIRMAN KLEINMAN: I just wanted to give you
3 some idea of the questions that were addressed to the
4 committee. And those are going to form the basis of a
5 lot of the discussion for this afternoon.

6 So the first issue was were the relevant
7 studies in the various disciplines -- dosimetry,
8 epidemiology, toxicology -- were those identified
9 appropriately, were they interpreted appropriately,
10 and were there any prominent omissions were studies
11 that needed to be included in the review.

12 Were the susceptible populations identified
13 appropriately? Are there other populations that did
14 not receive sufficient attention, and are the data
15 specifically on infants and children considered
16 appropriately, because, as was mentioned earlier this
17 morning, one of the reasons for this re-review and a
18 major reason is the question of whether our air
19 quality standards were adequately protective for
20 infants and children.

21 We're also looking at the possibility are
22 there critical data that should have been considered
23 regarding metrics, averaging times or any other
24 characteristic that should have been included in the
25 review and perhaps were not.

1 gaps in our knowledge base? Are there other
2 susceptible groups, appropriate sampling methods and
3 issues such as coarse versus fine particles and how
4 those can be dealt with in the future, what sort of
5 research is needed. So that's going to provide the
6 framework for most of the discussion.

7 Now, there was an arcane way in which the
8 makeup of the committee was developed, and I'm going
9 to let Mike Lipsett tell us how that came about.

10 DR. LIPSETT: Apparently there is some
11 confusion among the committee members and members of
12 the audience as to who the Air Quality Advisory
13 Committee actually is, and why you people are actually
14 here. And the Air Quality Advisory Committee is
15 actually an ad hoc non-statutory committee that's been
16 in existence -- I won't say continuously because it's
17 had a few gaps in meeting time, but since 1974. It
18 was initially a committee to the Department of Health
19 Services, which our department used to be part of, and
20 since 1991 when our group was separated out from
21 Health Services and made a part of Cal EPA, the Air
22 Quality Advisory Committee provides technical peer
23 review in our recommendations to the Air Resources
24 Board regarding the ambient air quality standards.

25 Now, a couple of years ago there was a bill

1 passed by the State Legislature -- This is Byron
2 Shares' bill -- that required that environmental rules
3 all get technical peer review. And for major rules
4 like this one for the particle standard, it required
5 that the individuals participating in it basically be
6 considered to be world-class scientists appointed in
7 consultation or by the president of the University of
8 California. And so our department submitted a list of
9 names to the University of people who we thought, in
10 addition to our Air Quality Advisory Committee, would
11 be helpful in this process. And if you weren't
12 contacted directly by the president's office, that's
13 how you were selected, was by the president of the
14 University.

15 And so the larger group that's meeting here
16 was convened specifically for the purpose for
17 reviewing this particular standard. In terms of
18 what's going to be happening with the Air Quality
19 Advisory Committee itself, of whom three of the active
20 members are sitting here today -- that is,
21 Dr. Kleinman, Dr. Balmes and Dr. Sherwin -- we're not
22 sure what we're going to be doing in the long term, or
23 whether -- I mean, it may involve a number of you if
24 you're so willing to continue to serve in this
25 process.

1 So, with that, we're going to go to I guess
2 the first section is going to be on deposition. And I
3 had just a couple of slides on this, and then John, I
4 guess you're going to be leading the session; is that
5 correct?

6 DR. BALMES: Well, my understanding from our
7 brief organizational meeting this morning was that I
8 was going to start off the discussion.

9 DR. LIPSETT: Okay. Then why don't you go
10 ahead, that's fine.

11 DR. BALMES: Well, the Dosimetry section of
12 the document, 7.1, goes from 108 to 113, the top of
13 113. So it's a relatively short section, and
14 dosimetry may actually be the wrong title for it, to
15 some extent. Much of the section is about deposition
16 of particles, and it doesn't pretend to be an
17 exhaustive review. But basically outlines I think
18 fairly accepted knowledge about how particles are
19 deposited in the airways and how there is some size
20 selectivity in terms of deposition. And it generally
21 references textbook reviews in terms of deposition.

22 It does acknowledge that there are
23 differences between normal deposition of particles and
24 normal individuals versus those with obstructive lung
25 disease such as asthma or chronic obstructive lung

1 disease, and there is some discussion about in such
2 individuals there can be focal hyperdeposition of
3 particles. And there is some citation of specific
4 papers in this regard.

5 There is a section on clearance, which
6 indicates that some particles remain in the lungs for
7 quite a long time. And then I think probably the most
8 interesting section or subsection of the section,
9 7.1.3, deals with potential differences between
10 children and adults, since this review is in part
11 because of the SB 25 mandate and to specifically
12 respond to children, or just to consider children and
13 their special susceptibilities.

14 I don't know if my pediatric colleagues on
15 the committee will have any major concerns about this
16 section, but it actually does contrast studies that
17 give a different overall result with regard to whether
18 there's greater deposition or not in children,
19 relative to their weight or relative to their absolute
20 minute ventilation.

21 But I thought that the section achieved what
22 was necessary, which was just to provide an overview
23 of what's known about particle deposition and
24 clearance, and some potential differences in
25 susceptibility related to pre-existing lung disease,

1 and actually, a little bit on children's potential
2 special susceptibilities. So I didn't actually have a
3 major problem with this section.

4 CHAIRMAN KLEINMAN: Okay. I'd like to throw
5 it open to the rest of the committee.

6 PROFESSOR CROPP: Well, you made some
7 comments about children, and I think I do want to
8 point out that there are some important considerations
9 in regard to deposition of pollution particles, air
10 pollution particles in this age group. You must
11 remember that a child isn't only young in number of
12 years but the lung is small, the anatomy of the lung
13 is different in that the surface area to volume is
14 different and, therefore, there is a relatively larger
15 surface area available for particle deposition in
16 young children, in infants in comparison to adults.

17 Their movement of air, of gas in and out of
18 their lung is much larger compared to the volume and
19 the size of their lungs in comparison to the adults,
20 so that the exposure of the young child to pollution
21 even at rest and particularly during exercise is much
22 greater than it is in adults. Now, I think that
23 children also exercise spontaneously much more than
24 sedentary adults, and constantly again that will
25 increase their exposure.

1 I think it is also important for everyone to
2 realize that children in general are much healthier
3 than adults. They have not been subject to repeated,
4 many injuries to their lungs, from viral infections,
5 from exposures to cigarette smoke, and to any number
6 of potential injurious materials. And, therefore, it
7 will require a much greater insult to a child's lung
8 to demonstrate the measurable effect than it is for a
9 so-called healthy adult.

10 And then you must remember that children are
11 supposed to live for 80 years or hopefully 85 years.
12 And the injuries are cumulative. It is not just that
13 they suffer measurably in their first five or fifteen
14 years of life. The more they have been injured, in a
15 small way perhaps, the greater effect it will have on
16 their health during their adulthood.

17 I think this is something that is important
18 to remember, particularly the repeated short exposures
19 will each leave a small amount of injury, and that
20 accumulates, even if the effect of the single or a
21 dozen small exposures are not easily measured, not
22 that they cannot be measured. So it's important that
23 we have both short-term consequences of exposures in
24 children that may cause them to miss school or develop
25 an asthma attack or have other signs that are

1 measurable, but it is also important to sort of learn
2 to predict and develop the proper models and
3 methodology to see how the many exposures that they
4 experience, what effect it will have on their life
5 expectancy, as well as on their adult health.

6 DR. BALMES: I'd like to say one more thing
7 to amend my comments. I alluded to the fact I didn't
8 like the title of this section. I actually this
9 Dosimetry is a little bit misleading here. I would
10 change it to Particle Deposition and Clearance.

11 CHAIRMAN KLEINMAN: Okay, but I think, you
12 know, the point being made is very important, that
13 dosimetry plays a role, especially when we're looking
14 at the children versus adults. You know, if the
15 theoretical models in some of the measurements are
16 correct, that children do deposit more material in
17 their lungs for a given exposure level, then it's
18 possible that the standard needs to reflect that to be
19 adequately protective of children.

20 And so one of the questions I think that
21 could be addressed in our thinking is has that been
22 taken into account adequately, which is one of our
23 specific charges.

24 DR. BALMES: The data presented here in the
25 section on differences between children and adults

1 suggests that it's not clear whether children have
2 greater relative depositions than adults. And I think
3 that that's probably a fair assessment of the state of
4 the knowledge.

5 PROFESSOR SHEPPARD: I'm not sure that's
6 really right. Although you can quibble about adequacy
7 of each study that's looked at children and adults, I
8 think there's consistency between models of deposition
9 and data that the deposition of particles in the range
10 we're talking about would be likely to be greater in
11 children. Coming up with a correction factor for how
12 much greater isn't going to be simple, but actually
13 the document suggests that it probably is greater, and
14 I think that more accurately reflects the current
15 state of knowledge.

16 DR. BALMES: I would agree with that, that
17 there's probably a greater deposition but to say how
18 much greater -- I think that's fair.

19 PROFESSOR CROPP: The activity level is
20 particularly important.

21 CHAIRMAN KLEINMAN: Sheldon?

22 PROFESSOR FRIEDLANDER: I have some comments
23 on a later section in that chapter seven. Is that
24 relevant? Can I --

25 CHAIRMAN KLEINMAN: With respect to dosimetry

1 or was it --

2 PROFESSOR FRIEDLANDER: Well, there's a
3 section on pulmonary and systemic inflammation. It's
4 at chapter 7.8.

5 CHAIRMAN KLEINMAN: Would that be more in the
6 mechanisms?

7 PROFESSOR FRIEDLANDER: Well, it was more on
8 the -- Well, it's related to the mechanism in the
9 sense that there are a whole group of studies that are
10 discussed involving various mechanisms and introducing
11 the agent, the pollutant to the biochemical system.
12 In other words, they talk about installation,
13 inhalation, in vitro studies, animal studies. And, in
14 my view, those are -- from the point of view of an
15 engineer looking at this, those are all quite
16 different mechanisms for getting particles in contact
17 with tissue, and --

18 CHAIRMAN KLEINMAN: But I think that's going
19 to be related to our discussion of the toxicology, so
20 maybe we ought to hold that until later.

21 PROFESSOR FRIEDLANDER: Okay. I'd be happy
22 to hold it off, then.

23 CHAIRMAN KLEINMAN: Russ?

24 DR. SHERWIN: Yes, Russell Sherwin. I have a
25 question for information, and the question is the

1 aerodynamically equivalent diameters don't take into
2 account the fact that there actually are particles
3 bigger than ten micra that actually get into the
4 peripheral lung tissue. So the question I'm asking is
5 do we have any information on an understatement of the
6 standard because inhalable particles larger than ten
7 micra of importance -- For example, fibrous silicants.
8 I find them in the lung.

9 And they undergo long access fragmentation.
10 Fibers are much more toxic in general than are non-
11 fibrous particulates. So that could be a meaningful
12 thing. I just have no information. I've tried to get
13 this, but only, you know, with a very personal and
14 limited effort. So the question is what information
15 do we have on inhalable particulates greater than ten
16 micra that enter the lung, including pollens. I find
17 35-micra, 50-micra pollens in peripheral air spaces,
18 we're talking about alveolar spaces, small bronchials.

19 CHAIRMAN KLEINMAN: Well, that's true, but I
20 believe when they talk about PM 10, the 10 represents
21 an aerodynamic diameter and not a microscopically
22 measured diameter. So it takes into account particle
23 density, equating to a density of one.

24 DR. SHERWIN: Well, this is precisely my
25 point, and PM 10 is not microscopic. So

1 theoretically -- Well, for example, no fibers, as far
2 as I know, are counted on PM 10 measurements.

3 CHAIRMAN KLEINMAN: Sure, they are. It's by
4 weight.

5 DR. SHERWIN: What's that?

6 CHAIRMAN KLEINMAN: PM 10 is a definition by
7 mass.

8 DR. SHERWIN: Well, that's why I need
9 understanding, but my understanding is the way those
10 monitors go, the fibers are not brought down because
11 of the way they flow. Now, maybe somebody can
12 enlighten me. That's my understanding, that the
13 regular collectors do not catch fibers.

14 MR. COOK: The question was fibers in the
15 sampler that are five microns?

16 DR. SHERWIN: Well, we'll take them one by
17 one. When you collect PM less than ten, are fibers
18 part of that -- does that monitoring actually pick up
19 fibers? When fibers are actually a different kind of
20 -- obviously, they're not going to be of an
21 aerodynamically equivalent diameter less than ten.

22 MR. COOK: And I don't know that we can say
23 what size range is or is not any fiber equivalent to
24 aerodynamic PM 10 or not, but fibers do make it to the
25 filter, of some diameter, some length.

1 CHAIRMAN KLEINMAN: Well, my understanding
2 is, for example, that asbestos fibers are not picked
3 up; is that true or not?

4 MR. COOK: We use a different sample for
5 asbestos, and a different whole technique for looking
6 at asbestos so we've never really taken a PM 10 sample
7 and looked at it for asbestos. It's completely
8 different.

9 DR. SHERWIN: Well, anyway, all I'm saying,
10 it's very unclear in my mind what actually is --

11 PROFESSOR FRIEDLANDER: I think that fibers
12 often do get caught up, make it through, the less than
13 10 micra, even though the fibers are quite long. They
14 tend to line up in the different of the flow, because
15 the resistance is smaller that way. So they line up
16 and they will go through the kind of collectors. They
17 may go through.

18 PROFESSOR CROPP: As long as the flow isn't
19 turbulent. If you have 40 cubic feet per minute,
20 there may be some turbulent flow.

21 MR. COOK: Well, I guarantee the flow is
22 turbulent because if there's an impactor on the SSI
23 filter, it has to make several 180-degree corners, and
24 so it's --

25 DR. SHERWIN: Well, let me make one other

1 statement. My interest in this was simply I was
2 trying to correlate my human material with what was
3 being picked up. And, as far as I know, there's only
4 one report, and Mamoni did it, I think, M-a-m-o-n-i,
5 who is the only one I know of that's done microscopic
6 studies on what you actually pick up with your
7 filters.

8 It's unclear in my mind, so I'm finally
9 raising it to see if we can get that information. My
10 personal feeling is that we may be understating the
11 actual material that's come in. And we certainly are
12 understating -- Let's say a silicate goes through,
13 even though it's greater than ten micra. It
14 fragments. It fragments in vivo. I have pictures of
15 silicates fragmenting, long axis in the body and
16 macrophages lining up, breaking them up.

17 So we're certainly understating some of the
18 pathophysiologic aspects of dynamic formation of fiber
19 silicates.

20 UNIDENTIFIED FEMALE SPEAKER: My
21 understanding is that the PM 10, that's the 50
22 percent? It's --

23 MR. COOK: There's a penetration curve that's
24 called D 50, and that is the point at which 50 percent
25 of the particles pass through. And so we try to

1 develop ahead such that that cut is fairly sharp, and
2 that you admit certain numbers of particles that are
3 larger than ten microns, hopefully, few, and capture a
4 large percentage of the particles that are smaller.

5 But I don't profess to be an expert in
6 aerodynamic diameter. The definition of that maybe
7 Costas can answer.

8 PROFESSOR SIOUTAS: Well, can I speak to
9 this? The definition of a PM 10 is actually based on
10 the sort of common languages that, you know, it
11 assumes that the particle is a perfect sphere and has
12 a density of one and a diameter of ten micrometers;
13 however, there is a thing. There are normalizing
14 factors for irregularly-shaped particles including
15 fibers. And these particles could be, for all that I
16 know, 15 micrometers long and maybe .5 micrometers in
17 diameter, and their same factor such that, you know,
18 in fact, a PM 10 limit would allow these particles to
19 penetrate and be collected by a filter.

20 The whole notion of a PM 10 is basically
21 trying to mimic the way our throat works, and our
22 throat collects particles or not based on the
23 aerodynamic diameter. So if these particles were to
24 penetrate our throat, they would be collected for the
25 most part by a PM 10 filter.

1 DR. SHERWIN: Well, my basic question simply
2 was I welcome information on somebody who has actually
3 done microscopic studies on what you find in the
4 filters. That was the question.

5 PROFESSOR SIOUTAS: Well, if I may offer
6 again from the PTEAM study, I remember reading the
7 publications of Jack Spindler and his colleagues at
8 the time mentioning about the personal cloud and the
9 fibers in the filters. In the personal monitors that
10 were PM 10 they did, in fact, see fibers. So, you
11 know, that means that these fibers may --

12 DR. SHERWIN: Well, the one guy who did that
13 with the PTEAM was Mamoni, and I looked at the
14 photographs that he prepared, and I didn't see the
15 things that I was seeing in the human lung, and that's
16 what raises this. Because I just think it's something
17 I would like to know about, and I personally believe
18 there is some understatement, either
19 pathophysiologically or mechanical.

20 CHAIRMAN KLEINMAN: Most of the PM filters
21 are fibrous to begin with, so it's very difficult to
22 try to identify ambient fibers from the fibers in the
23 filter. But I think that, you know, we've kind of
24 covered that.

25 DR. SHERWIN: All right.

1 CHAIRMAN KLEINMAN: Are there any other
2 comments related to dosimetry? If not, I'd like to go
3 on to the question of the actual exposures, then, as
4 addressed in here.

5 Dr. Friedlander, would you like to kick off
6 on that?

7 PROFESSOR FRIEDLANDER: Chapter six I think
8 has a general theme that deals with exposure, doesn't
9 it? Let's see, page 47, chapter six, called Exposure
10 to Particles. And in general, I felt that it was well
11 done, had a good overall presentation of the chemical
12 nature and sources of particulate matter throughout
13 the state of California.

14 I felt that the pie diagrams which are shown
15 should have the -- should show the average total mass,
16 because what they show now is the percentages. All of
17 them show percentages. So if someone wanted to know
18 the real -- For example, they show the elemental
19 carbon, five percent. An example, one of them,
20 Redwoods National Park, shows elemental carbon, five
21 percent; soil, five percent; sulfate, 27 percent, and
22 so on. But they don't tell you the total mass, so you
23 can't tell what the absolute amount was, and there are
24 many, many diagrams of that kind. So I would urge
25 very strongly that you include the total mass for each

1 one of those pie diagrams.

2 CHIEF BODE: Actually, we can do that, but I
3 notice that most of the pie charts do have total mass,
4 it's just selectively it looks like we've left them
5 out. So we'll go in there and make those changes.

6 PROFESSOR FRIEDLANDER: All right, good.

7 On page 32, page 32 shows signs -- Wait,
8 that's not the page, that's not in that chapter. Page
9 82 shows particle size distributions, say, for
10 Bakersfield, and there are other figures like that.
11 But they're not really particle size distributions,
12 because they're MOUDI stage distributions, right? The
13 abscissa, the X axis shows the MOUDI stage.

14 So then you have to compare that with the key
15 that's on the right-hand side. And it turns out that
16 the diagram is going from high sizes, large particle
17 sizes to small ones, all of the diagrams of that kind.
18 And that's not a common way of looking at it, at least
19 for people who are not directly working on an air --
20 You're probably accustomed to thinking of it in that
21 way, but it doesn't show the distribution with respect
22 to particle size, it shows with respect to increasing
23 MOUDI stage and decreasing particle size.

24 So it's difficult, I think, at least for me,
25 to translate. And it's not really a particle size

1 distribution, it's a MOUDI stage, so you have to
2 decide whether you want to keep that or translate it
3 into particle size and then re-plot it.

4 CHIEF BODE: We might do that, we might just
5 re-plot it.

6 PROFESSOR FRIEDLANDER: That would be my
7 preference, yes. Because then I could tell what this
8 distribution looked like.

9 Now, this very important section on page 99,
10 on a summary of the last section on that, section 6.5,
11 which deals with characterization of personal and
12 indoor exposure, and the section begins with a
13 statement that "Outdoor PM is usually a major
14 contributor to indoor and personal PM exposure,
15 especially when few indoor sources are present;
16 however, the relationship between indoor and outdoor
17 concentrations and personal and outdoor PM
18 concentrations are complex, and correlations are often
19 low."

20 Now, that's really a very important, one of
21 the more important statements I think in the report,
22 and that is the issue of whether an air pollution
23 monitoring site on which the implementation plans are
24 based and which are used in enforcement and
25 everything, whether they are really related to what

1 people are exposed to and are actually inhaling. I
2 mean, that's a crucial issue and you always get
3 criticism from that point of view.

4 But I think that some of the difficulties are
5 identified, but I don't think that they're -- that
6 it's sufficiently definitive. In other words, I think
7 that you have -- the last sentence is, "However, there
8 remains much uncertainty in the current understanding
9 of these relationships." Well, now, I think that
10 should be reflected in the beginning of the executive
11 summary -- I don't think that that is really singled
12 out -- and I think that somewhere you're going to have
13 to come to grips, you're going to have to make a
14 statement that we're either going to have to live with
15 this the way it is, or we're going to have to do more
16 research.

17 But there has to be -- the second shoe has to
18 drop. Much uncertainty remains, but so? You know,
19 what are you going to do about it? What has to be
20 done about it?

21 CHIEF BODE: And you're thinking of
22 addressing this where in the -- you mentioned in the
23 executive summary?

24 PROFESSOR FRIEDLANDER: I think that there
25 could be some statement here, but it should go back --

1 I think that should be highlighted in the executive
2 summary, which has a list -- which really strives, as
3 I understand it, to pick out the highlights of the
4 report, which I don't think is highlighted in the
5 executive summary, although I think it's very
6 important.

7 It's a crucial issue: How do you relate
8 monitoring site data to what -- If you're going to
9 persuade -- Is this question legal? There are legal
10 issues involved, and whether you can defend these
11 standards in court, because people -- I've been to
12 many meetings where standards are attacked on the
13 grounds that what's the relationship between what an
14 air pollution monitoring site measures and what people
15 are exposed to, and why should we accept your
16 standards because there's not a convincing
17 relationship.

18 So I think you should identify that in the
19 executive summary, and then also, if necessary, it
20 could be a kind of a pious recommendation for more
21 research. But I think you should try to focus it a
22 little bit better.

23 PROFESSOR THURSTON: Could I -- just one
24 response to that issue, because I think you're right,
25 this is something that has to be discussed clearly in

1 the document. Because there is a confusion out there
2 that what we should be using is personal exposure to
3 total PM whatever, PM 2.5, PM 10, and that is not the
4 case. What we want is personal exposure to outdoor PM
5 2.5 and personal exposure to outdoor PM 10. Because
6 that's what's being regulated.

7 Indoor air pollution is not being regulated.
8 So if, in fact, for, let's say, the epidemiology
9 section, if a study had been done using personal
10 monitoring, the first thing you'd have to do is go
11 through and extract out the indoor exposures from the
12 personal data before you could relate it to, relate
13 health to the outdoor pollution.

14 So that while the correlation is pretty good
15 between the central site monitors and total personal,
16 it's really good when you're looking at -- because, as
17 indicated in the document, I mean, they give these
18 exposures, you know, very high -- I think on page 96
19 they're talking about outdoor particles contributed 76
20 percent of the PM 2.5 mass and the PM 10 mass, indoor
21 particles. So the outdoor is a big chunk of the
22 indoor, but it's -- These central-type monitors are
23 very good indicators of personal exposure to outdoor
24 particles.

25 And I think that distinction needs to be made

1 clearer to avoid that confusion that enters into this
2 sometimes, where people say, well, you get most of
3 your personal exposure indoors, so outdoor is
4 irrelevant. But actually, they are very relevant to
5 what's the subject matter of this process, which is
6 what are the health impacts of outdoor pollution.

7 CHIEF BODE: Why don't I actually -- I'm
8 going to have Peggy Jenkins from our Indoor Exposure
9 Group kind of respond to that section.

10 MS. JENKINS: Right, and actually,
11 Dr. Thurston I think characterized it very well.
12 That's, in fact, I think how the epi studies, what the
13 epi studies are looking at. What the added indoor and
14 personal exposures really I think tell us, and what
15 that uncertainty is, is really what else is going on
16 kind of above and beyond what we feel we know
17 something about, relative to the outdoor air
18 pollution.

19 So there's perhaps something in addition to
20 what we're able to measure in the epi studies,
21 relative to outdoor pollution, in terms of those
22 heightened indoor and personal exposures when we see
23 them. But I don't feel that that really negatively
24 impacts the conclusions that are drawn from the epi
25 studies that we have.

1 I think there are some very recent exposure
2 study data that have been very useful in helping us
3 understand this a little more, and I would have to say
4 we may not have explained this as fully as we should
5 have or could have in the document. The most recent
6 personal exposure studies really have begun to show a
7 stronger correlation in at least a segment of the
8 population to the outdoor ambient levels that have
9 been measured. We see a great variability. There is
10 a part of the population whose personal exposures
11 don't appear to be very well correlated with outdoor.
12 But at the same time there's a group who are very
13 strongly correlated.

14 And we don't know all the reasons why. It
15 appears to have to do with their personal activities,
16 if they're indoors, keep their house closed up, don't
17 do much in terms of going out, they may have a lower
18 personal correlation, or higher, depending on what
19 they do and don't do. So there's a variability,
20 there's a continuum or a spectrum, and the more recent
21 longitudinal exposure studies have done a better job
22 of really teasing that out.

23 Also, I think as we're starting to look more
24 and more at PM 2.5 or smaller size cuts and fractions,
25 we're seeing stronger correlations with those outdoor

1 levels. It may not be something we brought out very
2 well in the document, perhaps that needs emphasis.
3 Certainly, we think that that information in part
4 helps explain why we do see the relationship between
5 the ambient levels and some of the health effects that
6 were seen.

7 I don't know if that helps, but -- And I
8 think you're right, we need to probably do a little
9 bit of revisiting on the executive summary; that's
10 true about that.

11 PROFESSOR FRIEDLANDER: Yeah, I think that
12 there should be, there out to be a more definite
13 statement about where things stand, because you leave
14 it up in the air. And I don't think it appears in the
15 executive summary, does it?

16 MS. JENKINS: I don't think it does, no.

17 PROFESSOR FRIEDLANDER: And I think it's so
18 important that it definitely deserves a place there.

19 PROFESSOR CROPP: In that regard, one other
20 comment about children. Children ambient outdoor
21 levels are very important, because particularly in
22 California, children spend a great part of their time
23 outdoors. We adults who have to work for a living
24 spend our times on computers and indoors. But our
25 children fortunately have the opportunity to be

1 outside. And, therefore, I think the health effects
2 of outdoor pollution measurements are very relevant to
3 children.

4 MS. JENKINS: That's right, and I think they
5 tend to be pretty active when they're outdoors too, so
6 as far as, you know, there's a --

7 PROFESSOR CROPP: Right. I mentioned that
8 before, that the degree of activity certainly
9 determines your exposure, the more active. I mean, we
10 have other evidence from athletes, and athletes in
11 general, for instance, have more asthma than non-
12 active children and adults. And the reason is --
13 Possibly, I mean, this is hypothesis -- but it may be
14 due to greater exposure to outdoor pollutants.
15 Because they have to breathe much more when they play
16 soccer and football and whatever.

17 MS. JENKINS: Right.

18 PROFESSOR FRIEDLANDER: So it would be
19 appropriate, then, following the chairman's
20 admonition, to identify particularly susceptible
21 subgroups, to pick that out in the beginning and say
22 that, in a sense, fortunately, the reliance on the
23 monitoring stations is probably best for that subgroup
24 so we can have more confidence in the relationship of
25 the outdoor monitoring site, of the monitoring sites

1 to actual exposures of an important subgroup.

2 One minor point, on page 93, line ten, the
3 first PTEAM study, but I couldn't find -- I was
4 interested in that reference, but I couldn't find it
5 again in that chapter. It seemed to have been
6 omitted.

7 DR. SHERWIN: PTEAM?

8 PROFESSOR FRIEDLANDER: Unless it --

9 CHAIRMAN KLEINMAN: There's a reference to
10 Wallace. I think he covers the PTEAM in his article.

11 CHIEF BODE: But that's actually a general
12 problem. There are many references missing throughout
13 the document. When I went to look up references, I
14 mean, the section that I just described or just
15 discussed, the so-called dosimetry one, there's a
16 Lipsett reference, 1995, about children's physiology,
17 which I'm fairly aware of your bibliography, Michael,
18 and I don't know if you actually did write such an
19 article, but there are a number of references that
20 have to be checked and a lot were missing.

21 So the Pellizzari reference, I think, is the
22 one you're referring to isn't in there, you know, for
23 example.

24 MS. JENKINS: Pellizzari '99?

25 CHIEF BODE: Yeah. It's referenced in the

1 document, but in the back of the chapter you won't
2 find it. There's a number like that. So the
3 references just really have to be more carefully
4 checked in the document.

5 MS. JENKINS: Now, we do have a Pellizzari
6 '99.

7 CHIEF BODE: Well, not the one I'm looking
8 at.

9 MS. JENKINS: Page 102?

10 CHIEF BODE: Yeah.

11 MS. JENKINS: Line 39?

12 CHIEF BODE: Okay, sorry. Oh, this is six.
13 That specific reference I may be wrong about, but if
14 you look, I'll throw one out which I happen to be co-
15 author of, in chapter seven there's a reference to
16 Ares, et al., 1991, and looking alphabetically on page
17 188, there's not an Ares reference, so believe me, I
18 like to look back at references and there are a number
19 that are missing or wrong, in terms of date.

20 DR. SHERWIN: I was also concerned about that
21 PTEAM reference, but when I saw a reference 23,
22 Wallace, L., that's Lance Wallace, I knew that that
23 had the PTEAM references in it, so I excused it.

24 PROFESSOR FRIEDLANDER: So that should be the
25 PTEAM, if it's -- it should be Wallace.

1 DR. SHERWIN: Yeah, Lance Wallace was very
2 active in the PTEAM group.

3 MS. JENKINS: But you're right, I think we
4 have probably -- we left out one of the primary
5 references and there's a secondary. We'll get the
6 primary in there as well. I think they should both be
7 in the listing.

8 CHIEF BODE: Well, we'll go through and we'll
9 check all of the references as we put this document
10 together to make sure we've covered them all.

11 PROFESSOR FRIEDLANDER: Well, also, since
12 we're bringing up references that were left out, turn
13 to page 57, and you talk about source apportionment,
14 chemical mass balance models. Actually, the first
15 were those done by my group in the early '70's, and
16 they were sponsored by guess who? By the ARB, classic
17 work on source apportionment.

18 So I think that we're both suffering, because
19 we left out the ACHEX experiments sponsored by the
20 ARB.

21 CHAIRMAN KLEINMAN: I'd like to, you know,
22 try to keep to our time, so references and things like
23 that, and I think editorial comments we ought to put
24 in writing, because those aren't really -- unless they
25 misinterpret the reference or something like that, I

1 think that's valuable and important to bring out, but
2 if it's just adding in some additional references,
3 unless they change the tenor of what we're going to
4 discuss, I think those are best done in writing.

5 Were there any other comments about the
6 content or the science? Because I had one issue that
7 I just wanted to raise, and it probably relates more
8 to the standard-setting rationale but it kind of falls
9 under this category. And that is there is an
10 underlying concept that there is tracking between the
11 PM 10 and PM 2.5, and there's a statement on page 180
12 that says, "Short-term standards will address
13 intermittent seasonal exceedences; for example, from
14 residential, combustion," etc., etc. So that by
15 taking, setting a standard for PM 10 it's thought
16 that, you know, you'll cover short-term exceedences.

17 And I wanted to point out, if you look at
18 page 74 and 75, on page 75 there is an example of a
19 very short-term spike during the course of the day
20 which dominates 24-hour average. You get a spike up
21 to 250 micrograms per cubic meter in rural Sacramento
22 on a smoky day. If you look at all the data points
23 for the day, they're all down around between 25 and
24 50, and the 24-hour average is pushed up because of
25 this spike.

1 And I think it's important to keep in mind
2 that although we are monitoring things primarily on a
3 24-hour basis that there has been very little work
4 done, although now more is being done, with hourly
5 measurements. And I think it's very important that we
6 understand what the day-to-day peak exposures and
7 profiles of those peak exposures are. Because those
8 can dominate the exposure of individuals, especially
9 if, in this case, it happens right around noon when
10 people are outside possibly more than they are
11 indoors.

12 And so I think it's important to keep that in
13 mind. We don't have maybe enough data right now to
14 start taking into account in the standard-setting
15 process, but I think looking at these data makes the
16 point that we really do need to get the continuous
17 monitors up, running, calibrated, and in use so that
18 we can begin to understand some of this data.

19 The other point I wanted to make was if you
20 look, and this is -- I'll just say on page 74, there
21 are some examples from the Sacramento Valley. If you
22 look at the PM 10 versus PM 2.5 monthly
23 concentrations, they don't really track very well.
24 And so, again, these are I guess monthly averages.
25 But I think the take-home message here is it may not

1 be appropriate, and I think the data need to be
2 analyzed to, you know, put the issue to rest --

3 MS. JENKINS: Dr. Kleinman, what page is
4 that?

5 CHAIRMAN KLEINMAN: Page 74. It's a figure
6 on the Sacramento Valley from Colusa 2000, and it
7 shows PM 2.5 and PM 10.

8 UNIDENTIFIED FEMALE SPEAKER: Dr. Kleinman?

9 CHAIRMAN KLEINMAN: Yes?

10 UNIDENTIFIED FEMALE SPEAKER: Yeah, those are
11 not monthly averages, those are the maximum per month
12 for each --

13 CHAIRMAN KLEINMAN: Okay, maximum per month.
14 But I'm just pointing out that there are differences
15 in tracking between PM 10 and PM 2.5 And I think part
16 of the answer here the monitoring methods are
17 different.

18 UNIDENTIFIED FEMALE SPEAKER: Right.

19 CHAIRMAN KLEINMAN: And I don't know exactly
20 what the impact is, but if not during this cycle of
21 review, certainly during the next cycle of review we
22 really need to have a very good understanding of the
23 relationship between the PM 10 hourly changes or 24-
24 hour changes versus the PM 2.5 changes. Because then
25 we'll know whether, you know, if we set a PM 10

1 standard, is that really going to give us protection
2 against PM 2.5 spikes?

3 PROFESSOR SHEPPARD: I'd like to follow up on
4 that point. Just a general comment that comes up
5 throughout the document is the difficulty interpreting
6 the legends and axis labels that just came out in this
7 point. The axis -- I misinterpreted all of the
8 figures in this chapter, because the X axis just says
9 Month, and the Y axis just says Concentration.

10 And a similar issue comes up actually in
11 chapter seven in the epidemiologic data, which is
12 difficult to know what is actually being plotted,
13 whether it's annual mean, total mean over many years,
14 24-hour mean. So just to make the document clearer, I
15 would suggest that in all the figures there be more
16 detail in the legends and the axis labels.

17 But I also think, it's worthwhile
18 underscoring the point that was just made, that in
19 other places in the document where interpretations are
20 made, the point isn't really brought out clearly
21 enough how disparate the values can be between PM 2.5
22 and PM 10. So in the executive summary or the
23 rationale for standard setting, the implication is
24 made that by regulating PM 10, for example, on a daily
25 basis, it would have an impact on PM 2.5, whereas the

1 data in the document clearly show the discrepancies.
2 And I think it would be worthwhile to highlight those
3 discrepancies more prominently in the executive
4 summary.

5 PROFESSOR SIOUTAS: One last statement on the
6 issue of the exposure. It kind of goes back to
7 Professor Friedlander's original request about
8 emphasizing the difference between stationery monitors
9 and personal levels. Dr. Thurston pointed out that,
10 you know, basically when it comes to regulations, it's
11 the outdoor air that matters. And in that sense, some
12 of those monitors, some of the data actually collected
13 in stationary monitors are appropriate.

14 What I would like to point out is that, you
15 know, when it comes to personal exposure, it isn't
16 just a contribution of -- and health effects, in
17 particular. It's not just a contribution of indoor
18 sources that will add variability. There could be a
19 lot of potentially important toxicological components
20 of PM -- black carbon, metals, PAH's -- that are
21 highly variable spatially. They're not the same
22 within an area like Los Angeles. People who live in
23 downtown LA, they're not exposed to the same
24 concentrations as people who live in Riverside.

25 So I do want to emphasize again the need at

1 some point to create more of these databases based on
2 personal monitors and move away from the assumption
3 that the stationary monitor does reflect personal
4 levels accurately. And that again goes back, not just
5 in terms of the contribution of indoor sources, but
6 taking into account your own report, in fact. And on
7 page 98, in the last paragraph it discusses the
8 elevated PM concentrations that can occur, for
9 example, during commute. The elevated mass and carbon
10 concentrations measured inside vehicles.

11 So these are all I think details that one has
12 to take very, very seriously into consideration when
13 it comes to assessing exposure and health effects.

14 PROFESSOR CROPP: Another small point might
15 be also the composition of the PM 10 and PM 2.5
16 material; namely, there are enormous differences in
17 what is in this PM 10 sediment that you measure from
18 one place to another, from one season to another, from
19 one weather condition to another; in other words, how
20 much metal, how much organic material, how much
21 sediment from smoke from agricultural materials and so
22 on.

23 I mean, the composition is very variable and
24 the toxicity may also be very variable.

25 CHAIRMAN KLEINMAN: I think that's an

1 important point, that not all particles are equally
2 toxic, although the way our current standards are,
3 we're dealing primarily with just mass.

4 PROFESSOR CROPP: But, I mean, even the mass
5 can change substantially if people live, you know, ten
6 meters or 20 or 15 meters from a freeway or business
7 street versus, you know, up on a hill far away from
8 transportation sources.

9 PROFESSOR FRIEDLANDER: Yeah, I think that
10 that should be brought out up front in the executive
11 summary or one of the health-related chapters; that
12 is, that the metric that we have, the primary metric
13 that we have for particulate matter is the mass, in
14 certain size ranges. We have two chemical metrics.
15 One is sulfate, the other is lead. That's also in the
16 particulate matter, although somehow it's uncoupled
17 from the rest of the particulate matter, but it's an
18 integral part of particulate matter.

19 And probably, one of the recommendations that
20 I've made is that this be integrated for the next go-
21 around where you recommend a revisiting of the
22 standard, that you look at it as overall component in
23 which you look at not only -- you limit it to sulfate,
24 PM 10 and PM 2.5. But the lead is just as much a part
25 of the aerosol as the sulfate. And you had a separate

1 standard for it, it may not be justified, it may not
2 be -- the sulfate may not be justified either. But
3 these are the kinds of issues that you have to give
4 some thought to.

5 And, as I said, as far as the chemical
6 components, I think that we've all kind of -- we've
7 entered into kind of a conspiracy of silence where we
8 don't mention the PM as very variable chemically, and
9 for a lack of other methods we simply use the total
10 mass, except for the sulfate and lead, in different
11 size ranges to characterize it. And I think that
12 scientists looking at documents like this, and I've
13 been guilty of putting my stamp of approval on
14 documents like this where we don't mention that -- we
15 know that chemically we know these components are very
16 different, but that we're --

17 And it's remarkable that the epidemiological
18 data seems to be correlatable by a relationship to
19 mass. There's a lot of scatter, as you've showed
20 earlier. And I think you have to, in my view you
21 should mention that, that it's surprising that we do
22 as well as we do by relating the epidemiological
23 results, the mortality, the percentage increase in the
24 mortality to the mass. That's really remarkable,
25 considering how variable the chemical components are.

1 And we have to live with it at this point.
2 Maybe in the future we'll be able to uncouple the
3 different components present in the particle, fine
4 particles and coarse particles.

5 DR. BALMES: I'm not an exposure, a sediment
6 expert or an atmospheric chemist, but in fairness to
7 the authors, there is a section called Physics and
8 Chemistry of Particles in the document, which I think
9 is from, again, a non-expert point of view, I think it
10 does a reasonable job of pointing out the fact that PM
11 is not a homogeneous --

12 PROFESSOR FRIEDLANDER: But that's not the
13 issue. The issue is that you can set standards for a
14 very mixed --

15 DR. BALMES: I think that should be
16 highlighted, that it's amazing that the epidemiology
17 does correlate with mass.

18 PROFESSOR FRIEDLANDER: That's right.

19 DR. BALMES: I second that. But I think that
20 for a document of this type, the chapter --

21 PROFESSOR FRIEDLANDER: It's good.

22 DR. BALMES: -- on Physics and Chemistry of
23 Particles is pretty good.

24 PROFESSOR FRIEDLANDER: Yeah, absolutely.

25 CHAIRMAN KLEINMAN: But I think

1 Dr. Friedlander's point is, if I may put words in his
2 mouth, would be that the lack of carrying this through
3 and integrating that inhomogeneity into the process is
4 a limitation which eventually we're going to have to
5 address.

6 And again, we may not be able to do it in
7 this cycle, but perhaps by five years from now there
8 will be enough data that these are the kinds of things
9 that should be considered as additional research
10 needs.

11 DR. BALMES: I agree with that point, but I
12 don't think it's buried in the document that PM is not
13 homogeneous.

14 CHAIRMAN KLEINMAN: No, I agree with that.

15 PROFESSOR THURSTON: Well, and I'd have to --
16 Say, I don't -- you know, first of all, there's no
17 conspiracy of silence here. I think that we're
18 focusing on PM 2.5, and that's what the standard-
19 setting process is about. So while I agree with you
20 wholeheartedly that we have to get to the components
21 point of view eventually, we don't have that
22 information yet. We ought to be getting that
23 information. I think that's the real message.

24 But, you know, the fact that we are able to
25 find these correlations means that it is the more

1 spatially homogeneous pollutants that are probably
2 responsible, because only those would keep showing up.
3 And that really points towards the fine particles,
4 things like sulfates and PM 2.5. PM 2.5 is amazingly
5 homogeneously, at least homogeneously or spatially
6 correlated. You know, if you do a correlation of PM
7 2.5 in Manhattan, and I've had multiple monitoring
8 stations going, we've compared, you know, a mile, mile
9 and a half away, and they just lay right on top of
10 each other, and very highly correlated. It's amazing.

11 And you go over to the Bronx. A high day in
12 the Bronx is a high day in Manhattan. And, in fact, I
13 have some results where we have in Sterling Forest,
14 and the highest days in Sterling Forest which is out
15 in a rural area are the same days that are the highest
16 in the city. That's because there is this regional
17 pollutant that's dominating the day-to-day
18 variability.

19 So it really -- there is a lot of spatial
20 homogeneity in the concentrations of PM 2.5, and I
21 think that's a lot of why we're able to get these.
22 And, you know, it's like a thermostat that you have in
23 your house. It's not going to tell you exactly the
24 temperature throughout your house. Some places near
25 the heaters are going to be hotter, some places near

1 the windows are going to be colder. But when that
2 thermostat goes up, every place in the house is going
3 up; and when the thermostat goes down, everything goes
4 down together. And that's really what the
5 epidemiology is telling you.

6 CHAIRMAN KLEINMAN: Well, I think that's true
7 in a well-mixed regime, and certainly the Northeast
8 Corridor, you know, the East Coast definitely has
9 that. But I think Costas can talk a little bit
10 differently.

11 PROFESSOR SIOUTAS: I was going to say,
12 George. You know, this is correct in areas like
13 Manhattan, where the aerosol is actually regional.
14 It's absolutely incorrect in the Los Angeles basin,
15 where the aerosol is primarily vehicular emissions and
16 photochemically generated aerosol. And within view,
17 you will be surprised, we know that from our supersite
18 measurements now, you would be surprised, even on a
19 mass, on a PM 2.5 basis how diverse the levels are
20 within a distance of 20 to 25 kilometers.

21 PROFESSOR THURSTON: Yeah, but are they
22 correlated I think is the bottom line?

23 PROFESSOR SIOUTAS: No, they're not,
24 because --

25 PROFESSOR THURSTON: So a high day in one

1 part is not a high day in another.

2 CHAIRMAN KLEINMAN: That's exactly right.

3 PROFESSOR SIOUTAS: That's exactly right.

4 PROFESSOR THURSTON: Then you're not going to
5 do as well in those places --

6 PROFESSOR SIOUTAS: That's right.

7 PROFESSOR THURSTON: -- with the
8 epidemiology.

9 PROFESSOR SIOUTAS: But that's not something
10 that needs to be borne in mind, because you tended to,
11 you know, you gave the impression that this is sort of
12 a universal truth about the special homogeneity of PM
13 2.5, and it's not. Especially not when it comes to
14 the largest city of this state whose air quality
15 standards you are reviewing, Los Angeles.

16 PROFESSOR CROPP: And I'm sure that's even
17 more so if you go to rural areas. If we go to north
18 of Sacramento where there's a lot of rice burning and
19 forest areas where there will be a lot of burning of
20 lumber, refuse, that composition of 2.5 is going to be
21 very different than it is in Los Angeles.

22 CHAIRMAN KLEINMAN: Are there any other
23 comments that anyone would like to make? I again want
24 to emphasize that all of the committee members are
25 going to provide written comments that are going to be

1 integrated and sent to the folks who have put together
2 the report so that they can respond to those where
3 appropriate. So we will get all those on the record.

4 If there are no further comments, I'd like to
5 move on to the epidemiology part, and Dr. Tager.

6 PROFESSOR TAGER: Okay. It's obviously a big
7 chapter. Could have been a lot bigger. So I want to
8 address several issues that I consider, from my point
9 of view, the important issues and a lot of smaller
10 things that are not so important right now.

11 Before I start with a list, I would point out
12 on the executive summary page, lines 15 and 16 -- Wait
13 a minute, let me make sure -- no, it's lines 13 and
14 14. It says, "While there are compelling studies
15 which associate long-term PM 2.5 to increased
16 mortality and morbidity effects, there are fewer
17 studies in the effect of short-term exposure." I
18 don't agree with that statement. I think that -- I'm
19 just stating a fact. Lines 13 and 14 on page two, I
20 think that's a misstatement of fact.

21 So I'm going to deal with five issues: Is
22 the overall summary a fair summary? What about the
23 emphasis on long-term effects versus short-term
24 effects and basing it? And similarly, the emphasis on
25 mortality relative to other morbidity end points. The

1 method of expressing uncertainty which is -- what is
2 done here has been done in many other situations and I
3 actually don't think it's the right way to go about
4 it, and I'll discuss it. And then the whole issue of
5 whether the arguments here justify the lack of a 24-
6 hour standard for PM 2.5

7 So the first is the issue of the overall
8 summary. Now, obviously, this is a huge literature
9 and you could pick out any huge number of other
10 papers to include in it, so the first question is was
11 it a fair selection from the literature? From my
12 point of view, it was a fair selection of the
13 literature. Now, that doesn't mean it was exhaustive,
14 and it doesn't mean that other people might not
15 suggest that two or three or five other papers would
16 or should have been included, but my reading is it was
17 a fair selection of an immense literature.

18 The second issue is were all the various
19 uncertainties dealt with. Well, at some level they
20 were all dealt with, but maybe not to the extent that
21 some could have. And I'm going to come back to this
22 when I talk about expression of uncertainty. I think
23 that all the major points were highlighted and the
24 areas of disagreement were noted, but I don't think in
25 some cases they necessarily were translated perhaps

1 into some quasi-quantitative terms that would help a
2 little bit with the uncertainty estimates, and I'll
3 come back to that in a minute.

4 But I think in a broad way, the issues were
5 touched upon, and the areas of disagreement and
6 uncertainty are certainly noted there. And I'm making
7 these comments based on the assumption when I read
8 this, which Bart mentioned when he made his summary at
9 the beginning, is that they didn't have the time, the
10 resources and the inclination to produce an EPA-type
11 multiple-thousand-page, everything last thing that's
12 been published. So obviously, choices had to be made,
13 and I think this was a reasonable set.

14 Now, I have really -- I guess I have a
15 disagreement with the emphasis on the long-term data
16 as the source of choosing the standard. I felt this
17 way in the EPA's analysis and I feel that way. I
18 think these data are -- well, they are as they are and
19 they've been portrayed accurately here, but they're
20 relatively sparse. They have things mixed in them
21 that we can't quantify, including long-term cohort
22 effects, which we can't take out.

23 I mean, if you're talking about long-term
24 exposures, you can't look at exposures over the last
25 six years of the a study or ten years or even 16 years

1 of a study and say that you've quantitatively assessed
2 what the effect of that chronic exposure is,
3 especially since, and I would concur that there is
4 lots of data from many different sources about the
5 effects of air pollution and other kinds of things
6 such as environmental tobacco smoke on children that
7 have long-term effects.

8 So I think there are some serious issues
9 apart from the fact there are only a couple of
10 studies. And the reality is there are only two
11 studies that have adequate data that could be
12 considered, quote, unquote, consistent. So while I'm
13 not dismissing those studies and I'm not suggesting
14 that they're somehow fatally flawed, I'm a little
15 concerned that they become the basis for setting or
16 making decisions, when there's a huge wealth of
17 studies on short-term effects.

18 Coincident with that, it also bears on the
19 focus. Now, while it is true that estimates have been
20 made about the loss of life associated with estimates
21 derived from these long-term studies, if you really
22 look at the numbers, and we'll come back to this with
23 the uncertainties, in terms of the morbidity, first of
24 all, you're talking about lost life towards the end of
25 life, which is all we can quantitate right now.

1 Because we don't have the kind of data to assess how
2 much life is really lost in a birth cohort due to air
3 pollution. We don't have those kind of data. So
4 you're looking at loss of life at the end of life.

5 But it also bears on the burden of morbidity
6 to the society, and some of the estimates even here,
7 looking at hundreds of thousands of these lower
8 respiratory illnesses are not necessarily minor
9 illnesses. They have tremendous impact on
10 individuals' lives, economic impacts, etc., and I
11 think we have a larger database in which to estimate
12 the precision of these effects, and a large enough
13 database to do a different kind of uncertainty
14 assessment than is typically done.

15 So I'm not sure I agree with the focus. In
16 fact, I'm sure I don't agree with the focus. I think
17 I would have put the focus on the short-term studies.

18 Now, the issue of uncertainty -- This is not
19 a criticism of what's done here, because I know what's
20 done here is what's been done, and I don't think this
21 is really -- a confidence interval does not give an
22 estimate of uncertainty in the sense that I'm
23 interested in. What is to me a more appropriate
24 uncertainty analysis is to look across the range of
25 estimates from different types of modeling, and say,

1 okay, under a wide set of model assumptions, what are
2 the possibilities of effect sizes here, and look at
3 their distribution.

4 Now, admittedly, there are going to be
5 studies that don't show effects. That's okay. But
6 the point is, I think you get a clear idea of the
7 uncertainty of the risk by looking at the distribution
8 of the risks associated with the various effect
9 estimates rather than a confidence interval, which
10 says basically, you know, if I did the study a hundred
11 times, 95 percent of the time the mean might be there.
12 It doesn't say that the mean lies somewhere between
13 those two values.

14 So it's not really an uncertainty analysis in
15 the sense that I'd like to see it done to really know
16 what the range of uncertainty is. And I know that
17 what was done here was done in many other places, so
18 this is not necessarily direct criticism of what was
19 done here, because it follows sort of a pattern. But
20 it's my criticism of the way it's been done in
21 general.

22 And I also think there are some points that
23 were touched upon here that should be clarified. I
24 think that -- I don't disagree with the statements,
25 but they need to be clarified in terms of what the

1 epidemiology is saying. And they bear on some of
2 these points about what central monitors do and what
3 happens when we talk about personal exposures.

4 In the time-series studies, which are looking
5 at population-level data and you're not looking at
6 individuals, you're making an assumption -- This thing
7 called Burksett errors -- that you have a machine
8 which is spitting out an average level of air
9 pollution. And what people get in the community,
10 however described geographically, varies in a random
11 way around that mean. And in many places, that's
12 probably a very realistic set of assumptions. And it
13 might even be in microscales in the environments of
14 Southern California and even the Bay Area, where there
15 is tremendous heterogeneity, and these things could be
16 combined.

17 And the fact of the matter is that you don't
18 produce biases with those kinds of analyses. That's
19 different when you start doing cohort studies or panel
20 studies and you're talking about individuals, in which
21 you're dealing with a very different kind of error
22 problem in which, at least as far as we understand
23 from empirical data, it looks like in the most
24 reasonable scenarios that the biases are towards the
25 no, but that's not guaranteed.

1 But I think there's been a lot of confusion.
2 I think the document starts to address that, but I
3 think it would serve itself well, especially if it's
4 going to focus it, as I would have it, on more of the
5 short-term studies to point this out, that a lot of
6 these time-series studies are, in fact, using a
7 statistical set of assumptions that are very
8 believable, certainly in areas like the Northeast and
9 in smaller areas on the West Coast.

10 And I think that the document could do a
11 better job, and I personally would like to see a
12 different kind of uncertainty analysis or a
13 supplemental uncertainty analysis, let's put it that
14 way, where we look across the range of effect
15 estimates under different kinds of modeling
16 assumptions.

17 And then the last point I want to bring up
18 and then I'll stop, is I don't think the argument in
19 favor of not having a 24-hour standard is supportable.
20 In fact, I think on page 179, the argument doesn't
21 stand up. If the argument is made that, well, you
22 can't really tease out the chronic effect -- you can't
23 really completely tease out the acute effect because
24 there's an underlying chronic effect.

25 The problem with this is that everybody says

1 it's a linear exposure response relationship, both in
2 the short-term studies -- in the short-term studies.
3 Well, if that's true, that means it's -- incremental
4 change is the same across a wide range of chronic
5 exposures. And if it were -- And if it were the fact
6 that the chronic exposure was affecting this, you'd
7 have a non-linear response relationship.

8 So you can't have it both ways. If you think
9 that this is really linear, then in essence the
10 chronic effect can't be driving these short-term
11 blips, because otherwise you'd expect to see different
12 short-term effects at different chronic levels. So I
13 don't think the argument stands the logic test, from
14 my point of view.

15 And I think also, as I said before, that the
16 focus on short-term effects, with this wealth of data,
17 there's more data available, at least as far as I can
18 see, to try to estimate what that would be. I think
19 there's value to it, especially if a real-time
20 monitoring system is going to be put into place to
21 begin to look more seriously. I mean, maybe one might
22 argue we don't have enough real-time data to do a good
23 job of knowing where that should be and the
24 epidemiologic data might not be sufficient enough.

25 But I think we could get an answer to that from the

1 kind of risk -- uncertainty analysis that I'm talking
2 about.

3 So I think I'm going to stop at that point.
4 There are a lot of other issues that could be brought
5 up, and now I'll give George a chance to rebut my
6 summary.

7 PROFESSOR THURSTON: No, I just -- I did have
8 a question about the -- that it can't -- if there's a
9 linear effect, that it can't be driven by the chronic.
10 I don't see how that's necessarily true, I'd like to
11 see the proof of that. I mean, just logically, if the
12 chronic exposure, if the acute effects or the fact you
13 see a correlation with short-term is due to the fact
14 that that's been like the last straw that finally
15 pushes somebody. If the chronic were lower, then that
16 might not push them, because they didn't have that
17 chronic exposure.

18 PROFESSOR TAGER: But now you're -- But then
19 you're arguing against -- because we're being told
20 that many of these things are not harvesting effects.
21 And now you're saying, well, that the argument really
22 depends on their being just harvesting. I mean, as
23 far as -- I mean, I can't --

24 PROFESSOR THURSTON: No, the harvesting
25 question is just whether they were going to die two

1 days later or whether they were going to die years
2 later.

3 PROFESSOR TAGER: Well, you have to explain,
4 if you same unit change, short-term basis, produces
5 the same increment of risk on this multiplicative risk
6 scale that we use, and it doesn't matter whether you
7 live in a world that averages PM 10 or 30 or 60, then
8 if chronic effect, which is supposedly driving
9 mortality over the long term, I don't understand, you
10 would expect that some of these studies would have
11 some evidence of non-linearity in the exposure
12 response relationship.

13 And that -- Because being pushed over the
14 edge would be a function of both your long-term
15 exposure as well as your short-term exposure.

16 PROFESSOR THURSTON: It should be.

17 DR. OSTRO: I have a bit of a response to
18 that. One possibility could be -- I mean, in those
19 studies that have really looked at high levels, looked
20 at a wide range -- I'm talking about studies like in
21 Santiago and Bangkok and cities where you really get
22 high levels -- there is some evidence of non-linearity
23 in the time-series studies; that is, you start to see
24 less linear effect.

25 So it could be the case that even in studies

1 in the US and Europe, the reason that you see those
2 linear effects is because at the higher levels, those
3 chronic effects are starting to kick it up. So that
4 it forces a more linear relationship over the whole
5 range.

6 PROFESSOR TAGER: I mean, I wouldn't disagree
7 with that, but the point is, we have to look at the
8 data such as they are. And, I mean, that's certainly
9 a hypothesis that you could test. But given the data
10 that they are, I would have expected, amongst these
11 hundreds of short-term studies, that if there were a
12 major chronic effect that some -- there would have
13 been a more, it would have been a consistent subset,
14 particularly where there is a broad range of chronic
15 exposures, especially in the higher range, that we
16 would have seen some of this.

17 And I'm simply suggesting -- not that I know
18 the answer any more than you do, but it's not a
19 consistent argument to say that this is linear, and
20 then to say, well, we can't tease out this because of
21 this underlying chronic effect. We would expect
22 something else there.

23 And even if you don't buy that argument, I
24 would still say that the bulk of the data that we have
25 is for short-term studies, it's not long-term studies.

1 And, therefore, I think we are on surer footing about
2 where the effects may be and something about the
3 uncertainty.

4 PROFESSOR SHEPPARD: I'd like to underscore
5 my agreement with what Ira just mentioned. I mean, I
6 think the data actually in chapter seven, reviewing
7 epidemiology, include large volumes of data that
8 address the issue of short-term effects. And
9 furthermore, the bulk of the data suggests that there
10 are short-term effects, both for PM 2.5 and PM 10.

11 Despite the presentation this morning that I
12 couldn't really follow the logic of there being
13 differences, I think, in fact, that figure this
14 morning showed that both for PM 2.5 and for PM 10
15 there are short-term effects. And it's really
16 difficult to understand how the data in this chapter
17 would lead one to the conclusion that an annual
18 standard or an annual average somehow better reflected
19 the health impacts of particles.

20 The other thing I think that might help a
21 little bit in a rationale for setting a specific 24-
22 hour standard would be to reformat some of the data
23 that's presented in the document, because the data in
24 the document don't really allow one to identify the
25 24-hour peak exposures that lead to these effects. So

1 the data are all really based, the actual numbers that
2 are shown in the document are all based on annual or
3 even longer-term averages. And it would really help
4 one trying to make a decision about setting standards
5 to look at what the 24-hour peaks really were in
6 studies that showed effects or didn't show effects.

7 But I think if -- We talked about this a
8 little bit before, but I think if you had the data in
9 that way, it would be a lot easier to make a rationale
10 argument for how you picked a particular concentration
11 for a standard.

12 PROFESSOR THURSTON: Well, I can -- as a
13 person who has gone through all of these studies and
14 tried to summarize them, I can just tell you that
15 you're stuck with what the people report in the paper,
16 which sometimes is the interquartile range, sometimes
17 it's the maximum over the whole period, sometimes it's
18 the 98 percentile, it's never -- so it becomes very
19 difficult unless you get the actual data from the
20 researcher or have them -- you know, to know what the
21 distribution of the concentrations were for any of
22 these studies.

23 The thing you generally know, the thing
24 that's generally reported is the mean. So I think
25 part of the answer to your question is we see the

1 epidemiology and, you know, I didn't write this, but I
2 can see where they're going from this here, that
3 what's reported, what you have is the mean. So that's
4 what you've got to use. You've got to use what you
5 have, you can't use what you don't have.

6 And I think that partially answers your
7 question.

8 DR. LIPSETT: Okay, and if I could amplify
9 this, this is a big problem in general with the way
10 these data are reported. You have a mean
11 concentration that might be for two or three or four
12 years, and you see these relationships, say, for the
13 changes in the interquartile range you might see a
14 relative risk of whatever it is, you know, 1.04, 1.05.

15 Some of the reports do give an indication of
16 what's at the 95th percentile or what the maximum is,
17 and we actually did go through and look at a number of
18 studies that, say, had long-term mean concentrations
19 below 30 and looked at what the peak concentrations
20 were. And most of them were well above 50, for
21 example.

22 So, in terms of trying to identify from these
23 studies, well, where do these events, these short-term
24 events first begin to happen? I mean, is there some
25 way to try and identify that within the concentration

1 ranges that are reported and the answer is no at this
2 point. So that's one of the difficulties that we
3 face, then, in terms of trying to draw a line based on
4 this.

5 PROFESSOR SHEPPARD: I mean, for other air
6 pollution standards, what has generally been done is
7 to try to identify a level at which -- below which
8 you're confident there aren't effects and to set the
9 standard lower.

10 DR. LIPSETT: Right.

11 PROFESSOR SHEPPARD: Because that's what a
12 margin of safety is. But this document is really not
13 written in a way that allows you to extract that
14 information.

15 DR. LIPSETT: Well, it's not because we
16 wouldn't have wanted to write it that way. I mean,
17 obviously, that would make our task and yours much
18 easier. The problem is that the underlying data are
19 not presented and probably cannot -- or for people who
20 have tried to analyze it in that way, they have not
21 been able to come up with any kind of inflection
22 points in the exposure response records, which would
23 make this job much easier.

24 What you see generally are these linear --
25 Okay, I'm almost done with it --

1 PROFESSOR TAGER: Go ahead.

2 DR. LIPSETT: -- a linear kind of exposure-
3 response relationship without any clear kind of
4 delineation of a level below which we would be
5 confident that there wouldn't be any effects that
6 would occur.

7 Ira?

8 PROFESSOR TAGER: Yeah, I'd just like to make
9 a comment which is not a criticism of anything here,
10 but I think it's a flaw in the way the whole process
11 is done. I mean, normally, as was pointed out, the
12 way these estimates are made is you take the
13 interquartile range or the 10th and 90th values of the
14 levels, but what you really want is to change
15 distribution.

16 Because that's going to tell you what --
17 Assuming these models are correct, and they actually
18 are reflecting the real world, if you want to assess
19 the risk, you need to know, let's assume for
20 simplicity that there's a one-day lag and there's some
21 change per ten-unit PM. Well, how frequently does a
22 ten-unit PM change occur? You really need the change
23 distribution to figure out how to accumulate the risks
24 over any period of time, and we don't have that.

25 And so we're partly missing, at least in my

1 view, we're partly miss -- And this is not a --
2 everyone does this -- we're partly misspecifying the
3 risk because we don't have the right distribution.
4 It's never provided and I've never seen a paper where
5 people have actually given you the change
6 distribution, which from my point of view is what you
7 really need to know to assess the risk.

8 DR. BALMES: Just to underscore what Dean and
9 Ira already said, and not to be beating a dead horse,
10 as a non-epidemiologist with some interest in
11 epidemiology and collaborative experience, the
12 document reads in a way that there's a certain
13 disconnect between an emphasis on the hundreds of
14 studies that have shown "consistently elevated risk of
15 daily mortality and diverse measures of morbidity" --
16 I'm reading from page 163 -- "(such as hospital
17 admissions, emergency department visits for cardiac
18 and respiratory causes, exacerbation of asthma,
19 increased respiratory symptoms, restricted activity
20 days, school absenteeism, and decreased lung
21 function)," and over five continents.

22 And there is a disconnect between the
23 presentation of these data and then a lack of a short-
24 term exposure standard. And I do understand the
25 difficulty in setting a precise standard, but I don't

1 buy the argument that because it's difficult, we
2 shouldn't try to do it. Because for public health
3 purposes, I think we need to do it.

4 We won't get it perfectly right because the
5 data don't allow us to get it perfectly right, but we
6 should nevertheless try.

7 PROFESSOR CROPP: If I see this information
8 correctly, the average annual averages are the
9 consequence of repeated high-peak concentrations. At
10 other times there are concentrations that are probably
11 so low, at least in many areas, where we are not
12 concerned about health effects. But each little spike
13 in PM levels will produce a certain injury. When it
14 is below a measurable effect, it doesn't produce any
15 injury.

16 And so, yes, it is true that probably if we
17 have lots of spikes we will have a higher annual
18 average. But we may be in an area where 80 percent of
19 the time, air pollution is minimal. But there may be
20 three or four times a year, and I'm particularly
21 thinking of the area north of Sacramento, for
22 instance, where they are burning rice straw, where
23 there are extremely high concentrations for short
24 periods of time that may be very injurious, even if
25 the annual average doesn't come up to what you

1 consider a toxic level.

2 And so I think that particularly in children
3 is the repeated exposures to toxic levels of
4 pollution. And there are probably also enormous
5 regional differences, if the air pollution monitoring
6 station is two miles away from the refinery or from
7 wherever the toxic pollutant is produced, the children
8 that are living right in the vicinity of the source of
9 pollution will be injured a great deal and it will be
10 ignored, not acknowledged by the average or annual
11 pollution level that is recorded at a monitor that's
12 two or three miles away from there.

13 PROFESSOR SHERMAN: I had a question for
14 Dr. Tager. You were talking -- You dealt with the
15 issue of confounders, and I wondered if you could ever
16 get to -- and it's clear, you know, where there's a
17 real effect versus the other associated effects in the
18 issue of epidemiologic studies of air pollution.

19 Do you believe you can get there?

20 PROFESSOR TAGER: You mean where you can
21 provide someone with absolute assurance that some
22 confounder hasn't been left out?

23 PROFESSOR SHERMAN: Well, at least some
24 reasonable thing that we believe that .2 is due to
25 particulates, okay, and the rest is due to everything

1 else.

2 PROFESSOR TAGER: Well, I mean, I don't know
3 how you would do that in a real-world situation where
4 the number of exposures from things other than air
5 pollution -- food, water, etc. -- I don't know how
6 you'd parse that out. And, I mean, that's why we tend
7 to work on relative risk scales and not absolute risk
8 scales.

9 PROFESSOR SHERMAN: Right.

10 PROFESSOR TAGER: Because I don't think we
11 know how to quantitate absolute risk, so we use
12 relative risk, which is the best that you can do.

13 PROFESSOR SHERMAN: Right. Well, that
14 follows up what was a concern for me in the document
15 and I think overall, you know, a good job was done to
16 try to get in relevant studies. But there were
17 studies that were put in, like from Mexico City or the
18 one that was important for me as far as pregnancy,
19 they were kind of shock value. And the one I'm
20 referring to is on page 190 by Dejmek, and that has to
21 do with fetal growth and maternal particulate
22 exposures.

23 And that study happened to occur in a country
24 that was controlled by the former Soviet Union in an
25 area where there was lots of other groundwater

1 pollution, a lot of malnutrition and other things that
2 would affect pregnancy.

3 PROFESSOR TAGER: But there were studies from
4 the state of Washington and California now that have
5 found similar kinds of things.

6 PROFESSOR SHERMAN: Right, found similar
7 types of things, and that's what -- I think we should
8 focus on studies that are similar to what might occur
9 in California, okay, in the document rather than, you
10 know, which are really pointed and which will make
11 believers out of everybody that this is real and this
12 is likely to occur in California, per se.

13 Then in concert about having a short-term PM
14 2.5 standard for infants under a year of age, there
15 would be three really high subpopulations. One would
16 be pre-term infants with very serious chronic lung
17 disease that are very premature, they're likely to be
18 very susceptible; infants with congenital heart
19 disease, and infants with cystic fibrosis. All of
20 those are going to have even much higher breathing
21 rates and tittlebimes (phonetic) breathing and much
22 more of particulate matter than the average child.

23 And, therefore, over a few-day spike as
24 you're talking about, whether it be rice burning or
25 some other event, those children may be over the edge,

1 and either get a pulmonary infection or some other
2 event may occur which could result in an infant
3 mortality that would go unrecognized with an averaging
4 of a year exposure on a daily average basis.

5 CHAIRMAN KLEINMAN: I think that's a very
6 good point, and I think one of the things that I just
7 wanted to throw out as well is that we put a very
8 large emphasis on mortality in setting the standards.
9 But the number of cases of morbidity are much greater,
10 and I think one of the reasons, or there are several
11 reasons that we focus on mortality. One is it's very
12 easy to define, we know when someone is dead and it's
13 a very clear end point. Some of the other end points
14 that we measure are much less easily defined; however,
15 they probably occur at much greater frequencies and
16 especially for the very young people and the elderly,
17 they can have very debilitating effects.

18 I kind of take part of the blame for an
19 emphasis on mortality because when you start to put a
20 dollar value to the benefits of cleaning up the air,
21 there is an overwhelming amount of money that can be
22 attributed to saving a life. I forget what the actual
23 number is that EPA uses, but it keeps varying, but
24 it's over a million dollars a life. And that adds up
25 very quickly. Pretty soon you're talking about real

1 bucks.

2 Whereas putting a value on a case of
3 bronchitis or an episode of bronchitis or an episode
4 of asthma is much more difficult, and when you add
5 them up you don't come up to the same amount; however,
6 in terms of personal suffering, it may be very
7 important.

8 So I think that although we focus on
9 mortality, we really should look at the many studies
10 that look at the short-term effects on morbidity. And
11 especially effects in younger children, because I
12 think that will carry out through a longer period of
13 time over the course of their lives.

14 DR. OSTRO: We found that when you look at
15 the studies of adults and children, it looks like the
16 effects are occurring basically at the same level, so
17 there's not evidence that you need to go to a lower
18 level to protect children. The studies seem to be
19 pretty consistent that if you're protecting or not
20 protecting one group, you're going to get the same
21 effects for the other groups. That's one point.

22 But also, I wanted to just respond a little
23 bit to Ira about the development of the long-term
24 standard. I think you might have said that we only
25 used two studies, but --

1 to do with the quality and quantity of the data
2 available to you, one, to come up with an aggregate
3 estimate, but equally important to me is the
4 uncertainties that are involved. And if you're going
5 to use -- And that, I guess, is part of my problem
6 with using confidence intervals for this.

7 If you want to use these, then I think the
8 uncertainty analysis has to look across the effect
9 estimates and say, okay, here are the range of effects
10 given the various studies, because they're not all
11 consistent. I mean, Adventist study finds effects for
12 lung cancer but it doesn't find it for overall
13 mortality, etc. So I think that that has to enter
14 into the uncertainty analysis.

15 I don't think that in any way you presented
16 the data unfairly, that wasn't my comment. I'm just
17 basically saying that I think there are really only
18 two studies, which I still have problems with because
19 of all these cohort phenomenon cumulating effects
20 which really allow one to get straight at the issue.
21 That was the point.

22 DR. BALMES: I mean, just to say it more
23 simply, I think, the Harvard Six-City Study published
24 in the New England Journal of Medicine and the
25 American Cancer Society study published in the

1 American Journal of Respiratory and Critical Care
2 Medicine are the two longitudinal respective cohort
3 studies that much of your thinking seems to be based
4 on versus the many short-term studies, the literally
5 60-plus that you have in here.

6 So, given that there are only two studies
7 that Ira thinks are worth discussing in this regard --
8 I mean, I shouldn't, that's overstating -- but the two
9 studies that I mentioned and given that there are some
10 uncertainties related to those two studies, it seems a
11 little bit of a stretch to base a lot of the thinking
12 and logic behind standard setting on just those two
13 studies.

14 PROFESSOR CROPP: If I may, Michael, I would
15 like to take issue with what you said about the cost
16 of morbidity versus mortality. I think there are
17 actually quite examples that once you're dead you
18 don't cost any more. And that's sort of final,
19 finality. But if you keep that person alive, that
20 patient is going to cost a great deal more.

21 Similarly, if children lose 300- or 400,000
22 days of going to school or being ill, that costs a
23 great deal of money to the parents that can't go to
24 work, and the lack of education that has occurred
25 during these many days. And there is no question

1 there is evidence that chronically ill children don't
2 do as well in competing for university entrance and so
3 on compared to healthy children, because they lost a
4 lot of time from school.

5 And if you look at the cystic fibrosis
6 children or the BPD children that are chronically
7 affected by their early disease, these children often
8 lose weeks and months a year from school and their
9 parents lose weeks and months from going to work. And
10 consequently, there is an enormous cost to morbidity.
11 And I would challenge that the cost of morbidity may
12 be more than the cost of having your life shortened by
13 one and a half years.

14 PROFESSOR TAGER: But isn't partly beside the
15 point? We're supposed to be evaluating the data, such
16 as they are. Admittedly, what the implications are is
17 another set. And the argument is, on one side we have
18 a ton of studies and on the other side we have a
19 couple, and where do we think we get the most precise
20 estimates of effect and the best uncertainty estimates
21 about the range of effects.

22 It seems to me that's the critical question
23 that has to be answered.

24 DR. LIPSETT: Ira, can I interrupt for just
25 one second here? Because I think we need to respond

1 to what you and John have said about there only being
2 these two studies that have entered into the
3 development of the chronic standard.

4 In Bart's presentation this morning he
5 mentioned that, and it is described in the document,
6 in section (K), these are important, there is no
7 question about that, but there are a number of studies
8 of chronic morbidity with long-term means that those
9 were looked at as well, and they go from I think 21 to
10 35 micrograms per cubic meter for --

11 PROFESSOR TAGER: Morbidity or mortality?

12 DR. LIPSETT: Okay, no, I don't dispute that.

13 PROFESSOR TAGER: Okay. Again, while these
14 two studies are important, the studies of chronic
15 morbidity, a number of them which are summarized in
16 the document were also evaluated in terms of where we
17 would put the annual standard, looking at the long-
18 term means of these standards of chronic morbidity.

19 In addition, the long-term means of the time-
20 series studies we talked about which Dean had
21 mentioned before in terms of trying to -- if we could
22 look at the peak concentrate -- we basically, we
23 mainly have mean values for these. The long-term
24 means of the time-series studies where we looked at
25 acute events, those are also incorporated into the

1 evaluation where the levels of the annual standards
2 ought to be.

3 Nonetheless, you're correct in that there are
4 these two cohort studies that were important, but that
5 was not by any means by the sole basis for the
6 decision of the annual standards.

7 DR. LIPSETT: No, I wasn't -- Let me just
8 clarify. It's not so much -- It's also the emphasis
9 on the mortality part. I mean, I understand the
10 morbidity studies are there and I'm not disputing, and
11 I'm not necessarily even disputing the arguments made
12 relative to the means in the short-term studies, that
13 that's not relevant.

14 What I'm suggesting is it doesn't make sense
15 to talk about a long-term standard, that was the
16 original point I was making, and not about a 24-hour
17 standard when you have all these data which allegedly
18 are measuring changes over very short periods of time.
19 I'm not disputing the long-term standard, I'm
20 disputing the emphasis on mortality, which is based on
21 a relatively small number of studies -- We can argue
22 whether it's two or three or what -- I agree that you
23 definitely pointed out the morbidity data, I'm not
24 disputing that, and I don't argue, I don't disagree
25 with your argument that you can make inferences about

1 the means from the short-term studies.

2 But that doesn't explain to me why there
3 isn't 24-hour PM 2.5 standard, given the other data as
4 I mentioned before.

5 DR. BALMES: And let me just clarify also for
6 the record that again, I was referring to the lack of
7 a short-term standard in my last comments. I actually
8 support your evaluation of the data with regard to the
9 annual standard.

10 PROFESSOR TAGER: As do I, I just -- He
11 summarized exactly my position as well.

12 PROFESSOR THURSTON: I think we all accept
13 that there's an acute and a chronic component to the
14 health effects of air pollution. And I think the
15 critical question that maybe the committee can help
16 the state here with is how to go about, what's the
17 best way to go about setting that standard. You have
18 these studies, how do you use these studies. You do
19 have many studies of acute associations, you know,
20 associations between acute exposure and adverse health
21 effects. How do you then take those studies and set
22 the short-term standard?

23 I think everybody would acknowledge that,
24 given that we accept that there are both effects of
25 chronic exposure and effects of acute, you should have

1 both. But then how do we set a defensible standard
2 for the short-term? That's the question that's out
3 there.

4 PROFESSOR TAGER: Well, the first question,
5 it would seem to me, would be to ask how was it done
6 in the past with those situations when short-term
7 standards were set. And so that would be the first
8 thing was the evaluation for that and acceptable
9 method for doing it.

10 The second, then, would be to sit down -- I
11 mean, I'm not going to offer the answer now because I
12 don't know it without sitting down looking at the data
13 and thinking carefully of what the possibilities are
14 for how to come up with it. But certainly, there's
15 historical precedent, given that there are 24-hour
16 standards for other pollutants. So I think the first
17 place is to try to reassemble that historical
18 precedent and see to what extent it applies to the
19 database that exists now, and then to sit down with
20 the data and think of alternative strategies for
21 coming up with that standard.

22 But I don't think you can avoid it. I mean,
23 somebody made the comment just because it's -- I think
24 it was John, just because it's hard we shouldn't try
25 to do it. I agree it, it's hard. But I don't think

1 you can avoid it, when the bulk of the data speak to
2 that question.

3 I agree, it's not immediately
4 straightforward, and I'm not prepared to offer an
5 answer that I'd regret having said.

6 [Laughter]

7 CHAIRMAN KLEINMAN: Dean?

8 PROFESSOR SHEPPARD: But I would like to
9 underscore something Ira just alluded to, I think,
10 indirectly, that for -- This is an example where we
11 have much more data to choose from to make a rational
12 decision about standard setting than almost any other
13 example of a regulated air pollutant. There are more
14 dramatic, more consistent and a larger number of
15 studies demonstrating an effect of 24-hour peak
16 concentrations at about 2.5 particles and PM 10
17 particles than for any of the other regulated
18 pollutants that the state or federal government has
19 regulated.

20 So creative analysis of this data set gives
21 you a much greater opportunity than people have ever
22 had before to set a rational standard. And so that's
23 not really a reason not to set a standard, because now
24 we have more information than we did before.

25 CHAIRMAN KLEINMAN: Well, I think, if I

1 remember right, Bart mentioned that one of the reasons
2 for not setting a short-term standard was the
3 difficulty in identifying a bright line; is that
4 correct? Yeah, and it might be useful, maybe what we
5 ought to do is -- We're scheduled to take a break --
6 take a break, and then perhaps Bart can sort of
7 revisit that issue and, you know, amplify on why it's
8 difficult to set that bright line.

9 So why don't we adjourn for a brief break. I
10 think we were scheduled for what, about a 15-minute
11 break? Okay.

12 (Thereupon, a recess was
13 held off the record.)

14 CHAIRMAN KLEINMAN: We are going to address
15 the standards and issues tomorrow again, so I think
16 what we'll do is move on with a discussion of the
17 scientific aspects of the document.

18 And I think the next thing scheduled -- Well,
19 first, does anybody else have any other comments on
20 epi for now? If not, we should move on to the
21 toxicology, and Dean?

22 PROFESSOR SHEPPARD: I was asked to lead off
23 on the review of the section on mechanisms. And I
24 thought, just to start off, that the document did a
25 fair and reasonable job of summarizing the information

1 that's available on mechanisms. And this is actually
2 one of the more challenging issues to address. But
3 given the degree of uncertainty about mechanisms, I
4 thought that the amount of space in the document that
5 was taken up with this topic was also appropriate,
6 even though it wasn't a very long section.

7 This has been one of the major challenges, I
8 think, in the field, that the epidemiologic data, as
9 we discussed earlier, is overwhelming and convincing
10 for effects of particles. But the laboratory
11 scientists haven't yet been clever enough to design
12 experiments to identify what components of these
13 particles under what conditions are actually causing
14 these in vivo effects in the field.

15 And I think that that state of the art was
16 fairly reflected in the discussion. It was
17 appropriately pointed out that there were some
18 experiments that show effects on lung inflammation
19 under varying circumstances. As Dr. Friedlander
20 pointed out earlier today, many of the experiments
21 involved pretty non-physiologic challenges to animals,
22 injecting material directly into the trachea and then
23 looking for inflammation. And obviously, that's not
24 modeling perfectly well what happens in the
25 environment, and it's not surprising that the

1 concentrations required to produce effects in these
2 circumstances are much higher than the concentrations
3 of what would infer as producing effects from
4 epidemiologic studies.

5 I think that a good job was done of reviewing
6 the literature about potential cardiovascular effects,
7 and I think the authors of the document did a nice job
8 of really not putting their money down on saying that
9 any of those experiments really proved how these
10 particles were causing toxicity in the real
11 environment. Similarly, the discussion of potential
12 neural mechanisms was I think appropriately skeptical.
13 And really, the bottom line is that we really don't
14 understand -- And I think that this comes across in
15 the document -- we really don't understand how these
16 particles make people sick.

17 And obviously, when we discuss later on areas
18 where more research is needed, it's pretty clear that
19 we could do a more intelligent job eventually, one
20 would hope, of making rational decisions about these
21 particles when we have a better idea about why they
22 make people sick. But the fact that we don't
23 understand why they make people sick really doesn't in
24 any way detract from the overwhelming mass of
25 epidemiologic data suggesting that that's the case.

1 There's one very minor point that was
2 incorrect in the document is there was a discussion of
3 rats treated with monochrodolin as a model of
4 emphysema -- John Balmes pointed this out to me and
5 then I noticed it in the document -- and that's
6 actually a model of pulmonary hypertension.

7 DR. BALMES: I would also second Dean's
8 overall comments that I think that this is fair, and I
9 mean that in a very positive sense -- equitable
10 assessment of the voluminous literature on potential
11 toxic mechanisms for PM, none of which really have
12 proved definitive yet.

13 One comment that I would make, and it's
14 already been alluded to, is the discussion of effects
15 on cardiac autonomic nervous system and potential
16 arrythmia induction is good, and I know that because
17 Dr. Lipsett and Dr. Ostro know this literature well,
18 but I would just use that good discussion to
19 underscore a point that I don't believe that changes
20 in heart rate variability and increased arrhythmias
21 are primarily due to a chronic effect of PM exposure.
22 I think it's much more likely to be acute effects.
23 You know, it just highlights a point I've already
24 made, that I think there should be a short-term
25 standard.

1 CHAIRMAN KLEINMAN: I thought that the
2 summary of mechanisms lead to a very nice finding in
3 that it very strongly supports that there are
4 biologically plausible mechanisms by which inhaled
5 particles can have effect. I do want to second John's
6 point that in some of the findings, there is a -- I'm
7 trying to find the page again, but they find
8 significant associations between symptom onset in
9 patients with myocardial infarction, on page 157, and
10 short-term effects or short-term PM 2.5 exposures.

11 And I think that theme is carried out through
12 the section on cardiovascular effects, that there does
13 seem to be a short-term effect that does seem to be
14 very important in that form of morbidity. Having said
15 that, I think that the paragraph that's written in the
16 summary section on biological plausibility --

17 PROFESSOR FRIEDLANDER: What page is that?

18 CHAIRMAN KLEINMAN: Hold on, let me look --

19 PROFESSOR FRIEDLANDER: 167.

20 CHAIRMAN KLEINMAN: Yes, Biological
21 Plausibility of the Associations, page 167 -- is
22 rather short and very noncommittal, and I think it
23 could be strengthened a little bit.

24 Because I do think that that association is
25 now becoming more relevant, and certainly strengthens

1 our feeling that we do need to improve our standards.

2 Open to other questions or comments?

3 PROFESSOR FRIEDLANDER: On the -- Reverting
4 to section 7.8.2 on page 152, the Pulmonary and
5 Systemic Inflammation, there were studies there on, as
6 I stated before, on the exposure of tissue by many
7 different configurations, so to speak; that is,
8 installation, the use of collected samples, in vitro
9 studies, inhalation studies, human inhalation, animal
10 exposure. And as someone who has an interest in the
11 transport mechanisms and articles to tissue at risk,
12 so to speak, this seems to me to be difficult to
13 follow.

14 I would think that it would be better to
15 group these studies into those different areas; that
16 is, installation studies. To the extent that there
17 are subgroups, discuss studies that were done by
18 inhalation, separating animal and human; and then
19 installation studies and in vitro studies. And, for
20 example, in the in vitro study that's mentioned in
21 line 13 of that section, 7.8.2, it mentions high-level
22 exposures, but I can't tell what's meant by that.
23 Perhaps people who are working directly could say.

24 Is that high-level in comparison to what an
25 inhalation study would involve or atmospheric

1 exposure? In any case, I couldn't understand what
2 that was, so since I think these are significant --
3 that is, the whole issue is significant -- I think it
4 would be, I think this should be reorganized and
5 grouped together in a more accessible fashion.

6 CHAIRMAN KLEINMAN: Other comments?

7 DR. LIPSETT: Okay. Having written this
8 section, I guess I should respond to all of these
9 comments on it. And I actually thought, based on some
10 of the comments that we received from the public, that
11 even though I've tried to be tentative in describing
12 these mechanisms, that I would even be a little bit
13 more tentative for some of the conclusions here.

14 Because, like with respect to some of these
15 very high-level exposures, I mean, they're very -- I
16 mean, they're unphysiologic, they're not
17 representative at all of what you would see in the
18 ambient environment or even, in some cases,
19 occupational types of exposures. They're exceedingly
20 high types of exposures in vitro, for example.

21 And we could certainly include information
22 here indicating what those levels were; that's not a
23 problem to do that sort of thing if that would make it
24 more useful. I think that their main value lies,
25 though, in being able to look at some of the changes

1 that -- You couldn't expose people to these sorts of
2 levels experimentally, for example, or even in some
3 instances the kinds of toxicity we'd see in animals
4 would obscure the kinds of end points that you might
5 be interested in looking at here.

6 With respect to the biological plausibility
7 part of the causation argument, which, Mike, I guess
8 you had commented on, we could expand that certainly
9 to include a little bit more. But again, I think
10 overall, while toxicology has begun to make some
11 inroads into mechanistic understanding, we're far from
12 really having a good idea about what's going on.

13 I mean, the data here, as I've indicated in
14 this section, are not entirely consistent from
15 experiment to experiment or epi study to epi study,
16 but they do give a sense that inflammation in the lung
17 is important, and that there are certainly systemic
18 consequences of that. But how this might happen,
19 based on the kinds of exposures that people
20 experience, you know, we're very far from
21 understanding at this point.

22 PROFESSOR CROPP: I would just like to make a
23 general comment. I think anytime that there is
24 inflammation, there must have been injury. I think
25 inflammation is the basic pathological response to

1 injury regardless of the insult.

2 So if we can document evidence of
3 inflammation, there must have been injury. And there
4 has to be healing, and if it is recurrent, there may
5 be scarring. And certainly, there is the set-up for
6 lung injury.

7 DR. LIPSETT: Yeah, I would certainly agree
8 with that and we do have some controlled exposure
9 studies of humans where these inflammatory changes in
10 the lung have been demonstrated. But the exposure
11 concentrations have been more what you would encounter
12 in occupational environments or, say, at busy traffic
13 intersections than necessarily what you would see in
14 large regions of California.

15 But having said that, we have documented or
16 experimentals have documented that these kinds of
17 inflammatory changes occurred, that, I agree, they're
18 kind of a stereotype response to injury of some kind,
19 injury or infection.

20 CHAIRMAN KLEINMAN: Any further discussion of
21 the mechanisms? If not, we should move on to the
22 monitoring issues.

23 Costas, would you take a lead on that,
24 please.

25 PROFESSOR SIOUTAS: This is actually a brief

1 and very straightforward sampler in this report. In
2 fact, Mr. Cook did a very fine presentation in the
3 morning that pretty much explained everything that
4 there was to explain in this. But the chapter
5 basically describes the FRM's of the existing
6 monitors.

7 But, more importantly, it discusses some of
8 the problems that all of these standard methods have,
9 whether these problems are volatilization of labile PM
10 species over long sampling or absorption. We talk
11 about artifacts, and finally, both today in the
12 presentation as well as in the draft, there is this
13 acknowledgment that we should introduce more different
14 types of monitors and should actually embrace the
15 emerging array of new continuous monitors that will,
16 literally will provide more accurate data I think and
17 less labor-intensive data.

18 But also, these monitors will, in fact, point
19 to sources whose time scales fluctuate in time periods
20 that are substantially shorter than 24 hours, and that
21 will help the state and ultimately the federal
22 government implement more effective control
23 technologies and control strategies.

24 I wanted to make sure that I point out one
25 thing about the high-volume method that is used, as

1 Mr. Cook pointed out. They're using quartz filters.
2 The rationale for that now is that, in lieu of glass
3 fibers, that there wouldn't be any SO2 absorption,
4 which may have been an obvious problem in the previous
5 data, which, of course, in and of itself, raises some
6 questions about what did the previous data collect
7 over the last couple of decades.

8 One other thing I would point out about
9 quartz substrates is that they're terrible when it
10 comes to absorption of organic vapors. So you're
11 going to have, depending on the season and the time
12 and the place you are in the basin, especially in the
13 South Coast basin, you might have anywhere from six to
14 ten micrograms per cubic meter added on the filter,
15 just by absorption of organic vapors under those
16 quartz substrates. So you definitely want to be aware
17 of that, particularly because you have those high
18 vols, using quartz substrates, and then you have the
19 low-volume filters, the new FRM's, using teflon
20 substrates, which wouldn't have this problem.

21 Another thing that I would certainly implore
22 you to do in your new implementation of continuous
23 monitors is to also favor monitors that measure
24 separately quartz from fine particles. If you look,
25 for example, on page 55 of your report, there is a

1 graph that shows the ratio of PM 2.5 to PM 10, at
2 different locations in California.

3 And you're going to see this immense seasonal
4 variation. You're going to see that fine particles,
5 not surprisingly, are 70, 80 percent of the total PM
6 10. During the winter months you have stagnation.
7 You don't have any winds, you know, all of those
8 factors that would have suspended coarse particles.

9 And in determining the coarse concentration
10 by difference, you're essentially subtracting, you
11 know, two large numbers, and that will lead to major
12 errors. So if there are ways, and there are, out
13 there to measure separate coarse from fine particle
14 concentrations, it's certainly something you should
15 consider.

16 I believe this is most of my -- We've already
17 talked about implementing new continuous monitors, so
18 I'm not going to revisit this. In my view, there are
19 a lot of wonderful technologies that are out there
20 today. I'm committed to forwarding to you all of our
21 supersite data, which now are, in fact, evaluating all
22 of these continuous monitors. That's a major
23 component of this effort, so we've had all of these
24 arrangements before and I'm committed, of course, to
25 sharing all this data with you.

1 CHAIRMAN KLEINMAN: Sheldon, would you like
2 to comment?

3 PROFESSOR FRIEDLANDER: Yes, just to revert
4 to another point that I had made earlier about the
5 potential for having the sulfur dioxide and hydrogen
6 peroxide that are present separately in the gas phase
7 being absorbed by liquid, either liquid that's present
8 due to fog droplets or from sources of that kind, or
9 from associated with accumulation mode aerosol. It's
10 well known that, especially in high humidities, that
11 there's quite a bit of water present in the .1 to 2.5
12 micron component of the aerosol.

13 If that's deposited in the filter, it can
14 serve as a site for a parcel of gas that follows, say,
15 that's rich in SO₂, for the SO₂ to dissolve in that.
16 And then another parcel to come through with H₂O₂, the
17 SO₂ might come from a refinery; the H₂O₂ might come
18 from vehicular emissions and photochemicals, and the
19 two might then react in the filter and produce that
20 artifact of a high sulfate concentration.

21 Because that's the way the sulfates form in
22 the atmosphere, it's by -- the general consensus is
23 that it's formed by SO₂ and H₂O₂ dissolving in droplets
24 in the atmosphere. So the bottom line is that there
25 are other instruments now which involve flash

1 vaporization, and don't require the accumulation of
2 aerosol over a period of hours to measure
3 concentrations.

4 CHAIRMAN KLEINMAN: I guess I wanted to just
5 again raise the issue, I wanted to congratulate ARB on
6 doing the evaluations of the continuous monitors, and
7 hopefully they'll be able to identify a monitor that
8 is suitable for field work and will produce good
9 continuous measurements of PM 2.5 and PM 10. Because
10 it, I believe, will be crucial in eventually being
11 able to better define where standards ought to be for
12 the future if not, you know, if we don't have that
13 capability now. So I was very happy to see that as
14 part of the report.

15 Any other comments?

16 PROFESSOR THURSTON: I think that the points
17 that were raised about the continuous methods are very
18 good, but I really want to, from my own personal
19 perspective, point out that we don't want to move
20 entirely away from filter collection methods where you
21 actually can collect the particles and then analyze
22 them later. And I don't think that's what's being
23 said here, but I just want to stick that caveat in.

24 I can just give you an example, where we,
25 with the World Trade Center disaster, NYU, our team

1 went in and located samplers for an array of
2 collection methods on filters. And what EPA did, and
3 this is not a criticism of EPA, but they set up TEOM's
4 around the site, and so on. So they were able to get
5 hourly measurements of what the TEOM measures, for
6 better or for worse.

7 But they have nothing collected to analyze
8 now. They know what the mass was, but they don't know
9 what was in it, whereas we have all these samples that
10 we've collected over many, many months twice a day on
11 filters and so forth, and now people are saying, oh,
12 you have those filters, do you think maybe we could
13 get a hold of those filters or maybe you could analyze
14 those and find out what people were exposed to as a
15 result of this disaster?

16 So I think that we should always remember to,
17 you know, have our baseline filter method so that
18 gives us the opportunity to look at constituents and
19 learn new things, while, you know, obviously moving
20 forward with new methods that give us more
21 information.

22 PROFESSOR SIOUTAS: One last thing? Can I
23 make one remark?

24 CHAIRMAN KLEINMAN: Sure.

25 PROFESSOR SIOUTAS: Thanks. I agree with 100

1 percent of the comment that we also need substrates so
2 there can be chemical analysis. I would like also at
3 some point to see this state's and every other state's
4 monitoring network to address the issue of not just
5 exposure assessment, which is one rationale for
6 measuring the other concentrations, but at some point
7 dose. And to do that, you need more information on
8 particle size.

9 The reason I'm saying that is, you know, all
10 of us lump PM into one broad category, PM 2.5, you
11 know, from 0 to .5 micrometers. We've done studies in
12 Los Angeles, we see the population living in downtown
13 are exposed to an aerosol that -- whose mass in
14 diameters is about .2 micrometers. We see the
15 population in Riverside being exposed to an aerosol
16 whose mass in diameter is .8 micrometers. These are
17 two very different aerosols, in terms of surface area,
18 in terms of deposition in the lung, in terms of the
19 ultimate dose.

20 And I think that information, in my
21 view, is -- I mean, it has to be important, ultimately
22 when it comes to assessing health effects. So at some
23 point I would like to personally see some of these
24 size fractionated monitors being out there.

25 CHAIRMAN KLEINMAN: Any other comments?

1 MR. COOK: Thank you. Thank you for those
2 kind comments too about the work that our staff did.
3 We realize that we're not bringing forward the perfect
4 sampler, and we wish that we could.

5 We do think we're bringing forward some
6 improvements in some particularly critical areas, and,
7 building on the comment about maintaining some of the
8 substrate samplers, we're quite confident that a good
9 number of those samplers will remain until money flows
10 to support any new samplers that we may designate.
11 Things won't move that rapidly, despite our best
12 wishes and despite whatever we may designate, and
13 that's about as much as I'm going to say about money.

14 [Laughter]

15 MR. COOK: I wish that we had data to show
16 you about the continuous samplers today. We are being
17 particularly stingy with that until the study is over
18 to try and avoid any sense of -- particularly vendor
19 involvement and possible vendor influence to the
20 study. This is one of the aspects that EPA has been
21 very forthcoming in saying that they liked this part
22 of the study. Vendors have literally not set foot in
23 the Bakersfield site, with the exception of one when
24 they had a major malfunction and the instrument
25 virtually went flat on them. But short of that, they

1 have no idea what their instruments have been doing
2 for the past three months or so, and they won't until
3 it's over.

4 So it's around the corner, and I wish we
5 could have data to present to you today, plus this
6 space. We do have a fair number of plans to collect
7 filters of a variety of particle sizes, whether it's
8 in this program, the PM 2.5 program and the federal
9 auspices. The federal program has a constituent
10 aspect to it, the speciated PM 2.5, so we will have a
11 variety of far more sophisticated measurements for
12 some very critical PM parameters for PM 2.5.

13 It's a separate instrument from the
14 compliance instrument, and that's where we find
15 ourselves oftentimes is you have to have two
16 instruments in order to get what it is you really
17 want, whether it's greater time resolution, greater
18 chemical speciation or what. So that provision will
19 be provided for under the federal program.

20 We have also an air toxics program, where we
21 collect 24-hour filters about every 12 days, then we
22 look at a variety of metals, these would be TSP
23 metals, so we have a good bit of that. And I really
24 do believe that the site-selective inlet sampler that
25 we have will not decrease appreciably in size, that

1 network over time. And off that we do get the
2 principal constituents of PM. You could almost
3 reconstruct the mass if you had just the constituents
4 alone on that.

5 CHAIRMAN KLEINMAN: Well, if there are no
6 other comments, I think we've looked at the scientific
7 issues as summarized in the document, and the comments
8 of the committee are going to be compiled and supplied
9 to the authors for them to respond and perhaps modify
10 or amplify sections of the report.

11 Tomorrow we're going to start out with the
12 oral public comments. That will start at 8:30 through
13 10:00 o'clock. Those who wanted to make a statement I
14 believe have already signed up, and we will use that
15 90-minute period as equitably as possible so that
16 everybody will get at least a chance to present a
17 summary. Most people have provided written comments,
18 some of them are quite extensive, but at least they'll
19 be able to present a summary of the key points of
20 those.

21 And all of the written comments have been
22 supplied to the committee. They will be reviewed by
23 us, reviewed by the staff, and our committee will
24 review the staff's response as well as respond to them
25 ourselves as part of the overall process.

1 Yes?

2 PROFESSOR SHEPPARD: I just want to point out
3 to you we do have copies of the written comments, and
4 we've put them outside on the outside table. So
5 anybody in the public or any of the committee members
6 that didn't have their copies, we can make those
7 available to you.

8 CHAIRMAN KLEINMAN: And then for those who
9 don't really want to read the written comments, there
10 will be a summary presented -- I guess, I don't know,
11 Bart, are you going to do that, a summary of the key
12 points? And brief responses from the committee and
13 from the staff.

14 And then in the afternoon the committee will
15 present a summary of the findings of the committee on
16 the report, and comments about the scientific basis
17 for the recommendations. So we'll have a pretty full
18 day tomorrow as well.

19 So at this point, I think that we can
20 adjourn --

21 PROFESSOR SHEPPARD: Mike, could I say
22 something before we adjourn?

23 CHAIRMAN KLEINMAN: Yes.

24 PROFESSOR SHEPPARD: I held back because I
25 only had a perspective to offer on the biology and

1 pathology. I thought it was a great review, and
2 whatever I had to say had been said.

3 But there is one perspective which I would
4 like to have an opportunity to relate to everybody,
5 and that is that the findings that were presented
6 correspond very well with what my special interest is
7 in pathology, and that is things called sudden death
8 and unexpected death, and, above all, a subclinical
9 state which I have termed morbidity.

10 And what it says is mortality is the tip of
11 an iceberg, morbidity is a little bit more above
12 water, but what's below the surface is the great
13 magnitude of disease, as, for example, emphysema,
14 where you can lose 75 percent of your lung before you
15 come to medical attention. Now, you start complaining
16 and the doc says, well, you've irreversibly lost 75
17 percent of your lung. John may comment on this, but
18 the point is that emphysema -- All adults have some
19 emphysema. I don't see any adult lung without
20 emphysema, with lung destruction.

21 So the point I wanted to make is that from
22 the perspective standpoint, I'm more concerned with
23 morbidity, subclinical disease, say, heart disease,
24 for example. I'm never surprised when somebody dies
25 suddenly with a heart disease because if they don't

1 have a cause, you've got a nicely built-in sign.
2 Everybody has coronary disease. So when they die and
3 you can't find a cause, it's called ASCVD.

4 Major occlusion of a coronary artery is very
5 common. I think somebody estimated that a half a
6 million joggers over the age of 35 had major occlusion
7 in one or more coronary arteries. This is on an
8 extrapolation of a clinical study. These are -- Now,
9 whether that's true or not, the point is morbidity is
10 an extremely important consideration and I had to take
11 that into consideration with my understanding of
12 whether this, these levels proposed were adequate or
13 in order.

14 And I just wanted everybody to know that the
15 concept of morbidity is totally overlooked. The
16 concept of lung decline is given too little attention.
17 Everybody is on the slope down with lung destruction,
18 and the problem is we have a disease, the question is
19 would ameliorating the levels of particulates
20 appreciably slow down the damage we are sustaining,
21 for some of us, a cause that's going to be very
22 serious and lead to death. There are going to be
23 people with lung disease with not enough oxygen, and
24 it won't be a coronary problem, it's going to be the
25 lung not feeding enough oxygen.

1 So this is, I think, a very important concept
2 and I just wanted to, if there were time, I just
3 thought it would be a nice, here at the end, to put in
4 the fact that morbidity should eventually be our
5 greatest concern, and politically speaking, the
6 greatest advance we're going to make is to get
7 information on morbidity, and that can only be done by
8 meticulously performed post-mortem examination, and we
9 don't do that now.

10 So until we get some good data, quantitative
11 data on the rate of decline of the lung structurally
12 as well as functionally, we're not going to have some
13 of the sharp answers we need for setting reasonable
14 standards.

15 CHAIRMAN KLEINMAN: Okay. Then we are
16 adjourned. See you tomorrow.

17 (Thereupon, the meeting was
18 adjourned at 5:05 p.m.)

CERTIFICATE OF REPORTER

I, JAMES RAMOS, an Electronic Reporter, do hereby certify:

That I am a disinterested person herein; that the foregoing Air Resources Board, Air Quality Advisory Committee Meeting was reported by me and thereafter transcribed into typewriting.

I further certify that I am not of counsel or attorney for any of the parties in this matter, nor in any way interested in the outcome of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand this 4th day of February, 2002.

James Ramos
Official Reporter

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