This verbatim transcript of the WTC Health Program Scientific/Technical Advisory Committee, Committee Meeting held telephonically on March 28, 2012, has been reviewed for concerns under the Privacy Act (5 U.S.C. § 552a), and personally identifiable information has been redacted as necessary.

THE U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL AND PREVENTION
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

convenes

MEETING TWO

WORLD TRADE CENTER HEALTH PROGRAM

SCIENTIFIC/TECHNICAL ADVISORY COMMITTEE

VOL. I

DAY ONE

WEDNESDAY, FEBRUARY 15, 2012

Jacob K. Javits Federal Building
26 Federal Plaza New York, NY

The verbatim transcript of the

Meeting of the Scientific/Technical Advisory

Committee held at the Jacob K. Javits Federal

Building, New York, New York, on February 15, 2012.

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TRANSCRIPT LEGEND

The following transcript contains quoted material. Such material is reproduced as read or spoken.

In the following transcript: a dash (--) indicates an unintentional or purposeful interruption of a sentence. An ellipsis (. . .) indicates halting speech or an unfinished sentence in dialogue or omission(s) of word(s) when reading written material.

-- (sic) denotes an incorrect usage or pronunciation of a word which is transcribed in its original form as reported.

-- (phonetically) indicates a phonetic spelling of the word if no confirmation of the correct spelling is available.

-- "uh-huh" represents an affirmative response, and "uh-uh" represents a negative response.

-- "*" denotes a spelling based on phonetics, without reference available.

-- (inaudible)/ (unintelligible) signifies speaker failure, usually failure to use a microphone.

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PROCEEDINGS

(12:08 p.m.)

WELCOME

DR. MIDDENDORF: Good afternoon. If the committee members will come to the table, appreciate it, we'll get started. I have a few administrative details that we need to take care of here at the beginning. I'd like to extend a warm welcome to the members of the public who are here in the room and also those who are on the phone. We very much appreciate your interest in these proceedings and look forward to your participation. For those who have signed up who would like to make comments, we do have public comments scheduled to begin at 3:45 this afternoon and then we'll have another public comment session tomorrow morning.

For those of you who are here in the room, I'll point out the emergency exit routes. If you look around the room, you'll notice that there are three doors that have exit signs above them. You need to ignore two of those exit signs. The exit sign back here behind me to the left is not an exit door. Please don't go out that way.

The double doors in the back far corner of this room are not exit doors. Please do not go out of those either. If, for some reason we need to evacuate the room, this door that's about three quarters of the way down here on my left is the door to go out. And the quickest way to get out is when you go through that door, turn to your right, go until you see two double glass doors on your left. Go through those double glass doors, immediately turn right, go down that hallway, and you'll see a door that says Fire Exit on it, and that's the way you get out of the building. So please, that would be the best way to do it.

For those of you on the phone, I suggest that you look around, figure out the evacuation route for your buildings. I need to point out that we do have copies of the agenda for this meeting. They are on the back table, and they're also available on the committee's website for anyone who is on the phone. You can download the agenda from our website. We also have copies of the public comments that were received as of about 11 on February 13th. They have been offered, filed to the committee before the meeting, and they're here on the back table. If you don't want to haul around a lot of paper with you, these comments will be posted on NIOSH's docket, which is docket number 248 for this committee and that's also available through the committee's website.

We need to do a quick roll call, and so we'll go around the table first and I'd ask each of the members to identify themselves and state whether or not there have been any changes in their employment or interest that would affect their conflicts of interest, and then we'll go to the members on the phone.

This is going to be a little difficult because we only have two working microphones.

MS. MEJIA: Good afternoon. Guillermina Mejia, no changes.

DR. QUINT: Julia Quint, no changes.

DR. ROM: Bill Rom, no changes.

MS. FLYNN: Kimberly Flynn, no changes.

MS. HUGHES: Catherine McVay Hughes, no changes. I'll bring the mic over.

DR. TRASANDE: Leonardo Trasande, no changes.

DR. MARKOWITZ: Steven Markowitz, no changes.

MS. DABAS: Valerie Dabas, no changes.

MR. CASSIDY: Stephen Cassidy, no changes.

DR. NORTH: Carol North, no changes.

DR. TALASKA: Glenn Talaska, no changes.

DR. ALDRICH: Tom Aldrich, no changes.

DR. HARRISON: Bob Harrison, no changes.

DR. WARD: Liz Ward, no changes.

DR. MIDDENDORF: Okay, and -- oh, I'm sorry.

MS. SIDEL: I'm Susan Sidel, no changes.

DR. MIDDENDORF: Thank you, and on the phone?

DR. DEMENT: John Dement, no changes.

DR. WEAVER: And Virginia Weaver, no changes.

DR. MIDDENDORF: Okay, thank you all very much. To those of the members who are on the phone, please let me know when you leave and when you return so we can be certain that we continue to have a quorum.

Also, I want to remind everybody that there may be some topics which come up that present a conflict of interest for members. And when these topics come up, I'll ask each of the members to state that they are recusing themselves so we have that on the record. That's just the best way to handle that.

I also ask everybody to -- we have a couple of issues; one is the microphones. We only have two microphones available in this room.

Tomorrow we will be moving into conference rooms A and B, so we'll have more microphones in there. We're going to leave this microphone turned on so we don't have that problem with the lag time that we had before, and then we'll just pass it around. I just wanted to point that out.

One of the microphones will be up at the podium until we're done with presentations, or if presenters want to present from their table, they can do that and we'll just give them that one from the podium. I think that's all I need to handle right now, so I will turn this over to our chair, Dr. Ward.

DR. WARD: Good afternoon. The first speaker today will be Dr. John Howard. He will give us introductory remarks.

INTRODUCTORY REMARKS

DR. HOWARD: Can you hear me? Good afternoon. Welcome to the second meeting of the Scientific Technical Advisory Committee for the World Trade Center Health Program. It is with sadness that we begin our meeting. Today, not only noting the passing of responders and survivors since September 11th, 2001, but also the recent passing of **[identifying information redacted]**, Professor of Preventive Medicine at the Mount Sinai School of Medicine.

For over 40 years, **[identifying information redacted]** treated, counseled, and fought for thousands of patients who were ill because of hazardous exposures in their workplace. As Co-director of the World Trade Center Worker and Volunteer Medical Screening Program at Mount Sinai, he was an early and prominent figure fighting for a long-term health program to identify and treat individuals who worked or volunteered at the former World Trade Center site.

For all of his tireless work on behalf of the World Trade Center Health Program during its earliest and most difficult time, we honor him and his service to his patients, to the City of New York, his country, and to all of us. Please join me in a moment of silence to honor the recent passing of responders, survivors, and **[identifying information redacted]**.

(pause)

I have four items for you today before we begin the meeting. The first item is the teleconference meeting on January 24th. I apologize for the technical problems which caused the cancellation of the 24th January teleconference meeting of the committee. We are taking steps to ensure there will be no repeat of the technical problems if the

committee should want to hold another teleconference meeting in the future.

Second, during this meeting, you will hear a report regarding scientific findings and support for establishing the statutorily required criteria for Pentagon and Shanksville responders. Commander Robert McCleery of the NIOSH Division of Surveillance, Hazard Evaluations and Field Studies in Cincinnati, Ohio has provided a report which you have already received and today will make a presentation regarding his research on the potential eligibility criteria for these groups of responders.

I want to thank you in advance for your consultation on this important issue. Please note that no formal written communication from the committee on eligibility criteria is required. The meeting transcript will suffice.

Third, I also appreciate the committee's continuing consultative thoughts on research needs for the World Trade Center Health Program. Your thoughts to date have been extremely helpful. And in addition to the formal research funding announcement from the NIOSH Office of Extramural Programs, the committee's views about important knowledge gaps and research needs will be placed on the World Trade Center Health Program's website for potential researchers to review.

Again, thank you in advance for your consultation on this important issue. Please also note that no formal written communication from the committee on research needs is required. The meeting transcript will suffice.

Fourth, as you continue your discussion of Petition 001 to add cancer or types of cancer to the list of World Trade Center-related health conditions, please keep in mind that the Zadroga Act in Section 3312(a)(6)(C) notes that the advisory committee must submit their recommendation on the petition to the administrator within 60 days or by a date specified by the administrator, not to exceed 180 days from the date of the administrator's request.

A request for a recommendation on Petition 001 was made to the committee on October 5th, 2011. The maximum 180-day period for the committee's consideration of Petition 001 ends on April 2nd, 2012. I had asked the committee to provide its recommendation by March 2nd, 2012, in order to provide enough time for the committee chair to prepare the committee's advice to the administrator.

However, since the opportunity for the committee to meet on January

24th, 2012, was cancelled, I would consider modifying the due date for the committee's recommendation. If the committee believes that more time is necessary to reach a recommendation, I would ask you to discuss that issue at this date and for the chair to send a written request to me for more time by the close of this meeting on 16 February.

Any additional discussion on Petition 001 after 16 February, 2012, must occur in another public meeting, so please keep in mind scheduling issues when determining whether additional time would be beneficial to the committee. In any case, the April 2nd due date for a recommendation is a statutory requirement; and therefore, no extension beyond April 2nd can be approved.

I thank you again for your service. I wish you a successful meeting.

RESEARCH NEEDS

DR. WARD: Okay. So, Rob McCleery has not dialed into the call yet, so we're going to go on and discuss research needs and then go to Rob when he dials in.

So, I hadn't really planned a lot of discussion around the research needs since I think you've all seen the letter that we prepared. But I didn't know if there were any topics that any of you wanted to discuss regarding the research needs or the conflict of interest.

Oh, sorry, he's just gotten on the line, so we'll proceed as planned with Rob McCleery's publication -- I mean presentation.

PENTAGON AND SHANKSVILLE, PA ELIGIBILITY

MR. MCCLEERY: I apologize for that. I didn't have this particular number, so I, again, I apologize. So, good afternoon everyone. Again, my name is Robert McCleery. I'm an industrial hygienist at NIOSH here in Cincinnati, Ohio. I appreciate the opportunity to speak with you this afternoon concerning the Pentagon and Shanksville, Pennsylvania responses to the terrorist-related aircraft crashes of September 11th, 2001.

Next slide, please. As it pertains to the Pentagon and Shanksville sites, the World Trade Center Health Program administrator is required, conditioned to other responsibilities to 1) determine the end dates of cleanup at both sites and 2) determine eligibility criteria relating to an increased risk of developing a World Trade Center-related health condition resulting from exposure to airborne toxins, other hazards, or adverse conditions resulting from the 9/11 terrorist attacks.

In the following slides, I will provide information that addresses both of

these required determinations for the four responding groups listed in the Zadroga Act for the Pentagon and Shanksville sites: fire department employees, police department employees, recovery or cleanup workers and contractors, as well as volunteers.

Next slide. At the Pentagon, fire department personnel arrived on scene very shortly after the aircraft crashed. Personnel within the Arlington County Fire Department served as the incident commanders during the fire rescue phase of the response.

Numerous other fire departments responded to the incident by backfilling other fire stations or responding directly to the Pentagon. This was set into action by mutual aid agreements previously established between these fire departments.

On September 21st, Arlington County Fire Department transferred control of the site to the FBI. The site now entered into the crime scene phase of the response. At this time, one firefighter company, a technical rescue team, and paramedics remained at the site until the FBI turned it over to the Department of the Defense on September 26th or 28th.

The literature differs as to the date of transfer of this command. From September 26th or the 28th, the available literature does not provide any information as to what period of time fire department personnel were on site until the end of the demolition and cleanup phase of the incident on November 19th, 2001.

Next slide. The police departments. The lead law enforcement agencies on site included the Arlington County Fire Department, with jurisdiction of areas surrounding the Pentagon, Defense Protective Services, federal law enforcement agencies within the Pentagon, with jurisdiction of the Pentagon, and the FBI.

Many other police departments respond -- responded either at the Pentagon or by backfilling police stations, by way of the Northern Virginia Law Enforcement Mutual Aid Agreement or the Northern Virginia Sheriffs Mutual Aid Agreement.

The available literature indicates that the Pentagon response had a police department presence until the FBI turned the site over to DOD on September 26th or 28th, 2001. The literature suggests that while the Pentagon site was under DOD control, services typically provided by police departments were handled by military police or Defense Protective Service personnel.

However, the literature does not provide additional information as to what period of time police department personnel were on site until the end of the demolition cleanup phase of the incident on November 19th, 2001.

Next slide. The Pentagon response and initial cleanup of areas of the Pentagon surrounding the incident site as employees began returning to work on September 12th, 2001. The demolition cleanup of the incident site itself was delayed until after a memorial service recognizing the one-month anniversary of the 9/11 attack on October 11th, 2001.

The demolition and cleanup activity of the most severely impacted area began on October 18th, 2001, and concluded on November 19th, 2001.

Next slide, the volunteers. The information in the literature does not provide a comprehensive list of all of the volunteers onsite for the time frames of participation of those that did respond. Literature indicates that there were many volunteers that played a role in the response, with specific mention of the Red Cross and Salvation Army.

It is reasonable to conclude at least some volunteers were onsite through the FBI relinquishing the site to DOD on September 26th or 28th, 2001. The literature does not provide additional information pertaining to volunteers remaining onsite through the demolition and cleanup phase of the response.

Next slide. So the available information concerning the Pentagon response does have limitation. The information has uncertainties as to when each of the responding groups faced increased-risk activity at the Pentagon site.

Next slide. For the Pentagon response to the September 11th terrorist-related aircraft crash, the recommended concluding date is November 19th, 2001. To ensure that each of the groups that did respond are provided adequate opportunity for medical monitoring and treatment benefits, the World Trade Center Health Program eligibility is recommended for the period covering September 11th, 2001 through November 19th, 2001.

The available literature indicates that documented air and wipe sample monitoring conducted through September 28th, 2001, did not reveal any exposures of concern. However, no information is available on exposures during the demolition of areas directly affected by the aircraft crash.

The next few slides will cover the Shanksville, Pennsylvania response.

Next slide, please. At the Shanksville site, fire department personnel arrived onsite shortly after the aircraft crashed. The FBI controlled the site from the onset of the response. Most of the fire department personnel left the site after the FBI turned the site over to the Somerset County coroner on September 24th, 2001.

There was a limited fire department presence until the conclusion of the final sweep of the crash site which took place on September 29th and 30th, 2001. The available information does not indicate whether fire department personnel were onsite during the site restoration activity from October 1st through October 3rd of 2001.

Next slide, Shanksville Police Department. Law enforcement personnel were also on site quickly after the aircraft crashed. Like the fire department, most police department personnel left the site after the FBI relinquished the site to the county coroner. Police department presence was limited at the Shanksville site until the conclusion of the final sweep of the crash site for aircraft parts and potential human remains on September 29th and 30th, 2001.

The available information does not indicate whether police department personnel were on site during the site restoration activities from October 1st through the 3rd of 2001. The literature does suggest that law enforcement personnel remained at the Shanksville site for a number of years to provide security.

Next slide. For the recovery or cleanup contractors, the literature indicates that environmental restoration contractors restored the site as close as possible to the original appearance as they could from October 1st through the 3rd, 2001.

This included backhoeing the crater with soil, adding topsoil to the crater area as well as the forested area near the site and seeding the area with flowers and grasses.

Next slide, volunteers. The available information does not provide a comprehensive list of all of the volunteers onsite or the time frames of participation of those that did respond. The Red Cross and Salvation (sic) are cited as responding to the Shanksville site. Like fire and police personnel, most of these volunteers left the site on September 24th, 2001 and had limited presence until the final sweep of the site on September 29th and 30th.

The available information does not indicate whether volunteers were on site during the October 1st through the 3rd site restoration activity. As

with the Pentagon, the Shanksville site has limitations in the information and that information has uncertainties as to when each of the responding groups ceased increased risk activity at the Shanksville site.

Next slide. The Shanksville response to the September 11th terrorist-related aircraft crash, the recommended concluding date is October 3rd, 2001. And to ensure that those who did respond were provided adequate opportunity for medical monitoring and treatment benefits, the World Trade Center Health Program eligibility recommended for the period covering September 11th, 2001 through October 3rd, 2001. Environmental monitoring at the site indicated that surface soil, subsurface soil, and groundwater did not exceed Pennsylvania Department of Environmental Protection health standards. Remediation was not required at the site. No indication that surface water contamination was attributable to the crash.

Next slide. The following is proposed eligibility criteria for the Pentagon responder: being a member of the fire or police department, whether fire or emergency, active or retired or worked for a recovery or cleanup contractor or was a volunteer who performed rescue, recovery, demolition, debris cleanup, or other related services at the Pentagon site for terrorist-related aircraft crashes of September 11th, 2001 for at least one day during the period beginning September 11th, 2001, ending on November 19th, 2001.

Next slide. The following is the proposed eligibility criteria for the Shanksville responder: member of a fire or police department whether fire or emergency, active or retired or worked for a recovery or cleanup contractor or was a volunteer who performed rescue, recovery, demolition, debris cleanup or other related services at the Shanksville, Pennsylvania site for the terrorist-related aircraft crash of September 11th, 2001, for at least one day during the period beginning September 11th, 2001, and ending on October 3rd, 2001.

This concludes my presentation for this afternoon.

DR. WARD: Are there questions for Rob? So, does anyone on the committee want to ask any questions or make any comments about Rob's presentation?

DR. HARRISON: Thank you very much for all the comments. I think it's very reasonable.

DR. WARD: I agree. Is that the general sense of the committee, that it's

reasonable? Okay, well, we'll record that for the record.

RESEARCH NEEDS

So, now we'll go back to the research needs and where we were on that was I was asking if anyone had any questions or felt the need for more discussion on the research recommendations or the document that was circulated regarding principles for handling conflict of interest within this committee.

PETITION ON CANCER

Okay, hearing none, we'll move on, and I guess our next topic is the petition on cancer. For those on the phone, I am going to be moving to the podium so that I can present some slides I prepared, and that will take -- that transition will take just a minute. It will be another minute because Paul is conferring on something. Are we okay to proceed?

Well, I think as most of the committee members know but possibly some members of the public may not, we had hoped to discuss -- is this on? Is that better?

DR. MIDDENDORF: Would you prefer to use this one or that one?

DR. WARD: Maybe we should use the other one, and probably we should turn this one off. Thank you. I do have a small voice, so this will be very helpful.

As most of you know, when we had to -- when we weren't able to have our last meeting by teleconference, one -- the plans for how we were going to address the petition on cancer was one of the things that we were going to discuss as a committee, so in the absence of having that meeting, I really thought hard about how we could approach this topic in a way that we could really have meaningful discussion at this meeting despite that circumstance.

And as you all know, we received a letter from Dr. Howard subsequent to a letter he received from several congressmen asking us to review the available information on cancer outcomes associated with exposure resulting from the September 11th terrorist attacks and provide advice on whether to add cancer or a certain type of cancer to the list of World Trade Center-related conditions.

And as we discussed that at our last meeting, I think we realized that there were a number of very complex and difficult questions embedded in that -- in that request. And one of them was basically whether, based on what people were exposed to at the World Trade Center, do we believe it's possible, probable, or not that the exposures could cause

cancer.

And it's -- whatever our recommendation is, we would need to provide a scientific rationale. Now there's a second topic. There's at least one other really complex topic that came up at our last meeting, which was what are the criteria for having a health condition?

And so my idea was to focus today's presentations and discussion on the first question: Do we believe it's unlikely, possible, probable, et cetera, that exposure to the dust may cause cancer, and then depending on where the committee stands at the end of the day, we'll decide how best to use our time tomorrow.

And I think it's important. My boss says -- at the American Cancer Society -- says this all of the time, so I guess he's implanted it in my head. I think when we talk about the scientific rationale, it's really going to be important to talk about what we know, what we don't know, and what we believe, because I think that, you know, we'll all -- in all the presentations today, one recurring theme will be we wish we had more data; we wish we understood the exposures better; we wish we knew more.

EPIDEMIOLOGY AND OVERVIEW OF MECHANISMS OF CARCINOGENESIS

So what I'll be doing is just reviewing the epidemiologic studies that are completed and ongoing. I am going to talk about the potential carcinogens present in the World Trade Center dust, and then I am going to give a quick overview on mechanisms of carcinogenesis, really focusing on those issues that I think pertain most to our discussion today.

So with respect to the epidemiologic cohorts, we had several presentations on them at our last meeting and we also have access to published information on them. So I am just going to go through them very quickly.

Among the cohorts that are under study, there are -- there's studies going on of the Fire Department of New York, and I think these studies probably from an epidemiologic point of view are the most -- are going to be the most complete and informative because we know that they really have a well-defined population and a population that is, you know, highly exposed, a comparison group.

And they also have a separate set of EMS workers that has not been published on. They're also doing an employer-based medical screening program, which will provide additional information.

The second large cohort that can be studied is the New York -- is from the New York and New Jersey World Trade Center Clinical Consortium, and I think that will also be a very informative study. It will suffer from the limitation that it essentially was a self-referred group of people.

The third one, which I'm not sure is actually being studied for cancer or not. I'm sure someone in the room knows. It's the cohort that's been identified through the World Trade Center Environmental Health Center, and this population is unique because it includes some children.

And then there's the very large World Trade Center Health Registry that's being run by the New York Health Department. And that one is clearly the largest in terms of sample size. Probably the most severe limitation is that about 70 percent of the cohort is self-referred rather than identified from the list or records, and that group is being followed both by surveys and by linkage with cancer registries and mortality data.

So in the first publication of cancer incidence data from the firefighters cohort, the incidence ratio for all cancers combined was 1.10 compared to the general population. And depending on particular adjustments used, it was 1.19 to 1.32 in comparison to non-exposed firefighters.

There are also some excesses for particular cancer sites. The findings differed a little bit based on which adjustment was used, but basically, there were significantly elevated or borderline excesses observed for stomach, colon, melanoma, prostate, thyroid, and non-Hodgkin lymphoma compared to the general population rates.

And I think one thing that's important to note here, because it's been noted by others in the literature, is that there are a number of these cancers that no -- are likely to be detected by screening or by just access to medical care, and the paper did attempt to control for that bias in the analysis.

But with respect to other epidemiologic studies, in the first publication from the World Trade Center Health Registry study, there was no excess of all cancers combined or eight major organ systems reported. There have also been case reports suggesting the possible excess of multiple myeloma in the literature.

So I think one of the things that it's important to understand before we move on from the epidemiology studies is that epidemiologic studies in general have their strengths and their weaknesses. One of the strengths is that you're actually studying the events, not animal systems or models.

On the other hand, it's often very difficult in epidemiologic studies to accurately estimate exposure, and I think that applies even more so in these studies; although, I think there have been really good attempts to use surrogates of exposure, like in the firefighter cohort, kind of developing exposure classifications based on when people arrived at the site, for example.

So I think that the existing studies are doing the best job that they can, but ideally, you know, what you'd love is an exposure matrix for each person so that you knew, you know, this person was very highly exposed and they didn't work well. And that's probably not going to be present. And so, when you don't have good exposure information, you may not be able to see some of the things that you tend to look for when we look for causal association, so we may not see a strong dose response, because we don't have good exposure data. We may not see the trends that one might expect to see.

Another criteria for causality that's considered is consistency between studies, and again, I think, especially in this case, we may not see that level of consistency because we don't have one exposure. We have many exposures, and we have different populations and individuals who were exposed to different things, so I would not be surprised at all with the different studies that they show increased risk for cancer. They may see increases at different sites, so I think we have to be really cautious about especially making negative conclusions about the findings of these studies.

And the last -- well, the last one on this slide is even though many of these populations are sizable, they're still, in many cases, small enough or early enough in the follow-up period that there are not very many cases expected based on population rates.

So if we don't see an effect, we really need to be careful in interpreting that because it may be -- the studies may be too small to rule out small risks or risks for rarer cancers. One of the most important things, and I know it came up in our discussions last time, and I'm sure it will come up again today, is that, you know, I think when we all were trained in occupational health, those of us who were, we all thought, well, you know, usually solid tumors you're looking for at least 20 years between the onset of exposure and disease and hematologic cancers, the latency period is shorter.

And -- but I guess what I wanted to emphasize is the issue of latency

period is most relevant in epidemiologic studies early in the follow-up period when we have negative results and follow-up may be too short to see a positive effect.

It's not necessarily relevant in the sense of saying, well, this cancer can't be related to exposure because, you know, the exposure only occurred five years ago. I'll get more into that later, but I don't think you can make those kinds of assumptions based on what I'll present to you about the mechanisms of carcinogenesis.

So, if -- I think we got the -- I got the sense in the discussion last time, and this doesn't probably represent everyone's viewpoint, but I did get the sense from the discussion that many people felt that they could not make a decision on the cancer petition based on the epidemiologic data alone.

Obviously, the strongest study is the firefighter study, but I don't -- I didn't sense an overwhelming consensus that the findings of that study were so definitive that it would be the basis for a recommendation. So then the question was, what can we learn from looking at the exposure data, but I think we have to acknowledge at the outset that it's incredibly difficult to interpret the -- especially air sampling data from the World Trade Center study.

And one critical limitation was that there's almost no data from the first week after the attack. A lot of people said that last time, and I think, you know, I think we all understand that. I'm puzzled about some of the air data, because it really seems like the low air levels measured in some of the personal air sampling studies done on the workers seems really inconsistent with the extent of respiratory symptoms that we're seeing.

And so I don't know how to answer that question, but it's my belief that it's, you know, I don't see it fitting together well. So, one approach to looking at the cancer hazard which I thought we could take today is really to focus on the composition of the initial dust and smoke as reflected in the mass dust samples that were collected.

And those samples were collected and analyzed by more than one group so at least we have some -- we can look at consistency of their findings.

And the other benefit, I think, of looking at the dust and smoke is that there were a lot of populations exposed to that.

So, for example, we know that there were fires at the site, and we knew that -- we know that firefighters and police officers who were on the

site itself were exposed to combustion products from the fires, but just for the purposes of having a simpler discussion today and a discussion that kind of encompasses exposures to all of the groups, I thought we could first focus on the dust and smoke, recognizing that there's more -- there's more to the story that we'll have to get to later.

So, in poring through the literature and, you know, all of the exposure papers, I have to confess, I am not a chemist; I am not an industrial hygienist, and it's not easy to read these papers. But, you know, one of the things that I got out of it was really, you know, what went into the buildings is really what came out of the buildings.

So, if you look at, you know, there was a lot of light-weight concrete; there was asbestos; there was gypsum; there was drywall; lots of glass. There was glass fragments and man-made vitreous fibers from insulation. We know that there were polycyclic aromatic hydrocarbons measured in the bulk samples. We know that there were metals measured in the bulk samples.

And then, we also know that there were volatile organic compounds in the mix. Now those probably, looking at the dust, is not the best way to look at exposures to those, which is why I have them in blue, because we know they were there. In the dust, though, they may have been absorbed onto particles and fibers and other things, so they may be there, but it's probably not the best way to measure them.

So, what, I mean, what -- so, two of the reasons I focused on these particular exposures is one, that they were pretty substantial. So, for example, the asbestos was, you know, in a few of the bulk samples was from .8 to 3 percent of the total weight of the sample. So that's pretty significant. The other thing is a number of them are -- have been recognized as human carcinogens for which, based on epidemiologic data, so they are substances for which we have fairly strong epidemiologic data.

So that's why we're focusing on these particular exposures. It doesn't mean that there aren't other classes of exposures of concern, and you know, we're not talking today too much, at least in the presentations, about PCBs and furans and, you know, TCDDs, but again, you know, we have a limited amount of time, and I wanted to focus on the things where I thought there was the clearest data to talk about.

So, now shifting gears a little bit, and I want to thank both Julia and the National Cancer Institute for these slides. Julia pointed out to me that

there was a slide set on the National Cancer Institute website that we could use for this presentation because I think that a picture is worth a thousand words.

So all of the slides in blue come directly from that website and have not been modified. So basically, what is cancer? So, when a cell becomes cancerous, basically, it loses the ability to control its own growth and to organize itself appropriately in tissues. And this -- one of the key things in that process is the damage to the DNA of the cell.

So this is a slide that summarizes a number of different characteristics of cancer cells, and it's really, at least historically the way that cancer has been recognized is pathologists look under a microscope at the appearance of the cells from the tumor. So the cells will be different. They'll have larger nuclei. They will not organize themselves into neat structures the way they're supposed to.

So that's a real quick review of that, but you, typically, you know, for our classic carcinogens, both tobacco and asbestos, we see a 20-year latency period, and that's -- but what that means is in 20 years from the onset of exposure to the peak of disease in the population, so in this case, men started smoking in the United States soon after 1900, and we saw the peak in lung cancer in the 1970's.

So the -- so as I mentioned, the key, you know, the critical step in carcinogenesis is an interaction of exogenous or an endogenous substance with DNA within the cell, and that can be a chemical, it can be a virus, it can be radiation. So there is a component where there is an interaction with DNA.

And typically, what happens, and this is grossly oversimplified, but basically the DNA is the cell's mechanism that basically codes for the production of everything a cell needs to grow and sustain life. So, what happens is when there's a chemical damage, for example, that might change one of the -- and so, and the code is really in the three -- it's in three, you know, it's in three chunks.

So, CAA codes for a particular thing, and if you substitute one of its -- one of the chemicals, it changes the whole, that whole code. So, basically, three things can happen. You can change a single base. Those things are called bases, and the three together are (indiscernible).

You can change a base. You can put an addition in a base, or you can make a deletion from the base, but in any case, it basically messes up the code such that the gene is not effectively doing what it's supposed

to do.

And there's really three kinds of genes that are involved in the process of carcinogenesis. One type -- and you know, this is large categories. One type is oncogenes, and what oncogenes do is they -- when they're -- they accelerate cell growth and division. Tumor suppressor genes enable the cell to put a brake on that kind of uncontrolled growth and DNA repair genes allow the cell to repair errors or mutations in the DNA itself.

So what happens, if you're exposed to a carcinogen and you have a mutation and in any of those three types of critical genes, if the cell does not repair that mutation before it divides, that mutation is going to be passed on to the daughter cells.

So typically what we see in cancers is multiple mutations, and it's kind of, it's thought that these mutations occur over a period of time, so possibly, you know, when you're 25, you get a mutation in a tumor suppressor gene, and if that is maintained, then as those cells divide and proliferate, they accumulate additional mutations, and in that process, though, you're not just -- the changes in, the mutations in the genes is not the only thing going on to lead to cancer. Other things are going on that kind of promote the growth of those cells.

So for example, for breast cancer, estrogen promotes the growth of tumors in the breast because breast tissues are naturally sensitive to, you know, hormones, for example. So it's not only the genetic mutation or the interaction with the DNA. It's multiple things going on.

And so, we tend to divide the process of the carcinogenesis into four big buckets: initiation, which is basically, at least an initial mutagenic effect; promotion, which is, you know, encouraging those abnormal cells to grow; malignant transformation, which means that the cell has kind of passed beyond the point where it can revert back to a normal cell. It's accumulated enough damage that it's essentially destined never to go back to normal. And then ultimately that tumor gets larger and invades other tissues beyond where it arose and it can metastasize to other parts of the body.

So the reason I'm emphasizing the promotion and progression is, is that it's important in the context of the exposures we're discussing today because inflammation is one of many -- it's one of the important mechanisms of carcinogenesis. And inflammation actually can do a large number of different things, but basically inflammation is a normal

response to tissue damage that can result from infection, chemical irritation, and/or wounding.

However, when it becomes chronic and it becomes chronic in a number of known diseases, it can damage the body and lead to illness. So, for example, we've all heard of Crohn's disease, which is kind of an inflammatory condition of the bowel, cirrhosis of the liver, which is an inflammatory condition of the liver. Many of the diseases, especially the infectious diseases that result in inflammation also result in cancer. And inflammatory processes can also occur as a result of chronic chemical and mechanical inflammation, but it's important to know that inflammation in general can really lead to cancer in a multitude of ways. Its increasing cell proliferation and turnover is actually generating mutagenic substances from some of the reactions that release oxygen and nitrogen species, and it's also producing cytokines and growth factors and other biologically active chemicals that are influencing the microenvironment around the area where the potential tumor is developing.

With regard to mechanism, I guess the other things I wanted to mention are that -- one of the things we have to consider is that for many of the people in the exposure group, the duration of actual exposure is relatively short, but I think it's important to note that at least in some of the populations studied, inhaled fibers and dust can remain in the body for a very long time. And so, in fact, a short-term environmental exposure can lead to a long-term biological exposure, and we've seen that in some of the bronchial lavage studies.

The other thing is, you know, we've talked about this average latent period for solid tumors, but I think it's important to recognize that it all depends on what stage in the cancer process an exposure occurs. So, for example, we see this curve in the population when in relation to onset of smoking in the population at large, you know, and then the lung cancer epidemic following 20 or 30 years later.

But when a person stops smoking, their lung cancer risk goes down dramatically within three to five years. So, what, you know, one thing that's probably happening there is that essentially tobacco smoking contains practically every carcinogen known to man, and some of those substances actually are promoting or, you know, causing the tumor to progress, so they're both initiators and promoters.

And so you see this much more rapid effect in an individual that stops

smoking than you would expect from the long latency period for the initiation, and we've seen something similar recently in breast cancer and this is really interesting.

So, in 2002, the Women's Health Initiative published a study showing that use of postmenopausal hormone therapy was associated with an increased risk of breast cancer and the surveillance epidemiologists noted in that year's data that there had been a dramatic drop in breast cancer incidence virtually the same month that those studies came out. And at the time, you know, everybody was saying it can't be related to HRT, it's not biologically plausible that something could act that fast. Well, if, you know, there's pretty good consensus now. I don't think anyone disagrees that one of the major factors or the major factor in that abrupt decline is that, you know, on a population basis, a lot of women stopped taking HRT, and HRT was really promoting or causing tumors to progress in the women.

And since that time we've actually seen a flattening out of rates. It's not continuing to go down, which further supports the hypothesis that it was that one time decline in HRT.

So, we'll be moving on. I have a few more things I'd like to present, but then we'll be moving on to the presentations that I asked people to prepare regarding specific exposures of concern. But before I wanted to go on, I wanted to mention that I think there is an opportunity to learn more about the potential health effects of the World Trade Center dust exposure that maybe we haven't explored as fully as we could.

So, one of the things I noticed in looking through the literature is that, you know, there was a lot of concrete in the buildings and concrete is a -- you know, two of the main components of concrete are cement dust and silica. Silica, as I mentioned, is an accepted lung carcinogen and it's also associated with autoimmune diseases and stage III lung disease. Pulverized concrete also contains a material called Portlandite, which is highly caustic and not shown in this slide, but I know many people in the room are aware of it. People who work with wet concrete often get skin sensitization because of hexavalent chromium in the cement mix. And many European countries actually regulate the content of hexavalent chromium in their cement, but the United States does not.

So -- but it appears, and again, this is very preliminary -- it appears that maybe the hexavalent chromium content of concrete once it's set would not be as high as the mesolithic form. But again, that is something of

concern.

But in fact, there have been a number of studies of cement dust exposure, many of them done, interestingly, in developing countries, but many of these studies, and again, some are small, but actually a few are, you know, large enough and well designed, at least on the surface. And many of the studies, not all, find increased respiratory symptoms among people who work in the production of cement, and they also demonstrate reduced lung function among people with long-term exposure.

What I found most interesting is that there was one study that actually found an increased risk of GERD-type symptoms among people exposed to cement dust. And by the way, all of these studies are on the FTP site under the folder that says cement.

Of even more concern is there have been some cohort case controlled studies that have suggested associations between cement-exposed populations -- and that could be either in the manufacture or in the construction industry -- in cancer of the lungs, stomach, colon, head and neck, pharynx and larynx.

So cement dust that has not been reviewed by IARC or NTP and the only kind of official review I could find of it on it popped up on the web, and it seems to have been done by the Health and Safety Executive of the UK, but the version of the document online is a little odd because it does not have a publication date. It has a number, but no date, but I think it was -- it looks like it was published in 2006.

And basically, their synthesis of the cancer literature at that time was that the epi data was not convincing, but that they felt that some of the associations that had been seen were biologically plausible in large part due to the known inflammatory responses associated with exposure to cement dust.

So one of the ways I thought -- I mean, I thought I had a pretty reasonable way to frame the discussion today and get into depth on some of the most important issues, but I think tomorrow, the agenda is wide open, and one of the things I thought that might help us frame an agenda would be to -- at the end of the presentations, we'll kind of poll the committee and ask each person to check one of these words and turn them in -- so, this is not a vote, it's just a poll.

And then what we'll do is we'll summarize the distribution of the results, just kind of arranged by the exercise. So, we'll summarize the

distribution of the results and that will help us know, do we have two really different viewpoints? Are some people really on the side of probable proof and are other people way off on unlikely, possible, or do we have, you know, a distribution centered at the middle?

And then we can really see, you know, how can we use our time tomorrow to, you know, to see if the group has a consensus or not or to figure out what issues are of most, we're most uncertain about. And again, we are all prepared to tabulate these result in such a way that you --

MS. HUGHES: I have a quick question. On the slides --

DR. MIDDENDORF: Wait a minute.

MS. HUGHES: Hi, I have a quick question. On the last slide, it says is the blank that exposure World Trade Center may cause cancer. Can we also use slash smoke, because not all of the exposure was dust --

DR. WARD: Yes.

MS. HUGHES: Because not all of the exposure was dust.

DR. WARD: Yes.

MS. HUGHES: Because then it would be more consistent with some of the other slides.

DR. WARD: Yes.

MS. HUGHES: Okay, great, thanks.

DR. WARD: We can make that -- yeah. So, anyway, I think this will be helpful in framing tomorrow's discussion and, you know, and these are various options that we could discuss tomorrow. There may be -- it may be that people feel that there's critical evidence that we didn't cover today that we should go into in more depth tomorrow.

It may be that there are clearly opposing positions that we should try to address tomorrow. If we're -- if there's apparently a high degree of consensus, then we can start talking about the rationale for the position.

If we are leaning towards saying probable, then we can discuss the issue of what sites do we think are probable, and then hopefully have whatever -- wherever we are, and certainly we can discuss the possibility of needing to have another conference call or meeting before we can make our recommendation.

So, with that, along with my presentation, are there any questions?

DR. MARKOWITZ: So just a couple of comments. One is I don't really favor taking a poll before we have the public comments. We have the

public comments at the end of today and beginning of tomorrow morning, because that would add to the discussion, influence our thinking, so I would advocate doing a poll after that.

I would also like to have, you know, do some discussion before we do a poll because I want to hear what people think. So if you want to do a poll, we could do it. We could change the time, though, until tomorrow after public comments and after there's at least some initial discussion. I assume the purpose of a poll is to sharpen further discussion.

Another comment I have is about the choices of unlikely, possible, biologically plausible, probable, definite, and that is that actually I think biologically plausible stands with both possible and probable, and so I'm not sure that these are exclusive categories. And I understand that it's preliminary, a rough way of getting a sense, and I wonder whether one alternative approach would be to consider reasonably anticipated as a substitute for one of the categories.

DR. WARD: Maybe probable?

DR. MARKOWITZ: Well a --

DR. WARD: I guess, that's the thing, it sounds like probable to me but, so I guess if -- we can make any changes that you all want to make. It did occur to me that maybe the timing was wrong, but again, the timing was kind of thinking about how can we tabulate these results so that we could leave people thinking about how we're going to use our time tomorrow.

And some people may even want to, you know, think about ideas that they'd like to present or do literature searches tonight, or, you know, people could prepare to argue the main points overnight and so I did -- well, I did bring enough paper ballots that we could have more than one poll, so that's one option. Valerie?

DR. MIDDENDORF: I think Catherine had a --

MS. HUGHES: Yeah, I had a quick question.

DR. MIDDENDORF: So, Catherine, then Tom, then Valerie.

DR. WARD: I think I need to have my eyes transplanted so --

MS. HUGHES: I know we're all -- we're looking at actually what was in the dust and what was in the fumes. Are we going to look at also the impact of the temperature, because it wasn't as though the temperature was the temperature of the day, because it was just so hot. It was like 1000 degrees -- if people were close would have been impacted and how the items could have changed due to the

temperature, too.

DR. WARD: Yeah, and I think, you know, that would fall under the category of things where there's something that where there are critical issues that we haven't discussed. I don't know if anyone is prepared to talk about the temperature today or, you know, has really looked into that issue, but if you feel that that's an important issue, we can see if there's anyone who wants to comment on that further or we can put it on a list of things.

Again, I guess the question is do we feel like we have enough information to make a recommendation now, or are there things that we feel are so important that we need to wait until, you know, somebody really studies them well enough to talk about them.

I mean, I certainly couldn't talk about that today, and I don't know if anyone else could.

DR. ALDRICH: I was going to suggest, if there's going to be a poll, maybe two questions: biologically plausible, yes or no; and then the other four, pick one.

DR. WARD: Good.

DR. MIDDENDORF: We forgot Valerie.

MS. DABAS: Just because I am not a scientist, I just want to get the definition of biological plausibility just because I've seen so many different ones on the websites.

DR. WARD: That's a good question. My definition of it is that when you look at the exposure and what was -- when you look at the dust and smoke and you look at what was in the dust and smoke, and you look at what the toxicity of the, of that we've already observed in the events and, you know, when you look at all of those elements of data, it makes sense that this exposure could cause cancer based on what we know about the cancer process and the components in the material.

Now, that's my definition. Someone else may have a better one. Julia?

DR. QUINT: I think I agree with most of what you said except I'm not limiting it to humans, because I -- the animal data that shows that something is carcinogenic, to me, means I don't think -- there are only a few cancers in animals that are not biologically plausible in humans, so I think the animal data is a plausible mechanism in humans as well.

DR. WARD: Yes, and I totally agree with that, and --

DR. QUINT: I thought you did.

DR. WARD: Yeah. I am going to return to my seat until we are done

with --

MR. CASSIDY: Thank you. You've discussed a lot of topics, and one that I think is interesting when you look at this is, you know, is it blank that the exposure to World Trade Center dust may cause cancer, and I think it's hard to, you know, may be hard for some people to answer that unless you're talking about a level of exposure, right?

So you were talking about cigarette smoke, and I would think that the studies show if you smoked one cigarette and stopped before you had an exposure to tobacco that the likelihood of developing something from that would be different if somebody smoked five packs a day for ten years, right?

So I think it's important that the part or at least part of the discussion to the level of exposure, and I tie that in to when you said that the air sample data seemed to be inconsistent. Well, the question is where was that air sample data taken? And, you know, my personal recollection is I didn't see anybody standing on the Pile taking it.

So, I don't know where -- if they took it five blocks away or ten blocks away or where they took it. And on that note, the air sample data, I would remind everyone that is -- there was much discussion about whether or not that was a political decision to say quote, unquote, the air was safe because they wanted to open up Wall Street. You know, we had to get back to business, the country was shut down. So, I just wanted to raise that point.

I think people that were there working at the site knew the air wasn't safe no matter what **[identifying information redacted]** witnessed, so.

DR. WARD: Yeah, and I do want to, I mean, I fully acknowledge those issues and I didn't want to spend a lot of time on them today just because I really feel like, you know, both the committee discussions and the published literature both, you know, essentially give that same information. But it's really trying to come up with other approaches that maybe can be a little bit more revealing and make -- help us make a decision.

But I think, you know, there's at least, there's a couple of exposure scenarios and I think we should acknowledge that too so we have people who were -- we have a very heavily exposed group that was working directly on the Pile, especially in the early time period. We also have the potential for the community residents and the workers to have prolonged exposure to the dust that entered the homes and office

buildings.

Now, again, I don't know that you would expect to see exactly the same health effects in those two populations, but they're both populations that may have significant exposure, possibly to different substances and different concentrations.

DR. MIDDENDORF: It's easy to forget that we have some committee members who are on the phone, out of sight, out of mind, so I just want to ask if any members on the phone have any questions or comments.

DR. WEAVER: I don't, but we're moving along fairly quickly and I just want to point out that I'll be teaching from 1:30 until 2:50 and I'm scheduled to talk at 3:10, so, you know, we can just juggle when I talk around class, but when I am in class I'll have my cell phone, so I can listen in.

MS. SIDEL: I just want to say that because we don't have air samples from, you know, from the day 9/11, that's why Officer Harris's uniform is so fascinating, because it's like a snapshot in time of what, what was there, and I believe that this also -- another study of what FDNY, I think, equipment that I've seen that are also from the actual day 9/11 from people that were working. So, you know, I feel as though there's a lot of different air samples and they sort of collectively say the same thing, and that is that there were a lot of carcinogens down there.

And then we start talking about, you know, different zones of exposure, but you're never going to get -- that's never also going to be firm and there's definitely people that were super-exposed, but then there's also other things that can happen, you know, you can just be in your home and, you know, cleaning up your bed and there's a big pile of dust, so is that the same as working on the Pile the first day? What difference does it make?

Because if you get one little drop of asbestos, then you get that whether you get it on the Pile on the first day or you get it while making your bed, you know, three months later, so it's kind of, I understand from scientifically for us to have all of these categories but working in real-time in what actually happened to people, I think you have to be more open-minded.

DR. WARD: And I think we are trying to do that.

MS. SIDEL: Oh yeah.

MS. FLYNN: I, you know, I have to agree with Steve Cassidy and with Susan Sidel. I mean, a lot of us were involved in the EPA World Trade

Center Expert Technical Review Panel where the flaws and inadequacies of all of the government data were, you know, pored over at great length. Unfortunately, the public record of that panel has been removed from the EPA's website and Congressman Nadler's request that it be restored as a resource for this committee and for the public has gone unheeded.

But, you know, there have been many, many observations made in that process about the ways in which, for instance, when a monitoring instrument picked up benzene spikes on the Pile, the instrument was shut down and moved to another site.

The errors in the, in the asbestos air sampling for lower Manhattan residences that was conducted by ATSDR and the City Health Department were reported by residents who were eyewitnesses to the fact that fans were turned to the wall, that leaf blowers were not turned on. I mean, it almost borders on the level of sampling fraud. So, first of all, they were, you know, we don't have really good sampling data to fully characterize exposures in exposed populations. And second of all --

DR. WARD: But didn't I say that? I mean --

MS. FLYNN: Yes. No, I just -- I think it really bears reemphasizing and also to -- I know that some people saw this article that I sent in by David Newman, the industrial hygienist with the New York Committee for Occupational Safety and Health, and but I -- he said in this article, under the category of exposure assessment:

If just one thing is to be learned from the WTC response experience, it should be that an exclusive reliance on environmental sampling data can be misleading and even dangerous. There has been a fundamental disconnect between what the majority of the sampling data would seem to indicate and the breadth of health issues that have arisen. WTC-related illnesses manifested despite reassuring results that came from traditional methods of data collection and assessment. Tens of thousands of WTC responders, area workers, and residents incurred significant and persistent respiratory and other chronic and incapacitating illnesses.

And I just want to make one more comment, which is that, you know, not to further complexify (sic) the polling language, but in fact, the Zadroga Act sets a criterion for linkage of illness to World Trade Center substantially likely to have been a significant factor in causing,

exacerbating, or contributing to, so is there a way actually to map that language on to the polling language? Because I think we're looking at a real -- I think we're looking at contributing to may get us where many of us feel we need to go much more quickly.

DR. WARD: So we can definitely change the language with the poll. I guess I remember at the last meeting there was a little bit of confusion about the criteria for listing something as a World Trade Center-related condition versus the criteria for determining that a particular person's illness was World Trade Center-related. So I don't know if the language that you quoted was -- which one that was. I don't know if it matters, but I think we can certainly change this.

I think it really -- what I was -- what we were trying to do is come up with a way to express it where we can understand the diversity of opinions among the group so that we can figure out how we can have a more productive discussion tomorrow. Whether the, you know, if we have general agreement on the overall issue of the potential for carcinogenicity, then we can move on and discuss other things. If not, we need to stick on that point until we understand why different people have different views.

DR. HARRISON: Thank you. I wanted to say something else, but I wanted to thank you because I am going to change what I was going to say, I think, because I was not aware that there was the language.

And I would ask, maybe, if we could clarify that point because I think, at least in terms of my thinking about whether or how or what we would recommend as a committee, if we need to use certain criteria that is legislatively mandated, I think it's very -- it's significant, pardon the pun. So, if we could just clarify that because there are -- because it actually ties in with the comment that I was going to make. I think there's all sorts of perspectives on how to come to a recommendation in terms of cancer causation.

There's the individual patient that some of us, including myself, bring to that perspective when I see an individual in my office with an occupational or environmental cancer, what criteria do I use. There's workers compensation criteria. There are civil litigation criteria. There are cancer presumption law criteria. There are many different frameworks that I personally am familiar with and bring to this discussion.

If there are other specific criteria that in the legislation that directs us

to consider, then I think we should at least understand what that is and come to whatever straw poll with a reasonably common set of understanding so that -- and this is my comment -- it's sort of agreeing with Steve. It's that if you do a straw poll before we have some common framework may just give us, you know, 15 different ideas about what we are voting on but not a common set of criteria to guide our vote.

DR. QUINT: Yet another frame is a public health frame and the prevention frame that I come from and also the toxicology frame. I just wanted to tie some of this back to Liz's presentation where she talked about mechanisms because one thing to consider, when she talked about mutations is one -- a lot of these carcinogens are thought to have no threshold, so that when you're talking about amount of the carcinogen or substance that the person was exposed to, it's thought to be linear, so it's going through zero, so any amount could trigger a carcinogenic response.

Of course, you know, normally we talk about some risk above background, but to do that, you have to know the potency of the carcinogen plus you actually need to know exposure information and something about the exposure profile: how many days a week, how many years, et cetera, that the person was exposed to it; and we don't have those data.

So and the -- there's an article in our file, the folder, Guyton, et al, in Mutation Research which is very compelling because it talks about these carcinogens operating through many modes of action, so it's not just one. It's not just that they cause a mutation. They can act on, you know, promotion and different aspects of the carcinogenic process.

So read by my count have 72 carcinogens in the dust, at least the ones that NIOSH listed. Some of these are human. Some of these are animal, so I think, you know, we have to keep all of these things in mind when we talk about biological plausibility.

There are a number of in vivo and in vitro articles where people have actually demonstrated with very short exposures, you know, a triggering, mostly the carcinogens that act on an inflammatory process, but, you know, have initiated a process that ends up, you know, that goes through all of the steps and so -- and in very short time periods, some acute and some sub-chronic exposures.

Again, they're in mice, and they're in human epithelial cells, but I think

all of this enriches our understanding of the mechanisms of carcinogenesis and argues that this is a very complex process when you add, you know, high exposures, very high exposures with a multitude of carcinogens, you add to that complexity.

And also ingestion. You can't forget about the fact that some of the exposures probably occurred through ingestion when you have dust on surfaces, especially in offices and homes, you probably have added to that probably also with the firefighters as well, given the amount of contamination on their uniforms. So it's not just the air levels. It's a, you know, very rich mix of information that we have to consider.

MS. SIDEL: Just in terms of ingestion, my supply tent was right on the Pile and we were serving coffee and food and all sorts of things, so I'm sure that things were flying in there.

DR. WARD: So are you -- oh, Steve.

DR. MARKOWITZ: I just want to follow up on what Dr. Quint was saying. So we don't have a lot of experience with people with short exposures and long-term follow-up and cancer in particular, so could you just discuss a little further what experience there is with animals about certain carcinogens with acute or a very short term exposures subsequently relating to cancer?

THE COURT REPORTER: Can I say something real quick? If you'll get that microphone real close to your mouth it helps me a lot. I will appreciate it. Thank you.

DR. QUINT: I agree with you. Dr. Markowitz said that there isn't a lot of data. I was actually looking for some dose rate data in animals to sort of understand better whether or not we had those models, but there is a paper by Beaver et al that -- let's see, I have it right in front of me here. And actually, she was looking at the exposure to chromium and looking at lung inflammation and injury and then a proliferative -- or from repetitive exposures.

And I think in that situation, she was able to expose one kind and then get a response. There's also some information where people are looking now for other than animal models, and so the Hammer Institute had a study where they actually had a training set of carcinogens, NTP, and exposed after 90 days and was able to -- they looked for a marker which was a -- it was a gene expression biomarker, and they were able to see that within 90 days. I think other people have seen it within 24 days, so they're looking at different -- they're not looking at the

cancers, but they're looking at markers for carcinogenicity, very specific. There's the other study that I mentioned was the -- a study in human epithelial cells, and I have -- in that study, they were looking, I think, as short a period as 24 hours or maybe shorter than that, and they were looking at -- they compared both silica, crystalline silica and amorphous silica and were able to get a difference again in the whole process, you know, leading that was carcinogenic-like process.

So, no, animal models, I don't know of any in the regular bioassay models that would mimic -- that we could look at with this.

DR. WARD: There's also a lot of data on the cancer patients who were treated with radiation and chemotherapy, and there's very good data on their development of second neoplasms, and in some cases, you will, you know, there's enough data, let's say if someone -- there's a lot of data, for example, on young women treated for Hodgkin lymphoma with high-dose radiation to the chest who subsequently developed breast cancer.

So you could look at age and dose if that's -- but those are -- those agents are very strong carcinogens, but it is a very rich resource if you're into understanding how relatively short-term high exposures can result in carcinogenic effects, but... .

Sorry.

DR. MIDDENDORF: That's okay.

DR. WARD: I keep forgetting about this.

DR. TALASKA: There are a number of studies that were done by intertracheal lavage of PAHs that were single-dose were able to bring lung tumors, particularly in strains of mice that were relatively sensitive, so there is -- there are data. I can't think of the citations off the top of my head where lung lavage of PAHs, benzo[a]pyrene particularly, has led to a, lung tumors in animals from a single dose, a single heavy administration of a material in liquid -- in corn oil or another vehicle.

DR. WARD: Yes, again, I think the other thing to keep in mind is what I mentioned in the presentation that for some of these exposures, they -- if there's a long residence time in the lung and thoracic lymph nodes, a very heavy short-term exposure can result in a long-term dose. And so -- and I think we have some evidence of that in some populations.

Okay, so any further discussion before we turn to John Dement's presentation on asbestos? Excuse me? Oh, sorry. Folks on the phone,

any further comments before we move into John's presentation? Hearing none, John, would you like to start with your presentation? Well, Paul will queue up your slides and let you know when they're ready.

ASBESTOS AND WTC

DR. DEMENT: Okay, very good. Thank you and my apologies for not being able to be at the meeting today.

DR. MIDDENDORF: They're ready any time, John.

DR. DEMENT: Okay, just move on to the second slide. I'm going to talk about the dust exposure, so there's clearly the type of dust cloud presented in this photograph is a major high-level exposure to a mixture of things that we have already discussed today.

Next slide. There were no measurements done, as we have already discussed, of concentrations in the initial cloud. I think **[identifying information redacted]** and some others have estimated that the concentrations were likely in excess of 100,000 micrograms per cubic meter, 100 milligrams per cubic meter.

And I've sampled some industrial operation as a hygienist where dust levels were consistently in the neighborhood of 20 to 30 milligrams per cubic meter, not as high as this. So I think this estimation is probably a reasonable estimation, maybe on the low side for at least the initial dust cloud.

[identifying information redacted] described what he considered, and I think is a reasonable consideration, five specific post studies on 911 exposure categories.

Go to the next slide. And clearly the highest exposed were those there during the initial collapse and the days that occurred afterwards. I understand there was a rain event like around the third day, which helped to dampen at least some of the dust exposures, but I think the scenario is something like this: We have high-level exposures initially, and then we have continued exposures to the individuals who were doing the recovery and cleanup longer term, and also exposures to a much more mixed of (indiscernible) and fires and materials in that.

Let's go to the relative -- next slide, please. One of the relatives to dust exposure is (indiscernible) based on the plume depicted in this slide. I think clearly the first day, extremely high exposure, followed by lower-level exposures during some of the recovery operations; however, if I could point out here, there were no dust measurements actually made

on this first day. So these are reasonably speculative.

I am going to talk about asbestos, and go to the next slide please. And I am going to talk about some of the measurements that were made.

First, I wanted to talk about the methods that have been used for measuring asbestos exposures, both historically and currently.

On the list on here is an old midget impinger method developed by the U.S. Public Health Service in the 1920s. It's been used, really, for exposure measurement in occupational settings for dust exposures up until about the mid-1960s. I mentioned that largely because the old dust measurements and the basis for a lot of the risk assessment for asbestos are based on the old impinger method.

First of all, it was a method that didn't collect fibers very efficiently. Secondly, the exposure method actually counted all particles, not just fibers in the dust and it did it at a low power using low power optical microscopes.

So there's some -- excuse me -- some severe limitations with regard to retrospective exposure assessments even in the occupational environment. The current method used has been used since about the 1960s. It's called phase contrast microscopy. Basically the samples are collected on a filter, membrane filter, and the particles counted by an optical microscope that has a special feature which enhances contrast called a phase enhancer. But still, it's relatively low magnification, 400 times.

There are certain limitations to this method. First of all, the cause of limitation with regard to being able to count short fibers. Only fibers longer than five micrometers are counted. Secondly, even if a fiber were longer than five micrometers, this counting system -- the microscope has no resolution or ability to actually see small diameter fibers.

So you could have very long fibers that were small in diameter and not be detected. Nonetheless, it's used as part of the OSHA, current OSHA standard, and it's the basis of a lot of the risk assessments. And I think it's -- the use of the phase contrast microscope has actually enhanced some misconceptions about the nature of exposures and what's important. That is, only long fibers or fibers longer than five micrometers -- I'm going to have more to say about this later.

Moving on to scanning and transmission electron microscopy. Scanning microscopy is better than phase contrast, but still not capable of seeing

the very small diameter fibers in an asbestos dust cloud.

The most useful method is transmission electron microscopy, and some of the measures of the World Trade Center exposures were done by TEM. There are different techniques that are used for expressing the concentrations. Some express structures per centimeter of surface. Some were expressed as structures for -- as a dust concentration measurement per cubic centimeter of air samples.

The limitation here is the fact that when you look at samples by transmission electron microscopy, you look at a very small portion of the dust cloud, and it's very expensive.

A little bit about the measurements that were done. The range of asbestos, primarily chrysotile, looks like from a less than one percent up to about three percent of the mass. And with most fibers being less than five micrometers in length, which you would expect given the length -- given the nature of the collapse, the pulverizing of material.

There's more to say about the less than five micrometer criteria as well because even in asbestos-exposed occupational cohorts, the majority of exposure is to fibers that are less than five micrometers in length, typically 90 percent of actual.

Again, no measurements were made of chrysotile during the extraordinary high dust cloud exposure. There was a range of exposure measurements done later and reported in the literature, some in peer reviewed publications, some in -- just in reports.

Most of these seem to show short-term exposures of not in excess of established criteria; however, there are lots of limitations of these as we've discussed already. One is reading the samples would be the preferable method for looking at exposures to individuals on the Pile. NIOSH did some sampling on these, used PCM and looked at some of the samples by transmission electron microscopy, and in general, when you look at the samples by TEM, the concentrations didn't exceed the OSHA PEL of 0.1 fibers per cubic centimeter of air. Again, that's fibers longer than five micrometers.

Realizing of course that the majority of fibers in the study are less than five micrometers in length. I think there is a disjoint, and I think Liz pointed that out. This dust cloud was extremely high in dust levels, certainly initially. No measurements, again, but we would expect that in that dust cloud, given a concentration of one percent or even much, much less, that the asbestos exposures to total fiber concentration

would be very high.

I'm going to talk little bit about the types of regulated asbestos because many of the risk assessments have just considered asbestos as one group of materials; that's a list of them. We're dealing largely with chrysotile here which was in the towers.

I am going to say there may not be amphiboles in there. I had the opportunity of being in the World Trade Center a number of years before 9/11, and I think there might have been at least some amphiboles in the building as well at some point in time.

Liz has already pointed out, I think, that asbestos is considered a carcinogen by both IARC and the National Toxicology Program. That includes chrysotile, certainly with regard to lung cancer mesothelioma. There's no question with regard to the carcinogenicity.

IARC also determined that there was sufficient evidence in human studies for cancers of the larynx and ovaries and limited evidence for colorectal and in the pharynx and stomach. And there have been a number of reviews of cancers at sites other than the lungs for asbestos. I think this determination by IARC is reasonably consistent with the data that exists, largely with regard to cancers of the GI system. Studies that show an excess risk of about two for lung cancer tend to show an increase, not a two, but an increased risk for GI cancer.

I'm going to talk a bit about the risk assessments that we have for asbestos. Nearly all of the risk assessments are based on populations occupationally exposed. Again, as discussed before, the measurement method is this phase contrast microscopy where the fibers longer than five micrometers in length are counted.

The typical metric is cumulative exposure expressed as the product of duration and concentration measured in fiber-years. I want to point this out because a lot of the data upon which risk assessments are made is really occupational groups with short exposures which are relative to high concentration, including the studies that our group has done of chrysotile-exposed textile workers.

Many of these workers had exposures of just a few months and nonetheless showed increased risk. Most of the models, including our own, were no-threshold models; that has been discussed already today. They seem to fit best to the actual data. And lastly, a point that needs to be emphasized is that there's no scientifically justified threshold for asbestos-related cancers, none that's been established in the literature

by recent studies.

Here are the limitations of the risk assessment, moving to the next slide. Historical measurements, as I said before, a lot of them were based on the old impinger method and unless you had some data to make a statistical conversion between the old method and new method, there's lots of misclassification in the data. And in most cases, in these types of studies, that tends to actually dampen the exposure-response relationship. So your effect is likely greater than you are actually showing in your data.

Again, the risk assessments were based on the phase contrast method wherein only a fraction, and typically less than ten percent of the actual airborne aerosol was actually measured. And as I said before, that's because of the diameter limitation of the PCM method and because of the decision to count only fibers longer than five micrometers. That decision is really not based on the decision that short fibers are without risk.

It's based on the fact that a practical method hasn't been developed for measuring exposures and enforcing standards. And NIOSH, in its 1972 criteria document for asbestos pointed out that the reason for the five micrometer cut was for reproducibility of the PCM count.

Lastly, mesotheliomas are not well captured in a lot of the mortality data that's been published at least through 1999. There was no code for mesothelioma specifically. Only in ICD-10 do we have a specific code for mesothelioma, so a lot of the mortality studies, including our own, looks at things like cancers of the pleura and assumes that those are mesotheliomas. And that's a reasonable assumption in most cases but likely does not capture well in other cases.

Next slide. I wanted to drive home the notion about what portion of fibers are actually counted by phase contrast microscopy. This is actually a slide from some of our data from a textile operation where they're using very long fibers, the best grade chrysotile. And even in textiles, if you look at this distribution of diameter to length, you see that the vast majority of the fibers are short and thin. So that's the nature of exposures, even occupational.

Next slide. I wanted, last, to point out two studies that have been published subsequent to the current risk assessments used for the OSHA standard. The two case-controlled studies, and these were for the mesothelioma, one in France and one in Germany, and they are of

reasonable size, particularly the France study. And what these studies are showing is that we now have measured excess risk of cumulative exposures that is fiber-years. In the France, study in France, less than one fiber-year.

Likewise, in the study in Germany we have an -- about an eight-fold risk for fiber exposures that are less than 0.2 fiber-years. There is a, I think, a legitimate discussion in the literature about the relative ability of chrysotile versus the amphiboles to produce mesothelioma.

I think, first of all, there's no question if chrysotile does produce mesothelioma. Whether or not it's less potent then amphiboles is a, I think, a subject for considerable debate.

Next slide. Lastly, I want to point out that the OSHA PEL, which is being used as a criterion in some of the assessments of the air samples from the World Trade Center on 0.1 fibers per cc as an eight-hour time-weighted average is not without risk. OSHA's risk assessment indicates that at .1 fibers per cc over a working lifetime, there's an excess risk of 3.4 cancers per 1000 workers, and of those 3.4 cancers, about two-thirds of them are lung cancers. The other third are mesothelioma.

So, the point is that we don't have a threshold for the cancer-producing effects of asbestos, including chrysotile. It's open for discussion.

DR. TALASKA: John, Glenn Talaska. Thank you very much. I've got a couple of questions for you on -- you cleared one up right at the -- in your last slide. I wanted to know the relationship between the numbers of lung cancers seen with asbestos exposure documented versus the number of mesotheliomas, and you said the ratio is about two-thirds to one-third.

But I also wondered what it was in terms -- if there were any data in terms of latency time relative to those two diseases.

DR. DEMENT: Well, I think the latency times are as Glenn just pointed out. Early in the lung cancer, in our own studies, we started to see a pickup in the relative risk, between 10 and 15 years and it really starts to escalate after about 20 years.

Mesothelioma has what appears to be a longer latency in many cases. The peak of that probably, in most states, hasn't occurred until 30-plus years after a person is exposed.

DR. TALASKA: Thank you, and I have one further question. You didn't talk about it. I am only going to mention it briefly in the next presentation, and I hope you will join me in the discussion then of the

interaction between things like PAHs and asbestos. Do you want to give a little -- if you had some information you could provide us right away or would you -- we could wait until after my talk, because I am going to just mention it briefly.

DR. DEMENT: I'll mention it briefly as well. I think in lung cancer, there's clearly an interaction with PAHs and particularly smoking. The question is whether or not that's a multiplicative additive or less a multiplicative fact, and I think most individuals, it may not be multiplicative but it's more than additive, so there is an interaction there. I guess we can discuss it later.

DR. WARD: Other questions or comments for John? John, I -- one question I had was if in the two case-controlled studies with mesothelioma, it was hard for me to conceptualize, you know, how small those units were. Can you help, I mean, can you compare it to like what a typical occupational exposure would be?

DR. DEMENT: Well, these levels are, if you look at the fiber-years, most occupational risk assessments are based on a 40 or 45 year lifetime risk, working lifetime risk. So if you take the current OSHA standard of .1 fibers per cc over a 45 year working lifetime, that's 4.5 fiber-years. These data, these case-controlled data, are clearly demonstrating excess risk at exposures that, cumulative exposures that are much less than that, which just really adds to the conclusions of the OSHA risk assessment. That is, these are not zero risk standards.

The OSHA standard includes lots of work practices in an effort to try to get exposures as far below this .1 fibers per cc as possible. The other thing I like to point out is the occupational cohorts. There are cohorts, including ours as I mentioned before, that do demonstrate excess risk with short-term workers at relatively high levels of exposure, of course. The one that was done in Paterson, New Jersey by **[identifying information redacted]** in Mt. Sinai many years ago demonstrated that individuals who worked down in that plant with one month of exposure producing asbestos, they had a significant excess risk of cancers, including lung cancers and mesothelioma.

DR. WARD: John, can you comment on half-life? I mean are the -- I mean, I know that different types or lengths of asbestos would have different residence in the lung, but is there -- I mean, there probably have been studies looking at pathologic specimens of workers exposed to asbestos. I mean, does it tend to stay in the lung for a long time?

DR. DEMENT: What it does -- there is some discussion, certainly in the literature with regard to the clearance rates of amphiboles versus chrysotile, and in general I think the amphiboles cleared less quickly than chrysotile.

There was a study done at Mount Sinai by **[identifying information redacted]**, who suggests that the clearing of chrysotile from the lung actually ends up concentrating in the pleura where we actually see mesothelioma in the study.

I think the studies that have looked at lung burden are sometimes problematic with regard to chrysotile because of its (indiscernible), and I think some erroneous conclusions have actually been drawn based on lung burden studies when you didn't actually have the estimates of the actual exposures to the individuals.

DR. HARRISON: This is Bob Harrison. Steve Cassidy, earlier this morning, earlier this afternoon, sorry -- I'm on West Coast time -- suggested that the samples may not have been representative of the type of exposures or type of activities that people had. I wonder, John, if you could comment on that.

You said that samples weren't taken, I guess, in the first three days. And then there were lots and lots of samples taken subsequently, but I don't have a clear picture of what people were doing, where those samples were taken, and whether there were other activities where we think exposures were probably higher that were not captured.

DR. DEMENT: Well, I don't have a good sense of that either. My sense of the data itself is that most of the personal air sampling that was done was either done by NIOSH or NIOSH contractors through NIOSH. Those were represented in the publication, I think, by (indiscernible) through NIOSH, and in the slide, where we showed (indiscernible) samples.

A lot of these were actually taken during the post-cleanup operation, but the extent to which they represent exposures of that group is really not known. I mean, an effort was made to do that, but, you know, I can't, you know, I don't know all of the cache that were not sampled.

DR. WARD: Any other questions or comments? Susan.

MS. SIDEL: Hi, John. Susan Sidel. Could you just explain again the different measurements that you used that -- you were saying a TEM is the -- is like the finest but it's also really expensive and it's not OSHA standard. So the OSHA standard doesn't pick up the tiniest particles, and what was used at the World Trade Center?

DR. DEMENT: The OSHA standard is based on the space contrast method.

MS. SIDEL: Right.

DR. DEMENT: So it's an optical microscope with a phase -- a phase illuminator or phase shift illuminator, and the problem -- just go back and place yourself in the 1960s. All of the old samples were collected by methods including (inaudible) with a routine sampling method that would first of all actually measure fibers, if not all particles, and measured a reasonable portion of the air samples.

So this method was the default method, and it measures, even in the asbestos industry, occupationally, it is really just an index of exposure. It's measuring a small fraction of the air blowing aerosol. Because of the limitations of the counting with regard to length and the resolution with regard to diameter.

So, typically, in an occupational setting with chrysotile in particular, because it tends to be more fine, you'd be lucky if you're counting 10 percent. In most cases, you're counting about five percent of the total number of asbestos fibers that are airborne that the workers are actually breathing.

If you move on to electron microscopy, it has the ability to look at these particles, but because of the high magnification, you're actually looking at a very small area of the filter, so you have a lot of statistical variability with regard to the count. It was not chosen as the method for routine occupational exposure assessment.

MS. SIDEL: So the method that was used in the World Trade Center is the method from the 1960s?

DR. DEMENT: Sorry, could you repeat?

MS. SIDEL: So the method they were using at the World Trade Center was the OSHA standard method that you talked about from the 1960s?

DR. DEMENT: No. Yes, most of the samples that were workplace samples. For example, if you look at the slide, 19,000 air samples --

MS. SIDEL: Uh-huh.

DR. DEMENT: Almost all of those were PCM, so they did not use transmission electron microscopy. So it's trying to measure these exposures against an OSHA standard. The NIOSH sampling used PCM, but they did -- didn't look at the ones that were in excess of the .1 fibers per cubic centimeter and looked at those by TEM. Samples which were mostly structures per millimeters squared filter area were TEM.

MS. SIDEL: Thank you.

MS. HUGHES: Hi. I just want to remind people, as a resident that lived one block away, the chaos that was there for a very long period of time, there was no electricity. So if you're going to do sampling or testing and there's no electricity, one of the concerns that some of the testers had was it could be done on a generator, and then you had to determine what kind of generator.

Would you be using diesel fuel, or would you be using a battery, and then where you would get that. So there was electricity on the east side of Broadway but not the west side of Broadway, and so when people are talking about the proximity of the testing, it took some time to actually get the machinery into place to actually do the testing.

And then one of the issues that has been argued about over the years was clogged samples, so the filters were clogged if there was a lot of material that was actually picked up. So I just wanted to remind people what it was like early on. Thanks.

DR. DEMENT: Those are good points to make. I think given a relatively low percentage-wise of asbestos in this material and the high concentrations of dust, one of the issues with regard to asbestos sampling is trying to optimize the ability to count it, and when you run a filter for a period of time, accumulation of dust on the filter can actually obscure the PCM count.

DR. HARRISON: This is Bob Harrison. I just wanted to make two points. I think both of them are probably obvious, but I think for the record, it's worth stating. One is that I think there's evidence that respiratory protection was not available, consistently used, and would not have afforded, in any event, protection against inhalation of potentially carcinogenic asbestos fibers.

I don't -- I'm not sure that there would be any disagreement about that point, but I think it's worth noting and if there's any, you know, any additional comment, we need to make that.

The second is that based on the lung disease that we've seen from other lines of evidence, (indiscernible) airways tends to show (indiscernible) lung diseases. I think we can use that as qualitative evidence that indeed inhalation of particles and fibers and smoke, et cetera, did occur.

I don't think we can make any correlation between those clinical effects and the dose of asbestos, but I think just qualitatively, we know that

this population had inhalation exposure, and I just think it's important to point that out as well.

DR. MIDDENDORF: John, this is Paul. I just want to ask if you would take a minute or so and address the issue of potency related to length of asbestos fibers.

DR. DEMENT: Well, I think, Paul, the issue of potency with regard to length, it really comes from some animal data. Now if humans are exposed to the whole spectrum of fibers, and so when I studied my textile workers, they're exposed to the whole dust cloud irrespective of how I choose to measure it.

Some of the animal studies suggest longer fibers are more carcinogenic, and those studies come from some inhalation, but mostly studies that are implantation are injection studies, some of the early studies from Merle Stanton at the National Cancer Institute, for example, and Dr. Hoch (ph) in Germany.

So with regard to cancer, I think longer/thinner may be more carcinogenic, but in the exposed aerosol, even if you consider an asbestos textile, the longer/thinner comprise a very small portion of the airborne exposure.

So I think the -- in terms of the actual effect of short fibers in that they greatly outnumber the long thin ones, even if fiber for fiber, they were a fraction -- had a fraction of the carcinogenic potential, I think the data doesn't support leaving those out with regard to risk assessment. We just completed a series of studies in the plants that we've looked at for many years in South Carolina and in North Carolina, and we did these in collaboration with NIOSH where we had the ability to go back and look at some of those old filters in the 1960s and to try to estimate a sort of size specific exposure measurement for these workers in these two cohorts and try to relate that to risk.

And when we did that, we found that all of the size categories by length and diameter correlated and predicted lung cancer risk. It's -- the longer, thinner fibers, when you look at them had a slightly greater impact; but nonetheless, all sizes that we were able to measure, including the short thin ones, impacted lung cancer.

DR. WARD: Any other questions or comments on this presentation? Thank you very much, John. We hope you can stay on for some more of the discussion. We appreciate you coming.

DR. DEMENT: I'll plan on staying on. Thank you.

DR. TALASKA: Okay, are we ready? How does that sound? Good? Everybody okay? Okay.

Well, I wanted to begin by making a statement about how being able to look at these data in detail, really it changed my mind about something about the exposure with the -- of the first responders at the Ground Zero site.

When I, as a scientist, and as a regular scientist with an interest in the area, but not an acute one, I looked at the abstracts. I looked at some of the tables, certainly of the ones with biological monitoring because that's my field.

And -- but I didn't look at the papers really hard, and the opportunity that I got today to look at them -- today -- in the past two weeks, at least, and certainly since being on the committee has given me a somewhat different -- considerably different perspective than I've had to begin with, and I will begin with this.

What I'm going to talk about today are the polycyclic aromatic compounds. These are the materials that are formed by the burning of any material as a fraction of the total mass of the stuff that's burned. Most of the stuff goes to carbon dioxide, but if there's not sufficient oxygen to go to complete oxidation of it, then these benzene rings fuse and form large plate-like structures that I give you three examples here. These are materials that -- from any kind of burning. I'll show you some pictures. PAHs are very lipophilic materials. They're well absorbed from both the lungs and the skin when they're contacted and from the GI tract, although there is a difference with the GI tract relative to these compounds that I'll get at later.

Just some examples from the occupational world first. You can see from here -- there it is -- that the upper left panel shows a coke oven. This -- the worker here is a topside coke oven worker -- these two workers. One of them is more obscured by the smoke than the others.

These are occupational exposures where we have both the knowledge of what the internal dose was for these individuals and the lung cancer risk, which is at excess. These people are in the worst possible situation because you're trying to make coke, not Pepsi-related coke, but coke which is used in steelmaking out of coal.

So it's burned in the absence of oxygen or almost the absence of oxygen and forms a dense smoke which escapes from the machine. It's a very

large structure. The right-hand panel is a foundry. And you can see, again, the hot metals are producing smokes which can be seen. The lower right-hand panel is an aluminum manufacturing site. At this slate, they're pouring.

The left one is extremely interesting from several points of view. One is it's a food product. Our PAHs are in many of the foods that we like. Barbecue, smoked foods contain PAHs from the prioritization of the materials, and we eat them.

But also look at this here. As you can see from closer examination of the walls of the smokehouse that this guy is in smoking fish, that the whole structure is coated with a tar-like substance. And those are -- that is often high in the -- very high in the PAHs.

Other examples are shown here. This slide shows an asphalt operation that we've all smelled. The materials that are coming off the gassing of the asphalt as we, you know, our body -- I think everyone uses orange barrels. And so the workers are exposed there.

One of the real advantages of the studies that have been done very much by NIOSH but with other players as well is that often times they will take area samples of areas near or around a -- some of these operations and then conduct personal samples at the same time. And that becomes important to us.

In the right-hand panel is the classic PAH exposure that causes lung cancer in cigarette smokers. Seven to ten-fold excess risk, depending on how many packs are smoked. It goes up with a various dose response that most of the toxicology is envious of, but it's from a very sad point of view that this is the major carcinogenic material in the United States and the world for causing lung cancer.

PAHs are also formed with the burning of any material, so the nasty smell that you get when the smoke comes your way at the campfire contains some of those materials and that's the stuff that stays on your clothes the next day when you realize that, you know, those were in a bar or where there was smoke.

The lower right-hand panel, of course, shows a more recent disaster caused by -- during the blowout last year of the oil rig in the Gulf, the Deep Water Horizon. And you can see -- and this is important from -- for our discussion because you can see two things. One is that here is where the closest you can get to this thing to do any sampling at all is the distance, several boat lengths between the fire and the -- and the

source of the burning itself.

And then you can also see the huge difference, if you collected a sample here, what would be the exposure level relative to what it would be if someone was at or near the plume? I'm not making direct comparisons, but keep this model in your mind is what I'm saying there.

And now we have the World Trade Center and slides that I have -- a couple of slides just to illustrate things about the smoke. Here we have a burning smoke which is -- probably has PAHs in it, almost certainly, and then the more general smoke that occurred, I believe, right after the collapse where the -- probably a multitude of materials in this one. Also important here is that at this point you can see there are civilians inside of this where they -- where the work is actually being done. Now, I'm not sure, and I have to tell you I don't know as well where the monitors were put at Ground Zero relative to the work zone.

And -- but that's extremely important. Even at this point, you can see your, you know, the smoke is going up. Oh, that was the other thing with this one. I'll go back a minute. The smoke is rising here very rapidly. Persons that are in the plume are being heavily exposed, but persons very, just to the outside of it, outside of the convection currents that are occurring, are not being exposed to the same levels. Nor would any monitors that are placed in that area be exposed to the same level.

Okay. PAH exposures are associated with lung cancer in tobacco smokers. It's thought that 70 percent of the lung cancer in the United States and the world is due to tobacco smoking. Coke oven workers are also at increased risk. Aluminum smelter workers are. And the classical exposure to -- of soots, dermal exposure on the scrotum in chimney sweeps was investigated by Percivall Pott in 1776 and associated with the soots that were -- people, kids mostly, who were exposed to that by actually being run through the chimneys at the time.

The PAHs are absorbed by the body and they are metabolized to compounds by the body that combine to DNA. So PAHs themselves are not carcinogenic. It's the PAH metabolites that are carcinogenic, bind to DNA, and cause mutations that initiate the carcinogenic process. So it is biologically plausible that PAH can cause cancer if there is sufficient exposure.

What are the sources of combustion materials at the World Trade Center? This has been reviewed in a NIOSH document, and I'm just

showing it for you.

There was approximately 90,000 liters of jet fuel, 500,000 liters of transformer oil, 380,000 liters of diesel and heating oil, and approximately, although no one knows for sure, the same amount of gasoline which was burned in the parking structures when the towers collapsed and over the next several days as those cars heated up and exploded or were demolished and then the gasoline leaked all over the place and then burned.

Area samples were collected and for PAHs specifically, not for dust in particular, but for PAHs in particular, were collected at the fence line beginning on 9/16 through 9/23/01. There were no personal samples taken at this time by these investigators. So the first samples seem to be five days after the exposure. There were biomarker samples collected once on October 1st, approximately, in a study that was reported by Edelman et al in 2003.

But I think it's also interesting, and I'm going to bring up the set of studies that I found in the Butt et al 2004, a Canadian group who looked at the window films and extracted the materials from the films of windows at various places in New York City and found considerably different levels of PAHs on them than were collected in the air samples.

So these are the data of Pleil et al at the fence line, and again, area samples. You can see many samples were collected throughout.

Samples were collected at the perimeter of Ground Zero, not in the work area, but at the perimeter and again, no samples for the first five days.

They were also collected distally at Broadway, so away from the site.

And one of the things that you can see clearly is that these two exposures have parallel curves. They run together down here, but they're parallel pretty much out here. So we have a difference between the two of them by at least a factor of two because based upon the distance.

So -- but again, they were area samples, stationary samples collected not following any particular worker, not following any particular activity at all, but sitting at the fence line, some distance from where the activities were being taken -- taking place.

So all of these samples are -- were air measurements and estimates based on area samples collected at the fence line, and these types of samples typically underestimate worker exposure and the differences

can be anywhere from three- to 40-fold, that if you take an area sample at a periphery, depending on how far away it is from the active sites of the workers, it generally is known to underestimate the exposure.

Now, that difference can be even greater than 40-fold, but it can be less than 40-fold as well, and the way that it can be less than 40-fold is if the study design uses an area sample to capture the worst case. So many times in my career, I've stationed an area sample in the worst possible exposure place where there are no workers, but to capture the worst-case scenario to see -- and the idea being if there's no problem at the absolute worst designed place, then there might not be a problem where the workers are.

But one has to consciously design their study to do that to be able to catch a worst-case scenario, and I don't believe that was done in the studies that were collected. Secondarily -- so we have a difference here that could be fairly large. Secondarily, only the PAHs that were in the particulate phase were counted because they captured the 2.5 micron samples, extracted those samples.

There's also PAHs in the vapor phase. PAHs, if they're heated, turn into a vapor, like steam, and then that steam rises into the air. And that is -- sometimes it binds to particles and it does bind to particles, but some of it stays in the vapor phase as well.

And depending on the type of study -- in Burstyn et al there was -- they found 10 times more PAHs found in the vapor phase than asphalt workers, but other workers have seen things much lower.

So they have seen 10 times more in this one study, but Quinlan et al, for example, in coal liquefaction workers saw that the amount that was in the particulate, bound particulate, was about equal to what was found in the vapor phase. And there are estimates all over the place between those extremes.

Okay. So what effects weren't measured? Well, the first question is what is the impact of being in a plume and how much more would that be, and how much greater, and again, I refer you back to the picture for the Deep Water Horizon.

If you're working right above the smoke as opposed to being away from it at the periphery, then the -- what would be the impact? And I have -- unfortunately, I wasn't there, and I can't tell you.

What is the effect of exercise and exertion, and I'll show you a slide about how important that can be. But if somebody is working hard,

they are breathing hard and they are breathing several times more than what the, on average, if I am working really hard riding a bicycle or jogging, you know, the worst place to jog is along city streets. Fortunately, the lead's out of gasoline but, you know, the worst place to jog is around there because you are breathing several times more and that means you are breathing more of this material into your lungs where they can be collected.

So that's an impact that one might want to consider, especially if different groups of people were working harder. From what I can gather, and I think in the paper, in the Pleil et al paper, they estimate that -- the purpose of their sampling was to look at some general environmental effects. They weren't looking for what was happening to the workers at Ground Zero, okay, so -- and they made no attempt to capture the peaks or assess exposed worker exposure, and they stated specifically that exposure to the workers at the site could be quote, much higher, end quote.

So there is a big weakness with the best PAH studies that were done at the site, and now -- oh, yeah, but here is something that I believe is illuminating as I was going through the voluminous literature that was provided us.

Butt et al did a series of studies where they washed windows with solvents, and they washed the windows to be able to extract the PAHs and other materials. They were looking for PAHs on them, okay? And what they saw was that there were different zones and -- as you might expect.

So within one kilometer -- they are Canadian after all -- which is 6/10 of a mile, the average was 77,100 nanograms per square meter. We were seeing in the other study, in the Pleil et al study that they were talking about 35 nanograms per cubic meter, so a meter is three feet approximately by three feet by -- a cubic meter is three feet by three feet by three feet. A square meter is three feet by three feet, but on average, Butt et al were seeing on these window films which admittedly collected samples for several days, they -- I forget the day that they collected them on -- they were considerably higher, thousands of times higher.

In fact, downwind sites within one kilometer averaged 130,000 nanograms per square meter. Upwind sites were much lower, averaged 18,500, still within a kilometer. Upwind sites that were greater than

two kilometers away averaged 6000, and this might be considered the background for New York City windows, okay? More than two kilometers away, and upwind, so the wind from the site probably wasn't blowing very often on these windows.

So you can see the types, now, you know, you can't use this for exposure estimates, obviously, but these are windows that may or may not have been in the major plumes at all. By luck, they sampled these, and I don't believe they had any selection other than they had access to the buildings. So I thought this, this was illuminating to me.

Here's some of the data about work rate. So, if you are working, light work is what we consider for most of our standards where the work load in watts is about 50 watts that the alveolar vent -- so, at rest, the people that are in this room are breathing in about five liters of air per minute, but someone who is working very hard can breathe seven times that. So they bring in seven times the amount of air. They pump the blood around much more efficiently. And so you can see the exposure metrics can give you another twofold over that if you're worried about heavy work as opposed to light work in terms of the amount of air they're breathing in and the potential for absorption.

Okay. So now I am going to change gears a little bit and switch to the biomonitoring data, and I have to tell you I am going to focus on one compound, pyrene. Pyrene is one of those PAHs that was in the first slide. It's an important component of PAHs. It -- of -- and it's representative of the four and five ring carcinogenic PAHs, okay?

So, of all of those type of compounds, pyrene is the most abundant. So it's oftentimes the easiest measured, and we do have a biological exposure indices for 1-hydroxypyrene, the major metabolite of pyrene, which is an ACGIH BEI. That was developed in -- I'm not sure it was in place in 2001. It may have been. We'll have to go back and check that when we think of it.

But biomonitoring can account for differences in absorption, distribution, and metabolism and elimination if it's done correctly. It can take into account both the skin and inhalation exposures and one very important thing with biological monitoring is that exposure can be reconstructed.

If you know the material that you are exposed to and you know the half-life of that material in the body and you know the relative time between when the sample was taken and when the exposure occurred, you can

reconstruct the exposure based upon the half-life.

On the other hand, it is a method that is easily misused, if not in terms of interpretation, if you don't know exactly what you're doing, so.

Let's look -- and this is an example of a biological monitoring on a model system. This has nothing to do with the Trade Center. This is just a model that I made up. So you see if you have exposure on Monday morning and the exposure during the day on Monday equals to a hundred, and the half-life in the material in the body is 24 hours, then the material -- you will increase the amount in the body, and then in the 16 hours the person is off until the next shift on Tuesday, that level will decrease by a fraction based upon the half-life.

So you can see right that you get a -- with each additional day, you get an increase, but it's not a doubling. So you don't get 200 on Tuesday; you don't get 300 on Wednesday and so forth. And then the other thing to notice is that because of the half-life -- and what is half-life?

Half-life is -- most of you probably know -- is the length of time a material resides in the body. Most of the materials that are absorbed by humans as xenobiotics are eliminated. And they are eliminated fairly rapidly because the body doesn't want to keep these things if they do nothing for it. I mean, some materials have long half-lives; cadmium has a 30-year half-life. Lead has about an eight-to-ten year half-life in the bone. But these materials tend to be eliminated fairly quickly and with fairly well-defined half-lives.

Notice what happens after work on Friday. So after work on Friday, the level in the body goes way down before Monday morning, and that's because there are several half-lives involved here, okay. So when would be the best time to sample for this material, something with a 24-hour half-life?

Now you wouldn't want to sample on Monday because the body hasn't reached steady state yet. Oh, and by the way, this continues every week. It doesn't get much higher. It never gets above 200 for this compound as long as that dose is the same.

When would you want to sample? Well, you don't want to sample here. You really want to wait until the end of the week. Sample in here and you'll have less variability, and you'll capture the exposure because that's when the exposure reaches its peak.

You wouldn't want to sample down here at this time because that would -- without knowing when the peak occurred -- because that would

underestimate exposure dramatically. So let's look at the data. These are the 1-hydroxypyrene data from Edelman et al, and this is one table I looked at, and I'm only giving the 1-HP data. And I've changed the numerals that have been used, and in that I use micrograms per liter and I'll tell you why momentarily.

They use nanograms per liter. Micrograms give smaller numbers, fractional numbers, but it's important because the BEI is set at one. Okay, so all exposed workers at the site when they were sampled on October 1st, 2nd, or 3rd had a level of 0.092 micrograms per liter. The controls had a level of 0.062 micrograms per liter, and that seems like a small difference, but it could be a significant difference and it was in fact significant. It was significantly higher.

If the firefighters were at the collapse on day one, then their average was about .11. If they were -- if they didn't come at the collapse, but came after the collapse on day one and two and started working, then it was slightly, slightly higher, so maybe if you could say the real fires that were happening at ground level didn't happen until here, at least in the majority of the -- after the collapse. That's when all hell broke loose.

There was a subgroup that was studied which was called the Special Ops Command, and they were considered to be the highest exposed, and indeed, they had the highest average level. Their level was .159.

Okay, now the reason when I looked at these data initially I thought that well, you know, you can see there's a significant difference here but it's not a big deal, was because the standard that occupational exposures are based on, the level is 1.0, okay?

So the occupational standard is much lower, but it specifies an end of shift, end of workweek sample and as I found out by reading the paper hard, one, they did not capture the peak. Samples were collected 20 some days after the exposure, which would be -- and also they reported no variances and other people can maybe reinforce this, but when we were worried about people who have exposure, it's the outliers that are really important, and the outliers weren't given in the paper.

Four percent were said to be in the upper five percent of the NHANES values, but I wonder how many of the controls were in the same upper five percent. It wasn't represented. Because then there's no comparison there. But there was no variation given. There was no standard deviations, no ranges that were given in the data, and no exposure time was indicated or no sampling time was indicated. They

did not indicate whether they sampled at the end of the shift, at the beginning of the shift or when they sampled at all. It's just unknown, and that really threw me, okay?

So we have a situation where the exposure may have occurred many days before and also -- and so you would expect them to be relatively low relative to the decrease in exposure that one might see with that decrease in the PAHs that were reported.

Going back to the -- if I may, this slide. So, regardless of what the true levels were if these were just area samples, you can see that the shapes of the curve are similar. So one may anticipate that if there was a higher level inside of Ground Zero, then it would follow a similar shape, so the levels that -- this is when the -- the highest level would have been reported here. The first samples weren't taken to here, out 25 days, and you can see what the shape of the curve looks like in terms of the exposure. It's already winding down at least.

Now how can we -- can we do anything with this data and -- okay. So the sampling time wasn't given. Firefighters -- and this is from my own experience that firefighters haven't -- in the studies that we've done in Cincinnati, the firefighters have a higher level after a fire than before, but generally they are not in the really high exposed level and I'll give you an idea of what that means here in a moment.

And then the question becomes are -- could absorption from the lung be complete? What about the large particle masses and the fact that PAHs might not be absorbed rapidly, and I'll show you some data on that in a moment.

So first things first. This is what happens in a workplace in an aluminum plant, and I showed you what those look like. In aluminum plant workers, and their exposure to 1-hydroxypyrene. These samples were taken pre-shift, so there was a baseline sample taken every morning and an after work shift, and you can see that their exposure follows the model for a 24 -- very similar to what I reported earlier.

But look at the magnitude of their exposures. By the end of the workweek, these levels are greater than 10 micrograms per liter -- per liter of urine, which is 10 times the standard. But notice that every day before the shift, they drop down considerably, so that if this is the peak -- and what this shows is that like in many workplaces, aluminum reduction workers don't produce as much on Friday as, you know, it's Friday.

But you can see that after Thursday's peak, that there is a significant drop in the 16 hours between the next day. So if you didn't sample, if you sampled in the morning, you would see a much lower sample by design, much lower level by design. And these are data that were developed by the BEI committee in running up, in developing the BEI for 1-hydroxypyrene.

And what they show -- it looks complicated, but what it shows is how exposures could be the sum of all of the different compartments for these things. It's known that PAHs have three compartments in the body: the blood, which is cleared very rapidly with a half-life of five hours; the lean tissues, which are cleared within 24 hours; and then the -- probably the adipose tissues which are cleared very slowly, just every -- the half-life is 23 days approximately.

And so what you see is that with every exposure, the major impact on the urinary levels shown in black is the sum of the three of them, but it's largely dependent on the lean compartment and the -- and what was in the blood, and then that rapidly disappears causing a drop in the urinary levels.

This was an example I found extremely illuminating for this discussion. This was a group of people, patients in this case, who go to the Mayo Clinic for what's called the Goeckerman treatment where they have psoriasis, and their skin is painted with as much as 70 percent of the total body volume of -- their skin is painted with coal tar in the treatment of psoriasis. It apparently works.

And what I'd like to focus on -- the slide is more complicated than it needs to be. I'd like you to look at the -- the values here for 1-hydroxypyrene. So these are the baseline values in this group of people. After one treatment, that baseline jumps up to 170, okay? Now this is applying it on the skin.

After five treatments, because they're given eight hours a day of this treatment, five days a week, and then it's stopped. After five treatments, it goes up to 270, approximately, but after one week of no treatment, this is the level. And it goes down -- remember there's a break here between 10 and 100 -- and it goes down between 275 and down to less than 4 within a week.

If you calculate that, that means that the half-life for this is about 24 hours, which is very consistent for a group of people who haven't been exposed chronically. Their exposure was just five times. So it drops

very rapidly with an apparent half-life of about 24 hours.
Why this is important is that if the half-life was indeed 24 hours, one could back calculate from the levels that are given to the levels that may have been at the peak on 9/11, 9/12 at Ground Zero.
What this slide shows is the data from Gerde et al, who looked at the impact on particle size. PAHs were absorbed onto particles and then they -- and then they modeled it into the lungs based on -- and then actually did actual measurements in the lungs, and what they saw was the smaller the particle that the PAH was held on to -- so these are particles with PAHs on them -- when they were deposited in the lung, a very small particle had a very short half-life.
So if it was .1 micron, the half-life is approximately less than a minute, probably 30 seconds; but if it was a very large particle, the half-life could be more -- much more extensive. So we're talking on the orders of a month or greater if it was 1000 microns.
Now how might a particle get to be 1000 microns in the lung? Imagine that -- and what we used to see in tobacco smokers was that you'd get these agglomerations of tars at the bronchial -- where the bronchia would split and tars would accumulate, and that makes the particle much larger and makes absorption from it much smaller.
So the idea is that an exposure even one time can result in a very prolonged exposure based upon the fact that it comes off a larger particle much slower.
Then there's the part of how with the amount of deposition, and I'm not going to go too long in this, but what it really shows is that if you breathe regularly, you -- regardless of the particle size, this is the fraction that's collected and deposited in various areas. But if you breathe a lot faster with a much higher tidal volume, breathing in deeper, then you're much more effective at collecting particles. So people who are working harder not only breathe in more air, but they also deposit much more readily.
So PAHs do absorb on particles. Soot, particularly, so on diesel exhaust and those types of things, they -- because of their lipophilicity, they are very much attracted to those soots. But they are also attracted to concrete particles, and that's been shown in the literature, to a lesser extent, but still, they're absorbed onto the particles and then deposited and held in the lungs.
The particles may accumulate in the lung and slow their absorption into

the body, and particles may be coughed up, expectorated, spit, or swallowed, but this, in fact, seems to be more of a detoxification pathway than an exposure pathway for a complicated reason dealing with the liver first pass. Okay, you know what I mean, but...

On the other hand, PAHs have known to interact with other exposures. PCBs and dioxin were found on the site. In fact, the highest ambient level of dioxin ever measured was measured in the world after 9/11. Dioxin is known to be used as an enhancer of the carcinogenicity of some PAHs, so if animals are treated with dioxin, they are more likely to get tumors than if they're not treated with dioxin and given the carcinogen.

Silica is something that we haven't mentioned too much, but PAHs are known to enhance the carcinogenicity of silica exposure. And in this case, when I'm talking PAHs, I'm really talking smoke. The interaction seems to be additive or additive plus, and then unlike what John mentioned, the data that I looked at saw that PAHs, again, smoking, enhanced the carcinogenicity of asbestos, but at least the studies that I -- the consensus was that it was multiplicative but I would certainly -- he's much more experienced in this than I am.

So the conclusions that I would make are that exposures to workers to PAHs within the Ground Zero site was almost certainly higher and maybe substantially so than was indicated by the majority of exposure studies. A fuller report of the biological monitoring data is needed to predict what exposures may have been during the early periods after 9/11 and who may have been at the highest exposures.

The people who are the outliers are the key. If the people who had the highest levels of 1-hydroxypyrene are the ones who later -- they have the highest dose, and they may be the ones who are at the highest risk, and understanding who, not who the outliers are from our point of view, but what the range of the outliers were and then moving that back is an extremely important thing, at least in my mind.

And if the effective half-life is 24 hours, then the 1-hydroxypyrene levels on 9/12 could have been well above the BEI assuming that there was no exposure, assuming that there was no exposure. Now, that's not the case. There was exposure afterwards.

The best thing to do would be to model that exposure, and the half-life would be -- with the curves that were used in the exposure studies. You'd have to integrate those together. I didn't have the time to do

that, and I -- yeah. It's something that one could do, though. Thank you.

MS. FLYNN: Thank you, Glenn. A quick question. What would the exposure metrics be for a 10-year-old child?

DR. TALASKA: No idea. I'm sorry, I shut it off, and I killed it. I've got it. I have no idea.

MS. FLYNN: Because in general, as I understand it, and maybe Leo could comment on this, but children actually take in more air than adults, so I wonder --

DR. TALASKA: Well, again, and you do have to realize that at the fence line, they were measuring those exposures and the exposures were tending to rise. I can't tell you, but kids weren't inside of Ground Zero, okay, so I don't know what the exposure would be because the data are so -- but kids tend to breathe more. They have larger surface area relative to their body, so they do tend to sometimes take in more materials. They do eat things.

MS. FLYNN: Kids were not inside of Ground Zero, but, we actually, you know, do have available -- I'd have to find them on the site, the High School Parents Association website, but information that show that on days when debris was being dumped on the hazardous debris barge outside of the Stuyvesant High School ventilation system, the particulate concentrations were comparable to Ground Zero.

So, I mean, there were lots of -- there was just tremendous potential for different kinds of exposures that have not been captured in the data, so we just -- this is something that -- I know I sound like a broken record, but I think it's really, really important to keep in mind number one, number two. Children were caught in the dust cloud in the initial collapse cloud, so I don't know if Leo if you want to add anything.

DR. TALASKA: I didn't look at that. I'll be honest. I was focusing -- there was more than enough here to cause me to -- so I really didn't look at that in a really hard way.

MS. FLYNN: Can I just make a plea on behalf of the stakeholder members of this panel? We actually -- we're not experts and we obviously defer to the scientists here, but we're equal members of the panel and we know a lot of things because we've been basically engaged with, you know, the facts on the ground from the very beginning.

So if it's possible for us to have in advance the drafts of your presentations -- I'm sorry I keep popping my keys -- the drafts of your

presentations, that would be tremendously helpful. I know that Susan Sidel provided extremely valuable information to -- to Virginia Weaver, and we want -- we didn't want to load you guys up, because we know that, you know, you're like, you're trying to condense a tremendous amount of material, but there were times when we actually can bring a useful perspective and we really appreciate that opportunity.

MS. HUGHES: Also, it seemed like most of the sampling was done at street level, and if you look at the topography downtown, it's surrounded by very large skyscrapers. So if the plume actually expands would the results of the testing might be different higher up? You have families living in these high rises in very close proximity, so I just wanted to mention that as an exposure route.

And the second thing is, it wasn't as though the only fire was where the two towers were. It spread, and you had gas lines feeding -- pardon me -- but there was gas lines feeding the World Trade Center site. So there is exposure within the area, and it went on and on and on, so I just wanted to put that in for the record.

DR. WARD: I suggest -- a suggestion, we are running late, and maybe we'll take one more comment and then we'll have a 10-minute break and then resume, because we do have a fixed time when we need to start the public comments.

DR. MARKOWITZ: So, John, John Dement made a point on discussing asbestos as there is no known safe threshold. So the question, since you frame the exposures among the firefighters around the biological exposure index, what's the relationship between the BEI and cancer risk for PAHs?

DR. TALASKA: It's not really known. The BEI is based upon specifically the level that is associated with occupational exposure if you -- and not with environmental exposure. There wasn't sufficient data to be able to say that there was any level of -- that was related to disease yet.

There weren't simply enough data there. There are data that shows at that level since then -- we've put out -- we've done studies showing that at the level of the BEI of one microgram per liter, there's an increase in PAH, but we don't know what it is relative to cancer as of yet.

There aren't sufficient data, but -- so that the level was set just so that it would rule out things like tobacco smoking because you can't get -- smokers don't have levels that are that high, as high as you want. Does that answer?

DR. WARD: Okay, we'll take a 10-minute break.

(Recess taken from 2:52 p.m. until 3:12 p.m.)

DR. WARD: Let's start. I think everyone's, virtually everyone's back at the table and we'll start with the presentation by Bill Rom.

PARTICULATES AND WTC

DR. ROM: Thank you, Elizabeth. Does Paul have some slides? My task is to talk for five minutes about particles, particulates or particulate matter. My job is to talk about exposure assessment, what were the exposures; second, how bad are these particles, are they really toxic or are they not toxics; and third, what is the evidence for these particles in humans, did they get exposed and how much; and lastly, for gravy, are these particles going to cause cancer, since that's the question we have to address soon.

On this slide you see the particles on the left and then you see the fires on the right. The point I would like to make is that there were two kinds of exposures here, but I don't want to make that point so much as to say that they overlap. This was a fire that was extremely hot, that burned the particles, and we have a particulate exposure that really has never been seen before. This is unique. This is a disaster medicine and these particles really can't be classified basically like coming from the mine or source 'cause they've been altered.

Next slide. So this is a grab sample of the dust particles on the right. This is WTC dust but a third of that dust comes from wallboard. So all this stuff that we're seeing right there. So that's gypsum, and gypsum is calcium sulfate. It's not -- it's what we always call with NIOSH, nuisance dust. We chuckle about that 'cause we wonder what it is. Calcium sulfate is not known to be very toxic; it's mixed in with calcite. Calcite has calcium carbonate and calcium carbonate is not very toxic, but it forms little crystals and when you see it in tissue, can actually be birefringent, and that's important to remember in regard to silica.

Third, there is some cement dust mixed in here and the cement dust is calcium hydroxide. And that is a basic salt and it's alkaline, so we know the pH of this World Trade Center dust was around 11 so it's alkaline and it's irritating. It's irritating to the mucus membranes, to your eyes, to your mouth, to your throat, makes you cough. So is that really something that's going to cause lung disease and cancer?

I had the good fortune of being funded by NIOSH to study trona miners, and trona miners were exposed to a sodium sesquicarbonate that we use for the New York Times and Coke bottles and things like that. And the trona mines are in Wyoming, so I had to go to Cheyenne and have a personal interview and get a medical license, and then spend a couple weeks in Rock Springs and Green River with cowboys, and

they would mine trona.

So we studied 230 trona miners and we looked at shift studies to see if they would have a drop in lung function over shift and any alterations in their breathing, and it was really a negative study. So pure trona, sodium sesquicarbonate, is a rather benign dust.

But they all complained of skin itching and dermatitis and irritation, and we got a second paper on just trona dermatitis. So that shows you that alkaline dust can irritate the mucus membranes. So in its pure form these dusts are rather benign. But then you also notice on the left of this slide that a lot of this dust was respirable, less than 2.5 microns, that's not mm, it's microns, so there's a lot of respirable dust that gets down into the lungs.

Last week [identifying information redacted]was visiting us at Bellevue, and we spent an hour looking at eight lungs that were from open lung biopsies of World Trade Center dust exposed people, and we looked for silica and we really didn't see birefringent particles sharp and bright like silica, so I'm going to dismiss silica as really being a critically important particulate exposure to the workers. And I'll point that out by looking at the next slide.

So we've documented an exposure and now I want to go on to the toxicity of these particles. So we had a firefighter who came within the second week of 9/11 to Bellevue who was critically short of breath and ended up in the medical ICU, and he had bilateral infiltrates and effusions, and we didn't know what he had so he was treated with antibiotics and steroids, and was getting better. But since I'm a physician-scientist and I'm the boss, I like to yell at my faculty, I said, you need to get him consented and do a bronchoscopy, you know, lavage and make a diagnosis. So fortunately he agreed to the consent and we were able to get some cells. And he had all those red cells on the right, that's acute eosinophilic pneumonia. So he had a very unusual disease that may be related to dust exposure. The important thing is we got those cells and you can see they're pretty clean. They don't have smokers' particles in them, so we sent these cells on the next slide to Victor Roggli down at Duke to analyze them for particles. And we said, this is a firefighter exposed for two weeks in the Pile, and this is the first lavage, and these are cells from his lung and we want to know what particles are down there.

So first of all, he showed us a fiber, and that's an amosite fiber on the left because he did an x-ray dispersive analysis for elements and found iron as well as magnesium and silica, and pointed out that that's an eight-micron-long fiber. The important thing is it's not coated. It's an uncoated fiber which means it's freshly inhaled, which is very unusual. You never see that in asbestos workers unless they're from the mines in Quebec.

The middle particle I want to point out to you, is what I think is a really toxic WTC particle 'cause that is something that looks like from outer space. I called it fly ash particle 'cause it reminds me of a clinker coming out of a coal fire. But I think that's a burned particle. And in your packet there's an analysis of particles from the Deutsche Bank building, and the analysis shows a lot of these particles are coated with other substances from the fire, and that probably enhances the toxicity of these particles, so that's a burned particle.

On the right is what we think is fibrous glass, and you can see it's not parallel on its sides. It's probably been exposed to 100 degrees temperature so it's been partially burned.

The fourth thing I want you to look at is on the bottom. There's 305 commercial asbestos fibers per ten to the million macrophages. So how much were these people exposed to? So in my tenure at the NIH, I lavaged about 500 coal miners and asbestos workers and silica exposed workers, and I had to do some normal volunteers. So I had eight normal volunteers and they had a mean of 30 asbestos fibers per million macrophages. So this firefighter has about ten times the normal number of fibers in his macrophages. And the asbestos insulators I would lavage would have about a thousand. So he's, you know, just after a couple weeks, he's up to a third of the way to what an insulator has in his lung.

Now, I would say that breathing the air with your nose and your lungs is probably a better measurement than the samples that EPA took, and we couldn't find any fibers in their samples. So this guy was on the Pile and trying to rescue that -- this whatever could be done to save others.

Next slide. So this is what chrysotile asbestos looks like, and the reason there was an amosite particle there, is that in New York, when we put chrysotile asbestos in the sprays and on the steel girders, we always threw in about five percent amosite. Reasons, I don't know why but they always did that so that's why you find a mixture.

Next slide. So this is from the asbestos insulators and the kind of fibers you normally find. That fiber has a coated iron and protein surface and that's what those beads look like. So this is a fiber that's been sitting in an insulator for 20 or 30 or 40 years. And you see the body tries to protect itself by walling off the fiber. And the other cells are macrophages, and this is a nonsmoking asbestos insulator, and there's no other particles in there. So he's a clean asbestos insulator from being nonsmoking, at least. Not clean in terms of fibers.

Next slide. So Dr. Selikoff taught a number of us in this room about asbestos insulators, and his very famous study about all of the North American insulators showed a five-fold increase of lung cancer and almost 10 percent had

mesothelioma.

Next slide. And when I was at the NIH I would spend weekends recruiting patients for a lavage, and I would sit with **[identifying information redacted]** at the Baltimore City Hospital recruiting in study subjects, and he had one of his patients from Sparrows Point Steel Mill who had silicosis, those are the nodules on the right, and he also had mesothelioma with the left, if you reverse looking at this patient, with a big pleural effusion. So mesothelioma is the other disease along with lung cancer that you get from asbestos. How much asbestos causes mesothelioma, I remember when I was working for **[identifying information redacted]** , he had me interview a 55-year-old man with mesothelioma, and he worked in a flower shop in Brooklyn, and I couldn't figure out any reason he got mesothelioma from flowers. And I remember that in Tyler, Texas, the flowers came in gunny sacks and maybe the gunny sacks were used for asbestos. I asked him about gunny sacks, he said I don't know. I never saw gunny sacks. Then I asked him if he worked in the shipyard, and he had worked in the Brooklyn Navy yard for one summer in 1942 as a helper, and had two and a half months of shipyard exposure. So very minimal exposures can cause this disorder.

Next. The marker for asbestos are pleural plaques, the blue and purple around this lung are pleural thickenings.

Next slide. And if you have those, Hillerdal in Sweden showed that if you have pleural plaques, you have a slightly increased risk for lung cancer and an increased risk for mesothelioma, so this is a marker of your asbestos exposure.

Next slide. And importantly, **[identifying information redacted]** would take us to Paterson, New Jersey, where there was an asbestos factory, making fire hoses for New York, and he followed a hundred men who worked for just two months, from 41 to 45 in this factory, and followed them to the end of the 1970s. And on the right you can see with the dotted line that 25 years the lung cancer observed rate increased over the expected, so just for two months of exposure 30 years earlier, you have an increased risk for lung cancer.

The project that I was involved in was doing lung function on the wives of these workers. And I did about 300 spirometries showing that they had a reduction in their spirometry from doing the work clothes washing of their husbands and hugging them when they came home from work from Paterson's factory. And among those wives, four of them ended up getting mesothelioma from that exposure.

Next slide. So Dr. Ward wanted me to go over particles and lung cancer, so the small burn particles that we have from diesel exhaust have been studied in the American Cancer Society cohort. The American Cancer Society enrolled over a

million adults in 1982 about the risk for cancer. But these people lived in metropolitan areas throughout the U.S. that had EPA-collected data on particulate matter of 2.5 microns in size. So almost half of this cohort had data on particulate exposure through the end of 1998 from 1982.

So in the next slide on the left, you can see the lung cancer mortality. On panel A is cardiopulmonary mortality; panel B on the lower is lung cancer mortality. The three circles on the far left are above the line of 1.0 so all three dots are statistically significant over time for an increased lung cancer mortality of approximately 8 percent from PM(2.5) exposure, which is the burn particles from diesel exhaust.

Next slide. And these are what the particles from diesel exhaust look like in macrophages from the lung. This is a collection from sputum in children in England. And these macrophages were looked at under a light microscope and you see the black particles, particularly in D and E, that are very tiny, less than 2.5 microns.

The next slide, we'll skip and go to the slide after it. These are from families, next slide, that did not have any smokers in the household and they were on at least a second level, so they were a little bit away from the street level. And on the slide on the upper left you'll see a declining FEV-1 in those children as they had increased numbers of those particles in their macrophages. Next slide. So these diesel particles cause adverse health effects.

And lastly is cancer. So cancer in the lung starts off as abnormal proliferation and survival of injured cells in the respiratory epithelium associated with genetic defects, whether they are specific genes that are up-regulated, down-regulated, insertions, deletions, mutations, amplifications and so on, that you end up getting a clone of cancerous cells.

Next slide. And the last point I'll make is that there are now ways to diagnose these cancers with a blood test. And you can now target proteins in the blood to diagnose these cancers. On the top in the white are little aptamers, that are nucleic acids designed to pick out a protein in the blood, and you can make more than a thousand of those aptamers to pick up specific proteins in the blood.

And next slide. This assay has been looked at in 1300 lung cancer patients and matched controls, and you can see that a panel of about 13 biomarkers can very accurately pick out the lung cancers with area under the curve of .9. So in looking forward at lung cancer and mesothelioma, there are tests at the early and past research level to identify these people both at risk and of getting the disease. And this test is about to be commercialized for mesothelioma as the first disease to look at.

I think that's it.

DR. WARD: Questions or comments for Dr. Rom?

(no response)

METALS, VOCs and WTC

Okay, is Virginia on the line?

DR. WEAVER (via telephone): Yes, I am.

DR. WARD: Are we ready to...

DR. WEAVER: I am ready. Can you guys hear me if I stay on speaker phone?

DR. WARD: Paul just cautioned me that we only have 14 minutes before the -- before the public presentation -- public comment period. And so why don't we get started and see if we can wrap up your presentation in that time frame and then if necessary, can you come back and we can have questions after the public comment period?

DR. WEAVER: Yes.

DR. WARD: Okay, great.

DR. WEAVER: You have my slides up?

DR. WARD: Yes, we've got the first one up.

DR. WEAVER: So after the title slide, moving to the second slide, I wanted to simply give you some of the thoughts that were going through my mind as I was looking at data related to volatile organic chemicals and metals. And one issue in my mind was the shortest exposure duration that results in a measurable increased risk for cancer, and I've been very happy to hear discussions about increased risk in very short time period. I was not aware. I'm not a cancer expert, and I was not aware about that data, and that's very helpful to us in thinking about risk from exposures that are of -- that occur only when you're actively exposed, which would be the volatile organic chemicals.

The other point that I was thinking about as I prepared these slides are that we are now learning that a steeper exposure rate may result in greater risk, so for the same overall accumulative dose, if you get the exposure faster, the risk may in fact be greater. And so what that means is that the exposure construct for cancer outcome differs from that that's been used in World Trade Center research for pulmonary outcome, so rather than looking at where you were at the time of the collapse and shortly thereafter, we have to think about burning tile, diesel exhaust and carcinogens in dust.

So on the next slide I had simply shown an example of one type of exposure characterization and I know Liz has already showed this type so I'm going to move right on to the next slide on key concepts and questions.

We've already heard that cancer of course varies by time since exposure onset, and so it is the nonsolid tumors that are the ones we could be seeing, even at this point,

from World Trade Center exposures but specifically the leukemias. And then a point that I think others have already made so far is that we have very little data about chemical mixtures overall, particularly in the World Trade Center yet. This is a common exposure scenario overall and of course clearly at World Trade Center. The next slide I simply wanted to show the group 1 and 2A IARC carcinogens that are in the volatile organic chemical category. I took this from NIOSH's summary. I want to point your attention to benzene, which has been classically linked to what we used to call acute myelogenous leukemia but we now call acute nonlymphocytic leukemia as our ability to analyze these types of cancers has improved.

I also want to point out that there is limited evidence that benzene causes acute lymphocytic leukemia, chronic lymphocytic leukemia and importantly multiple myeloma. That is from IARC and it's also supported by a meta-analysis published in EHP in 2008, again, supporting that. Other VOCs that were of concern from World Trade Center would include 1, 3-butadiene, which is a combustion product like benzene, from the Pile and also from diesel exhaust. Again, this has been linked to leukemia and also non-Hodgkin lymphoma, formaldehyde, nasopharyngeal cancer, and there's increasing evidence that formaldehyde is linked to leukemia as well. That's considered strong but not sufficient evidence based on the NIOSH summary and vinyl chloride. And then I've listed some of the 2A, which are -- Group 1 of course is known human carcinogens, Group 2A is, I think the categorization is probable, and it's based on adequate animal data but inadequate or limited human data.

So in the next slide, the important aspects about exposure to VOCs is that they're common in combustion products. I think about this a lot in the work I do for the firefighters union. So you'd think about this from working on the Pile, from the smoke and exhaust from that, and also diesel exhaust.

In general VOCs, as the name implies, are not persistent in the environment and they do not accumulate in the body so the exposure duration would have been while you were actively working on the Pile. But also importantly, these exposures are associated with some of the shortest latency cancers, ones that we could be seeing.

Next slide. As far as I can tell, and I'm no expert on World Trade Center exposures, there are very limited data on VOC measurements. There were grab samples that were taken on the Pile to try and determine if it was safe for rescue workers to enter. So Lorber et al noted that when samples showed, quote, extremely high concentrations of VOCs, end quote, entry was prohibited. I don't have levels about exactly how high those were. Lorber notes that for a number of the VOCs found elevated levels outside of Ground Zero but still within restricted zones, and when

they used 24-hour samples, which should give a little bit better measure. You know, generally in a work place we measure eight-hour samples. When they compared grab samples over four minutes to 24-hour samples, they found that levels were much, much lower for a number of the VOCs of concern, including ones from butadiene. However, that was not the case for benzene. The benzene monitoring showed many more grab samples that were higher and 24-hour samples that, rather than being a thousand times lower, were about ten times lower.

I'm not sure if I said next slide but I have a separate slide on benzene monitoring. And on that slide I included the samples for benzene in 24-hour measurements that were above the detection limit, and so apparently there were only fourteen 24-hour samples that were done for benzene, which doesn't seem like many. Six were above the detection limit and of those, a few were fairly close to the Agency for Toxic Substances and Disease Registry intermediate minimal risk level, which would apply for folks who were working for more than a month, more than 14 days up to a year.

In the conclusion in the Lorber article, which as the data suggests in exposures to benzene at levels that approach the intermediate MRL were not likely to have lasted longer than 45 days.

There's a few samples from truck drivers, done by my colleagues at Hopkins, that were not extraordinarily high either. You know, in the low parts per billion compared to workers are allowed to be exposed a thousand parts per billion.

And I was going to make the point with the text below that the monitoring levels seem inconsistent with the descriptions and pictures of the site, but I think others have already made that point more eloquently before me. There is an inconsistency between monitoring and what was visualized.

So in thinking about the potential implications of VOC exposures, in my mind it would be workers who were on the Pile would be at most risk, and obviously the longer they worked on the Pile, the more risks they would incur.

I was thinking about how much time you would need to work there in order to have increased or measurable increased risk, and with the understanding that probably the exposures were much, much higher than any of the monitoring data that we have. And so I guess it would be a matter of thinking about individuals near and on the Pile and the length of time that they worked in those capacities and that would be how we would consider risk relating to VOCs as an important consideration because this exposure that could be resulting in cancers early on.

And then I'm going to shift gears and talk about metals so that's the next slide.

There are a number of metals that have been associated with carcinogenicity in a

variety of different organs. I've listed those for you here, again, from the NIOSH summary document.

On the next slide, I want to step back quickly and thank Susan Sidel for helping me come up to speed over the course of the weekend on World Trade Center exposures, and I want to just make a disclaimer that this is totally outside of my area of expertise so the metals exposure levels are very complex in World Trade Center. And I tried to, in the next few slides, give you a sense for some of the concerns but I don't have any kind of a conclusion to the extent that I did for VOC.

So on the next slide, Cahill and colleagues have thought a great deal about the metals and other exposures generated at the World Trade Center site, and they've developed an incinerator hypothesis which provides an explanation for the very fine aerosols that were liberated. And a number -- and just basically it would be the temperature that would be involved in these very fine aerosols and there were, his quote, unprecedented levels of several metals. Also, his quote, and this again is from the very fine aerosol chapter in the American Cancer Society book that Liz had referred us to, he's commented that the health concerns focus on workers at the site, as plume lofting protected most of New York City. What I don't know in that regard is the impact on residential -- residences that were very near the site. I know others have commented this afternoon on high rises that were right near the site, so that's something to think about.

And the next slide, he comments that some metals, and lists a series occurring at unprecedented levels in these very fine aerosols, and then goes on to note that levels dropped off dramatically, even over the course of the month of October and definitely by the end of May.

There are other slides listing a variety of metals that have been found both in dust, but the concern that dust is present after the fact may not be representative of what people actually breathed in at the time. I'm told indicating that lead levels do not appear to be a huge concern.

Skipping to the next slide, Lioy's comment. The concern that deposited material with metals in it could lead to ongoing exposure -- because in contrast to VOCs, metals are very persistent in the environment. Lioy commented that concentrations of arsenic and cadmium were relatively low but still in the parts per million range, so we need to keep that in mind when thinking about dust.

Next slide, a little bit of data, some of the small amounts that I found regarding airborne levels other than in the plume.

And then finally metal implications. So the metals data are hard for me to synthesize in terms of thinking about risk to individual workers. There's been a lot of characterization of the plume, and I'm not up to speed on all of it at this point,

but the thoughts that I have in terms of the metals at this point are the potential risk for toddlers who spend a lot of time on the floor and do a lot of hand to mouth activities from persistent metals in dust in residential areas. And then my other concern is the impact that these metals in dust, these very small particles, being deposited in the lungs, and I'm wondering, you know, some of these metals do bioaccumulate. We, you know, lead and cadmium clearly reside in the body and accumulate but I'm wondering if that very high initial load could change the half-life of some of these metals in the body, and I'm also wondering about the potential for interaction with the very high pH, although I don't know that if some materials that I read commenting that the smaller particle size had a more neutral pH, so I don't know how significant that concern is. But I did want to mention that.

So that's all I have.

DR. WARD: Thank you. Where do we stand on time, Paul?

DR. MIDDENDORF: We need to get started.

DR. WARD: Okay. We're going to start public comments now and then we'll get back to Virginia with any questions.

PUBLIC COMMENTS

DR. MIDDENDORF: Okay, each of our public commenters has signed up on a first-come-first-serve basis, and each of them will have up to five minutes to present. I remind people that it's often surprising how quickly five minutes can go when they talk about a subject of great importance to you so when you reach four minutes, I'll let the commenter know that they have one minute remaining, so they can be sure to make the points that they want to make in that last minute they have.

If they get up to five minutes, I'll have to rudely interrupt them and thank them for their comments. I apologize up front to anyone to whom that happens but we have to be fair to all of our commenters.

We do have one commenter this afternoon who will be on the phone, and just remind them to keep the phone on mute until I call out their name, and then they can unmute the phone and they'll have the same five minutes everyone else does.

Also want to point out that everyone has the option of submitting written comments to the docket for this committee. The docket number is 248, and information on how to submit comments is in the Federal Register Notice; it's also in the NIOSH docket page, and it should be on our committee web page as well.

Lastly, I want to remind our commenters of the redaction policy for public comments. The policy is stated in the Federal Register Notice for this meeting; it's also on the committee's web page and it's posted at the registration table if anybody wants to look at it. And the policy outlines what information will be kept and what information will be redacted before it's posted to the docket.

So when I call your name if you would kindly come up to the podium. We need to get the microphone up there, wherever it is, handheld mic? Our first speaker is Micki Siegel de Hernandez.

MICKI SIEGEL DE HERNANDEZ: Good afternoon. My name is Micki Siegel de Hernandez. I'm the Health and Safety Director for the Communications Workers of America in District 1. Our union represents several different groups of 9/11 responders as well as area workers affected by 9/11 exposures. I'm one of the designated labor reps on the World Trade Center Health Program Responder Steering Committee and a member of the World Trade Center Health Program Survivor Steering Committee and was the sole labor liaison for the EPA World Trade Center Expert Technical Review Panel.

First, regarding adding cancer to the list of World Trade Center-covered conditions, our union supports that. The time is now and I believe that today's presentations, thankfully, provide ample support and rationale.

Secondly, regarding the research agenda topics, it was good to see such a breadth of topics suggested by the STAC. We support research on cancer, heart disease and other chronic conditions, mechanisms of inflammation and disease persistence which could hopefully lead to more effective treatments, immunological disorders including autoimmune conditions and nervous system disorders.

We would also like community-based participatory research projects involving affected responders, area workers and residents to be encouraged.

While funded research is important, it can't be the sole source of our understanding of World Trade Center-related disease, and I cannot emphasize enough the need for improved and continuous disease surveill -- disease and symptom surveillance in the World Trade Center Health Program. This deserves a closer look.

A couple of examples are headaches, loss of peripheral vision, symptoms which are nonspecific and can have many causes but are frequently described by responders. While aerodigestive disorders may be the most common World Trade Center-related conditions, they are not the only ones. However, if you are not looking for other illnesses, you will never find them.

And then I have some sort of random comments that were taken from the presentations today regarding exposures. First, in several presentations it was mentioned that there were no samples that were taken during that critical first week after the World Trade Center collapse. I think that needs to be revised to say that no measurements were reported rather than none taken.

In a joint statement of the EPA and OSHA on 9/14, they stated that sampling data for asbestos were below levels of concern, not likely to cause long-term health

effects. Christie Whitman's famous statement on 9/17, declared the air and water safe based on initial sampling. EPA pulled early sampling data from their website, the New York City Department in Environmental Protection hazmat team was onsite that first day, took samples that were never reported.

So this is indicative of a stance taken by government agencies that they have stuck to this day, and in part explains the disconnect between reported sampling, or non-reports, and actual health effects.

It also, as was discussed in several of the presentations today, it matters what you sample for, when you sample, where you sample, how you sample and how samples are analyzed.

This also explains in part the inconsistency with levels being reported as safe and the health effects. Sampling was not conducted in a consistent or even comparable way. It was done by several different agencies, much of the sampling was done by private entities and therefore not in the public record.

I would also argue that a wrong model was used. Individual contaminants were measured when the World Trade Center dust and fire, the plume from the fire, is a very complex mixture. There were different standards that were applied that were not health-based standards, and these were used to make statements about health; such as the OSHA standards. The PELs are not health standards and they are also based on 1960s science and knowledge.

Ambient air exposures are also but one part of an individual's exposure. In some of the articles, there was an article that was distributed about, the Lioy article, about environmental conditions and human exposures at a current post-September 11th, 2001, in 2006, --

DR. MIDDENDORF: You have one minute left.

MICKI SIEGEL DE HERNANDEZ: One minute? And in that it said that the second rain event washed much but not all of the remaining outside settled dust and smoke away; this is simply not true.

Lastly, the duration of exposures were short-term for many people. This was repeated in a couple of presentations, the committee should be careful about how it defines or thinks about short-term exposure, what is known and not known about exposures.

Is it short-term for responders working up to eight months at Ground Zero for 10- to 16-hour or more shifts? Is it short-term for responders who continued response and restoration activities in contaminated areas well after the site was closed? And you should also know that there is no known end date for any given individual or for areas since levels of contamination and exposures, particularly in indoor sites, were not assessed. Thank you.

DR. MIDDENDORF: Our next speaker is Bruce Edwards.

BRUCE EDWARDS: Thank you for giving us the opportunity to speak at this meeting. My name is Bruce Edwards. I am a permanently disabled IBEW Local 3 journeyman electrician. I was asked to work at the Verizon building at 140 West Street. The building is across Vesey Street from where the North Tower and Building 5 stood. 140 West Street was severely damaged by falling debris of the towers on its south side and the collapse of Building 7 to its east.

I arrived at Ground Zero early in the morning of September 14th. Our arrival at the site was delayed due to fear of instability at the site, and we were originally scheduled to arrive the previous day.

I was employed by an electrical contractor that was known as a Telco contractor, very knowledgeable in the operations of telephone central offices. We were tasked with the temporary restoration of electrical power by means of portable generators. The reason this work was so important was due to the antiquated underground cabling methods of downtown Manhattan. The Verizon building at 140 West Street was the main path of communications in and out of the Wall Street business district, and most importantly, the New York Stock Exchange. The president at the time, George Bush, had ordered Verizon to restore communications as soon as possible. Due to our efforts, the Stock Exchange was up and running on Monday September 17th, before the opening bell.

We continued working at 140 West to permanize (sic) the temporary work to safety and then actually repair the building. It was many weeks before Con Ed could get power to the area at Seven World Trade Center, was the substation, the power substation, of the area. Our portable generators were needed to operate the building.

In the first few weeks, we worked 16 to 18 hours per day, seven days a week. And then as our numbers increased, we went to two shifts, 24 hours a day. As a supervisor, my responsibility extended to both shifts.

I'm sorry about all the background but I believe that is important to understand that the reason that I was asked to work there, and believe me, you didn't have to ask me twice. I felt a bond to the World Trade Center, as my father and brother had both worked on the construction, and we had been attacked. Nationalism and patriotism was at an all time high.

Ultimately though, I was a civilian required -- requested to work in a disaster area with little protection and no knowledge of the long-term problems that could occur. My original crew on the first day consisted of myself and seven other electricians, basically an advanced team to lay the groundwork. Within a few days, we had well over a hundred electricians on site.

Now, if you ask me would I do it again, my first instinct is yes. Like many, I took this personally. But in further review, I'm afraid I might not do this because the price I paid was steep. In April 2007, I was diagnosed with stage IV, non-Hodgkin's lymphoma.

I spent nearly two years in and out of hospitals for chemotherapy treatments, and fortunately I was able to have a stem cell transplant in December 2008. I'm currently in remission but remission isn't a cure. I live with the constant thought that the next low-grade fever I get is a return of my disease.

But even then I consider myself lucky because of the original eight, **[identifying information redacted]** (ph) didn't fair as well. He succumbed to his disease in 2010 at the age of 50. I was 50 when I was diagnosed also. Now I'm no scientist but I do see of our original crew two cancers out of eight. That's a 25-percent disease rate in relatively young men.

I was forced to retire from my career at least ten years early. The financial hit was crippling. I had two children in college and practically no money flowing in.

The next problem was clinical depression from all the problems there. Fortunately, with some good doctors, I was able to clear that.

DR. MIDDENDORF: One minute, please.

BRUCE EDWARDS: In the time since 9/11, some troubling items have emerged. Our government seems to have downplayed, and I use the term graciously, some of the conditions at Ground Zero. **[identifying information redacted]** the air is safe declaration and the release of some information about the accident exposure. The report released around the tenth anniversary showed dioxin levels 1,000 times higher than normal, and the highest the EPA has seen. What is especially troubling is the sampling began on September 23rd. That's almost two weeks after the attack.

The next two months the sampling continued and showed steady decline, so I can only imagine what the levels were on day one, or day four for my crew.

The report from the fire department is also an eye-opener. Here's a segment of the population that is generally in good physical condition and well-monitored, and yet the cancer levels for those exposed at Ground Zero is well above normal.

What I have come to learn is that --

DR. MIDDENDORF: Your time is up --

BRUCE EDWARDS: Okay. Well.

UNIDENTIFIED SPEAKER: Let him speak.

BRUCE EDWARDS: I'd just like to let people know here that the cancer rates are very high for a young population where normally they would be in an older group. And I implore you to add cancer to the bill as the Senate, I should say the Congress,

has done with this letter that they sent to you. Thank you.

DR. MIDDENDORF: Our next commenter is on the phone. Rich Dambakly. If you would unmute and begin your presentation.

RICH DAMBAKLY: Hello?

DR. MIDDENDORF: We can hear you.

RICH DAMBAKLY: Okay. My name is Richard Dambakly. I'm an underground worker for Verizon, at least I was an underground worker for Verizon. I worked at Ground Zero from the moment of the disaster, every day for six months straight, 12 to 16 hours a day, no days off.

I developed the World Trade Center cough. And for those of you that are unaware what this feels like, it's a cough where your chest is exploding out of your body that doesn't stop.

In March of 2002, it had gotten so bad I had to go to emergency. After being diagnosed with lymphoma cancer, I started intense chemotherapy treatment that lasted five months.

Just recently someone mentioned to me that the actor Andy Whitfield from the television show Spartacus had died from lymphoma, and it was his second occurrence. And here I am with no CAT scan for three years because I have -- I can't afford one. I have no medical insurance. How do you think that makes me feel?

I'm a father of five children, my oldest being 15. My family needs me. I want to be around to walk my daughters down the aisle and play ball with my son. Should I become a beggar and maybe raise the money for a CAT scan? Just like our Vietnam vets, that they were forgotten?

So many have died already from cancer. Their families need help now. This can't go on. When other countries are in need, we don't waste a minute. Immediately we send them money. We ask for nothing in return. When President Bush arrived at Ground Zero, I stood and listened to him speak to us and tell us to stay strong, stay here, help us, do whatever it takes, whatever you have to do, work any amount of hours. We need you; we'll be there for you. And we did it, each and every one of us that stayed strong. Anything we could do in our power. No one said, I can't help or that's not in my job description. No, we did whatever we were asked and more. The country needed us and that's all that mattered.

So now that we need the help and when you should be strong for us, instead you're taking the position that covering us for cancer is not in your job description, and that's wrong.

On 9/11 terrorists came to our country and were responsible for thousands of deaths. Don't give them more reason to celebrate by not responding to our

country's aid and causing more American lives. Don't allow them more victory than they already have.

We were there when our country needed us, and our country should be there for us when we need them. God bless all my fellows and other survivors and first workers in the World Trade. God bless you all. Thank you very much.

DR. MIDDENDORF: Thank you, Mr. Dambakly.

Our next commenter is Alex Sanchez.

ALEX SANCHEZ: Good afternoon to members of the committee; my name is Alex Sanchez. This good? I am a 9/11 responder, clean-up worker. On September 11th I had a very close encounter with terror. I was standing not very far from where this building is today.

On September 13th to March 15, I performed cleanup with other cleanup workers in the skyscrapers surrounding the pit. Ten buildings in a period of six months. Twelve-hour days, seven days a week. Some of the buildings I worked in included 1, 2, 3 World Financial Center. I had a ringside seat to what police officers, firefighters were doing at Ground Zero. When I went past those barricades, as a citizen, as a New Yorker, I knew what was expected of me.

When men and women started getting sick and dying, I also knew what was expected of me. Since late 2003, early 2004, I've been walking the halls of Congress alongside many of the men and women who are in this committee and who are also here today. **[identifying information redacted]**, my mentor, president of the FealGood Foundation, an officer and a gentleman, paratrooper, United States Army. We do not leave ours behind. What message are we sending to future generations and to the international community when we overlook and not appreciate the work and the efforts of those who served at Ground Zero?

Let me give you some facts. Basically you should know these by now.

Seventy percent of the men and women who came to Ground Zero are suffering from lung disease, chronic gastric disease, post traumatic stress disorder. I'll give you another example.

[identifying information redacted]. Both on the same office, Senator Lieberman, two months later, I asked my assistant director, **[identifying information redacted]** (ph), who is this gentleman **[identifying information redacted]**disintegrated in a period of two months.

We don't need bigger government or smaller government. What we need is responsible government, government that takes care of the people. Enforce and enact laws, current laws. I am a single father of an amazing 10-year-old. This is not the message I want to send to my son, my country cannot get it right. Ten years down the road cancers are killing the men and women who came to Ground Zero.

Exposure science tells us that when you are exposed to high level of toxicity, you need 15 to 25 years of medical treatment. We only got five. We cannot continue to play games with human lives. We need to stand up. We need to serve those who serve our country. We shall never forget and may God bless the United States of America. Thank you.

DR. MIDDENDORF: Thank you, Mr. Sanchez. Our next commenter is John Feal.

JOHN FEAL: How's everybody doing today? Good? I don't think I need a microphone. I'll introduce myself when I'm done. This way I can get my five minutes in.

One, I want to thank NIOSH for doing this. I want to thank the STAC committee for hearing me today.

I'm not here to ask you to add cancer to the bill. I'm here to ask you add certain cancers to the bill. I'm getting a little tired of hearing we need to add cancer to the bill. You cannot add every cancer to this bill; that's impossible. I get it. I worked on this bill for eight years, more than most people in this room. But there are cancers, unequivocally, undoubtedly, that need to be added to this bill yesterday.

I am never the smartest man in the room and I'm not even the smartest man at this podium probably, but it doesn't take a scientist or a doctor to know that 9/11 and its toxins have caused these blood cancers.

For years when we walked the halls of Congress, we were applauded for the way we approached Congress to get this bill passed. And when we were lobbying to get that bill passed, we were lobbying to get cancer added to that bill. But during the negotiations, that was taken from us. But I am going to use the same zest and the same energy to help get those certain cancers added to this bill. I will occupy Ground Zero. Don't worry about Occupy Wall Street. I will do whatever it takes because at the end of the day, I care about human life. I don't care about what you're having for dinner, I don't want to go to your house for coffee. I care about human life. I care about adding cancer, certain cancers, to this bill.

And as for epidemiology, let that not be your only role model. Epidemiology can only do so much, like the cancers that we know that should be added, use epidemiology on that. 9/11's unprecedented. It never happened before. So use something else other than an epidemiology. And believe me, I can't even spell the word, that's how smart I am not. Okay? So I'm asking you guys, with power comes responsibility. You have a responsibility today, tomorrow and from this day forward to do what is morally right.

I just came from a press conference at City Hall, and I almost threw up on myself listening to people who do not know what they're talking about. But appreciate the magnitude of this 'cause I do. I lost half a foot ten years ago. Eleven weeks in

the hospital. I'm lucky but I feel guilty that I can go to Sheelar (ph) and say I want to apply for the Zadroga bill 'cause I lost half my foot. Boohoo. Say that to **[identifying information redacted]**, who have leukemia and blood cancers. That should be added yesterday. You're playing God right now. Our fate is in your hands.

I am the nicest guy in the world. I want to be your friends. But like I told every member of Congress and every member of the Senate when I met them for eight years with this bill, I will do whatever it takes to get cancer added to this bill. Thank you.

DR. MIDDENDORF: The document which you handed out to the committee members will be added part of the docket. Just wanted to let you know that but it may be redacted to some extent. We'll have to look further.

JOHN FEAL: Do what you please with it.

DR. MIDDENDORF: Okay, our next commenter is T.J. Gilmartin.

T.J. GILMARTIN: Good afternoon. My name is T.J. Gilmartin, and I'm 32 years as a foreman and a shop steward building high rises in New York City with the union. Now, I had to go to so many OSHA classes for these high rises of stuff they taught us was cancerous and, you know, don't do this, don't do that. Everything, everything I been taught to and told is dangerous and cancer-causing is being thrown out the window on this World Trade Center. I mean, I know what goes into building a high rise and one thing that was -- and the Trade Center was built prior to 1973, when the asbestos was in the pipes, it was in the cement, it was the silicosis, the heavy metals, the chemicals and the PCBs.

Does anybody know about those electrical vaults in the basements of those trade centers? You know that's totally cancer-causing chemicals inside those -- the vaults and the transformers? Okay? All that was there and we never hear of anything. Anything about any of that.

I mean, all this stuff is concern -- is confirmed as a federal cancer-causing chemicals. The building was totally filled with all these chemicals. The fire department, the PDA have done studies showing that their men are dying a lot more than they are usually dying fighting fires.

I mean, OSHA would lock me up if I was -- if I was grinding concrete on a high rise and that powder, if I didn't have a battery-operated respirator, I'd be locked up by OSHA, either thrown in jail or fined for having my men do that. I mean, you had 220 stories of pulverized concrete besides everything else that, God forbid, was going to happen in another nine years with the asbestos, with that 20-year lag time.

It's been over ten years since the World Trade Center was destroyed, and that's

been a time so many first responders have paid with their lives. The percentage is out of whack compared to how many first responders just tried to help their fellow man. It seems to me that this is all about the money. I mean, I understand that you'll have everybody claiming that they got cancer from World Trade Center but like John said, there were certain cancers from the ears, nose, -- I mean, your mouth, your nose or absorption that should be covered by this.

But it's -- you know, I mean, that's basically what I have to say. I mean, just that I been in the business of high rises and I know what causes cancer on these things and, you know, you put up a high rise, OSHA's there, you're doing it, you know, you're in a lot of trouble if you do it that way. Everything that could get you cancer on a new high rise was all down at the Trade Center, and it was a lot worse because it was built before 1973 when the world was changed. Thank you.

DR. MIDDENDORF: Thank you very much, Mr. Gilmartin.

Our next commenter is Thomas Fay.

THOMAS FAY: Good afternoon, ladies and gentlemen. Is this the speaker here? My name is Thomas Fay, and I come from a town at the Jersey shore called Spring Lake, New Jersey. On September 11th I was getting my wisdom teeth pulled; and the planes hit the building and I raced home and proceeded to watch on television for about 36 hours. And after the 36 hours, I couldn't take it anymore so being a volunteer fireman for over 37 years in the Spring Lake fire company in Spring Lake, New Jersey, I decided to go get my gear, jump in my car and race to New York. I got there in 50 minutes, which is unprecedented.

I was directed down to the south end of the city and parked my car on 14th Street and I walked in. Two other firemen drove by this desolated area of lower Manhattan and picked me up. I never knew them before but I know them now. Both are very sick.

They drove me down and they went out to get a camera that day to take pictures. I didn't want any pictures taken of me that day; I was there to work, not to have any pictures taken. But lo and behold, they took two pictures of me and those two pictures ended up being the proof that I needed to show that I was there.

The disease that I contracted from my 12 hours working on the south tower pile, solely on September 13th, was non-Hodgkin's lymphoma, stage II, B-cell aggressive. The way that was found in me was that I, in 2007, after the disaster, a friend advised me that I needed to go get checked out at the World Trade Center medical monitoring treatment program they had at Rutgers, which I did.

I went in 2007, 2008, and in 2009, I noticed a lump in my left leg. I showed it to **[identifying information redacted]** out there. She said you've got to go to New York City, Mt. Sinai immediately. Within a week the tumor was taken out. Four days

later I was told that I have cancer.

I fought the battle brave and hard. I'm in remission now which is a good thing, but for people like us that went up there and put our time in, I being a volunteer, I was paid nothing, I would go again tomorrow because of one thing: I love my country. That's it, pure and simple.

Being a guy from the Jersey shore, a popular person everyone knows who comes from down there is Bruce Springsteen. He has a new album out. And he has a song on it called, *We Take Care of Our Own*. That's the theme song for us first responders. We want our government to take care of us.

We went in there. We fought hard. I worked 12 hours on that burning pile. If I fell once, I would have been cut to shreds. But that wasn't on my mind that day. On my mind that day was to help as many people as I could. That's why I joined the fire department, to help people. I didn't join the fire department to get cancer.

My cancer's in remission but as of Monday, a recent trip to the doctor, has shown that I now have skin cancer. I'll fight that battle on my own and take care of that as I should. But it is my hope that this -- people here, grouped here today, do the right thing, which is to include blood cancers in the Zadroga bill. Thank you very much for your time.

DR. MIDDENDORF: Thank you very much, Mr. Fay. Our next commenter is Arthur Noonan.

ARTHUR NOONAN: Hello. My name is Arthur Noonan, retired now but back in September 17th, 2011, I was employed by the Chicago Fire Department. As the last speaker, we were watching on television nonstop at the firehouse. Finally we couldn't take it anymore, we saw what a devastating effect this had on the country as well as to New York, and we decided to come here. I believe there was a group of 14 of us. We flew in and we spent seven days working here.

I was a pretty healthy guy as well as the rest of the people that came with me. A lot of young firemen from Chicago, good firemen, and we did everything from cleaning tools and changing blades and batteries in the tool shed, until we finally got to work on the actual Pile. Some days we would cut aluminum off of steel beams so the iron workers could cut the beams in sizes small enough to fit on the trucks to haul them away.

Eventually we got to work on the Pile. You'd start at the back of the Pile, there might be a hundred firemen in front of you. You'd pass buckets forward empty, and backwards full. Finally you'd get up to the point where you were the one that was digging. You'd be on your hands and knees; what respirators we had didn't work, they kept clogging up or from the sweat would just turn like a mud on there. We finally had to take those off. But you kept working because you knew your

brother firefighters, policemen and many loved ones of civilians who were also in that Pile. And all we wanted to do was try to close a part of life for a lot of people.

In December 2004, I became ill at work, was taken to the hospital. Thought I had a bad touch of the flu; everyone was sick in the firehouse then. It was the day before Christmas Eve. They let me go home for Christmas Eve and Christmas Day, I had to come back the following week, and I was diagnosed with AML, acute myelogenous leukemia.

I went from 210 pounds to about 140 pounds in six months, had several chemo treatments, and luckily I am now in remission. But remission is not getting better. It just means they're holding you steady so every day you hear something on the radio, whether it be a celebrity or sports figure, just recently we had a famous singer die of leukemia. Every time you hear that word leukemia, it all comes back to you.

When we came to New York, we did it on our own. We did not expect to get anything for it. We just wanted to help our country. We wanted to show the world the support that New York and the United States, how they all come together in a time of need.

Personally I have taken a tremendous loss on my medical benefits. I've gone through about three-quarters of what I'm entitled to in my lifetime for myself and my wife and if this comes back, I probably only have a few hundred thousand dollars left in my medical plan from the City for treatment. After that, I don't know what I'll do.

So I'm hoping that cancers, certain cancers, will be included in this so people that came to help do not have to have that constant worry in their mind if their cancer comes back, they won't be able to get any treatment. Thank you.

DR. MIDDENDORF: Thank you very much. John Walcott.

JOHN WALCOTT: Hi. My name is retired detective John Walcott. Like everyone else here, I'd like to thank you for this opportunity.

I also was diagnosed at 38 with AML leukemia. As I stand here in front of you I've had six months of chemotherapy, stem cell transplant, and I have other illnesses that are recognized in the Zadroga Act. But looks are deceiving. All my nerve endings are burnt out all my -- in my hands and my feet. There's not a day that goes by I'm not in constant pain.

The City retired me due to my leukemia, which they said I got from 9/11. Social Security recognized it. It seems that only the country doesn't recognize it.

Before 9/11, I was approximately 36 years old. I was never sick a day in my life except for the common cold. I was a very extremely active narcotics detective, well over 3500 arrests in my career involved in. I was a high school hockey coach. Used

to do physical activity, lift, run every day. No longer can do any of that. I was on the fast track to probably becoming a hockey coach in college. We had an exceptional team, exceptional record and I turned down many jobs which I planned to take when I retired. Which, that's been cut short.

On 9/11 itself I wasn't scheduled to work 'til late that evening. I was told what happened, I was woken up, and I was down there in 93. So without hesitation, I ran right down there to help my fellow detectives or policemen at the time. Shortly after the second tower had collapsed, I arrived.

Did -- from recovering bodies, body parts, to Mayor Giuliani even assigned us one day to VIP tours for all his friends. So I've done everything, cut steel. You weren't a policeman when you were down there; you were just somebody trying to help. As I told you before I had the transplant and everything else.

Well, you know, let's talk a little bit why we're down here. We all know that the benzene and asbestos and all over cancer carcinogens were down there. That's no secret. I mean, that's been for a hundred years. We don't know what they do if you mix them all together nor do I think anybody really cares because if they did, it wouldn't have taken us ten years to get to this point.

We know there's a usually high number of early responders that are diagnosed with cancer. Yet no one seems particularly interested in trying to corroborate any of these findings at the site, at the cancer rate. The large population of responders and workers are being looked at, which I think you guys are doing a study of over 50,000 people. But I think that study's wrong. I think you should study guys and girls and everybody who was down there the first day, first week, first month. And if we do that, you're going to see that the 362 PBA Study, that rate is going to be astronomical. It's probably going to be in your 60s to 70 percent of cancer rate. There's many reasons. We all know there's many reasons why the City's and the country's not releasing these numbers. Because they're doing you a 50,000 population rather than a 2500 to 5,000 population. So that statistics are going to be extremely less and it's not going to prove cancer. But if you did, if there was actually 2500 to 4,000 that were down there the first week, day or month, it's going to be astronomical. And then the red flag is going to be up.

But when there's litigation going on and there's hearings about to happen, what do we do? We have to make the numbers look bad because the City kind of painted themself in a corner right now with this.

DR. MIDDENDORF: One minute left, please.

JOHN WALCOTT: Okay. You know, I think that's where we need to concentrate. We have to concentrate on -- let's concentrate on 2500 to the 3,000 that were down there versus that. I don't -- there's a part of me that envies you folks and

there's a part of me that doesn't envy you folks. You have to make a tough decision. But luckily for you folks you have ten years and weeks of hearings to make this decision.

I had a phone call and I had to rush down. Now I'm sick, my daughter'll never see me walk her down the aisle. I can put my head on my pillow and go to sleep at night knowing I did something that in the recovery that meant closure for people. You folks have that same power now. Twenty years from now if the cancer isn't added, and my grandchildren, that I'll never see or hear, do you say you made the right mistake? Did you make the right decision? Thank you.

DR. MIDDENDORF: The next commenter is Reginald Hilaire.

REGINALD HILAIRE: Hi. Good afternoon. I'm a police officer with the NYPD for 11 years. I was a rookie when 9/11 happened. I'm currently assigned to PSA 5, which is a housing precinct up in East Harlem. I worked over 850 hours combined at the World Trade Center and Sandman Landfill.

In 2005, shortly after my son was born I was diagnosed with thyroid cancer. I immediately asked my primary care physician if this was related. He said, he looks at my lump and said, what were you exposed to down there? I've seen him since 1999, before I became a cop. So 2005, I had total thyroidectomy, radiation and ever since then I take a pill, a synthroid, and it regulates my thyroid.

Winter of 2005, I go back to my primary care physician, he noticed my blood count was pretty low. He refers me to a hematologist and that hematologist does a bone marrow biopsy, and he comes back and he says, the pathology report -- I disagree with the pathology because it says you have multiple myeloma but I disagree. You're too young to have this. He repeats it in 2006, it comes back multiple myeloma. He's still confused.

I go -- I sent everything to Sloan-Kettering. They do another biopsy, bone marrow biopsy, April 2006. They confirmed it. I thought okay, great, treat it. No, we can't treat you because you have smoldering multiple myeloma, early stages. So I'm like, is there anything out there for me? No, you can't -- there's nothing. We have to wait until it gets worse in order to treat you. He says within two to three years, you have 50, 60-percent chance of it getting worse.

Thankfully every four months now I go to Sloan-Kettering, they do blood work, urine work, and if I get the phone call, that means it's not good. So far, knock on wood, everything's okay.

I have no family history of cancer. I'm pretty much the healthiest one. I am a son of Haitian immigrants. I am the only member of my family that's a police officer. I was born and raised here, still work here in Harlem. I can't retire because, even though I'm not really sure if I want to, but I can't retire because I'm not sick enough

so it's an oxymoron right there.

I have two red cancers. I don't -- I work with a lot of cops in PSA 5. I don't know why I have it. It's just one of those things I've come to accept it. In 2006 I read an article in the Post saying that there's other first responders with cancer. I contacted that reporter who introduced me to one detective who has lymphoma. He introduces me to others. I got to know about 11, and I'm pretty close to about four of them. Three of them have multiple myeloma. I never met them before in my life.

I met one police officer through the PDA who (unintelligible) I did. His name was **[identifying information redacted]** (ph); he had (unintelligible) cancer. We got to talk for about a year and then he eventually died in 2010. So I always think about him, think about his family, I'm still close to his widow.

I don't -- I'm not a scientist; I'm just a cop, I just want to do my job. I think a lot of us want to do our jobs. I don't think it's coincidence. I never met these people before in my life.

Someone asked me before if they had to do it again. I, like I said, I'm still with the NYPD. I'm doing clerical work. I'm pretty now senior now. If it happens, again, and I'm pretty sure it would, would I do it again? Would I tell my junior cops to go? I don't know. I love New York City, I love the people here. I'm not fond of the government. They showed so careless without a doubt.

What's really insulting, I could deal with cancer, I could deal with questions, how you doing. As a New Yorker, how you doing could mean ten different things. How you doing or in my case, so how are you doing?

DR. MIDDENDORF: One minute, please.

REGINALD HILAIRE: What I can't stand is politicians, everybody can say, okay, great, great job; you're heroes but when it comes to treating us, hold back. It's just too early to step up the study; it's not there yet.

I try to tell the cops in my precinct get yourself checked out. They look at me. We can handle perps, we can handle perps with guns, we can even handle bosses that are rough. We can't handle our own mortality.

So I urge all of you, just like us, when they call us heroes, all of you can be heroes by just saying, adding cancer. You will save lives by putting cancer in the bill because it will tell first responders to get checked out. You don't know how much of a difference you guys will make if you add cancers. You will tell somebody with the public -- when the report comes out, that one person would say maybe I will get checked out. That can make a difference. Thank you very much.

DR. MIDDENDORF: Thank you very much.

Next presenter is R.J. Lee.

R.J. LEE: I do want to thank the committee for giving people the opportunity to testify. I've been asked on behalf of the Policemen's Benevolence Association to speak on their behalf about the composition of the World Trade Center dust and some analysis we recently did on the uniform of one Officer Harris.

By way of background, R.J. Lee group worked in New York City for about four years following the disaster, characterizing, analyzing and characterizing samples of World Trade Center dust and exposures and things like that.

Today I want to talk about Officer Harris. Laboratory testing of Officer Harris's clothing worn on the morning of September 11th, clearly demonstrates the presence of what's now referred to as World Trade Center dust. And you can see the uniform on the first slide that he was wearing that day.

Fortunately, almost by, I don't know what fate, Officer Harris had the presence of mind to go home that morning and double bag his clothes so we have a virgin sample of World Trade Center dust. One that hadn't sat out in the rain, whatever, for months, and one that you could look at as it was created.

As you can see from what's called the World Trade Center well, the World Trade Center dust is a unique mixture of heavy metals, asbestos, fine cement dust and chemicals produced by burning, including PCBs, dioxins and furans. The chemical species found in WTC, chemical and physical species, found in World Trade Center dust can cause many harmful effects on the body including effects on the nervous system, kidneys and cancer.

It's, as you've heard it's widely believed that there's been an insufficient amount of time to assess the potential for increased cancer risk. However, I believe there's certainly reason to assume that the acute exposure experienced by first responders are significant and unique.

There are a number of factors to be considered that could play a role in increased cancer risk to individuals and the potential for more rapid progression than you would expect.

First of all, the initial dose, acute exposure was enormous.

Next slide? This is the dust we found on Officer Harris's clothing. You'll note that in something like two or three hours, about 59,000 structures per centimeter squared had been deposited on his clothes. Chromium was at 347 micrograms per foot square. That's a lot in a two or three-hour exposure. If you put that cast an imaginary membrane through the breathing zone, you can translate that kind of deposition rate into exposures and they're large.

There's an abundance of respirable particles in the dust, far more than ordinary. What's interesting, and one of the prior speakers mentioned it, in the analysis we did of these hundred thousand samples, and including Officer Harris, many of them

were coated. The asbestos was coated with lead; the asbestos was coated with mercury. The machines don't analyze for dioxins in the electron microscope but obviously dioxins and PCBs were there.

DR. MIDDENDORF: One minute, please.

R.J. LEE: The presence of dust on Officer Harris's uniform clearly demonstrates that the first responders were exposed to extreme conditions. There was reason to believe that you could postulate a model in which the dust carried, the caustic cement dust, carried toxins and those toxins and that interaction of the pH 11 or 12 cement dust could well interact with the lungs and deliver toxins much more rapidly than believed possible.

I think it's important on behalf of the PBA to say that given the service of the first responders that we've heard about today and the trauma they're going through, that any potential disease that could be covered should be covered on their behalf. And secondly the information they're seeking from the City and the government should be released anonymously so that it can be used scientifically. With that I thank you.

DR. MIDDENDORF: Our last commenter is Philip Landrigan.

PHILIP LANDRIGAN: Good afternoon, Madam Chairman. I'm Philip Landrigan, I'm a physician and occupational doctor. Chairman of the Department of Preventive Medicine, Dean for Global Health at Mt. Sinai School of Medicine. For six years I directed the Division of Surveillance Hazard Evaluations and Field Study at NIOSH, so in other words for those six years, 1979 to 1985, I directed the National Occupational Epidemiology Program for the United States of America. So we, we know for a certainty from multiple lines of evidence, that you've heard a great deal of data here today, and I thought that testimony presented just now about the contaminated police uniform was striking. We know that the responders to 9/11 were exposed to a complex mix of known and suspect human carcinogens. We know that the air sampling data that were collected undercount the true level of contamination. I think the testimony just heard substantiates that, but it stands to logic anyway that there were no sampling units extant in the first hours and days after the attack when the concentrations were highest, so we know that the responders were, especially those who were caught in the dust cloud, were exposed to unprecedentedly high levels of airborne contaminants.

Now, our group at the Mt. Sinai School of Medicine, in partnership with people at UMBNJ, Stony Brook, Queens College, North Shore LOIJ and Bellevue have just completed an epidemiologic analysis based on approximately 20,000 responders, and we looked specifically at cancer in them. This is an analysis that follows on our earlier studies showing persistence of lung disease and mental health problems and

GERD in the responders.

I'm not going to present great detail because it's going to be submitted for publication in the next couple or three days, but I am going to give you a broad sketch of the findings.

Overall we found approximately a 14-percent excess in cancer at all sites combined in this population, and we found statistically significant excesses of thyroid, prostate and hematolymphatic, hematolymphopoietic cancers, in this population.

In broad outline our findings parallel the findings that were released on September 10th of this year, that they would present from the fire department.

It's, I think, the 14-percent excess in overall cancer is striking given that in this population, we had a 58 prevalent -- 58-percent prevalence of never smokers, and we had sharp deficits for lung cancer and laryngeal cancer and yet despite those deficits in some of the most common cancers, we had an overall excess incidence of cancer in the population. These are striking findings.

Going back to your taxonomy this morning of the straw poll, I think we've reached a point where, to use Steve Markowitz's phrase, we can say with a high degree of certainty that the exposures that the responders experienced down there at Ground Zero, and at the other World Trade Center sites, can be said to -- we can reasonably anticipate that those exposures are going to cause cancer.

So I think, I think it puts you in a very difficult policies (sic), but you clearly don't have the kind of epidemiologic proof that you would like to have to declare with 95 percent certainty that there's a cause and effect relationship here. We're not going to be there for some time yet. But you have to bear in mind that in legal cases, you don't have to get to 95 percent; you have to get to 51 percent. It has to be more likely than not that the exposure caused the disease. And I think we're at, or very close to that point.

And what I'd like to ask you as members of this committee to weigh that as you make your decision. Thank you.

DR. MIDDENDORF: Thank you very much, Dr. Landrigan.

You have about 15 minutes left.

DR. DEMENT (via telephone): This is John Dement. I'm going to have to leave the meeting so I just want to make that note.

DR. MIDDENDORF: Okay. Thank you very much.

DR. WARD: So Virginia, are you still on the line?

DR. WEAVER (via telephone): Yes, I am.

DR. WARD: So I did want to give the committee an opportunity if they had any questions or comments on Virginia's presentation.

(no response)

DR. WARD: Okay, so --
DR. TALASKA: Oh, I have one question, if I may. I have one question.
DR. WEAVER: Okay.
DR. TALASKA: You mentioned a statement early on when you were talking about the VOCs, about that when the levels became, quote, extremely high, that people were removed from the area. And I just have to ask was the concern -- you know if the concern for that was because of explosion?
DR. WEAVER: I don't know.
DR. TALASKA: Didn't say it in the paper.
DR. WEAVER: I don't think so but I was reading seriously in the last week and I could have missed it, and perhaps others on the committee who spent more time with these data could weigh in.
DR. TALASKA: Thank you, though.
DR. MARKOWITZ: So I have another question for Virginia. So in your experience working with firefighters from previous studies, how common is it to find benzene at fires?
DR. WEAVER: It's extraordinarily common. We often use data that's now rather old but still very valid about the components, the VOCs in smoke; and in one study conducted by Harvard, benzene was present in about 92 percent of smoke samples obtained. And it's routinely found at levels well above the OSHA panel. Butadiene is also very common as a combustion product.
DR. HARRISON: This is not really a question for Virginia, just maybe an observation and a prelude to further discussion that we'll have. I guess I haven't heard anything from the presentations today that would lead me to understand that there was a minimum dose or duration of exposure that we could identify from the knowledge that we have to draw a line.
I think it gets, you know, back to maybe something that, Liz, you presented earlier about latency and duration of exposure. I guess I just would throw that out there just for an observation, that we really don't have, based on the limited amount of exposure data, you know, that we have from the site, the fact that it wasn't captured in the first several days, a way to define a minimum length or vocation related to the occurrence of cancer.
DR. WARD: So there is one question for Dr. Landrigan.
DR. MIDDENDORF: Yes, well, there was one question.
DR. WARD: Is he still there? Dr. Landrigan?
Okay, so would someone like to ask a question of Dr. Landrigan?
DR. TALASKA: Thanks for coming back, Phil.
DR. LANDRIGAN: No problem.

DR. TALASKA: I was wondering if you had done any analysis on the subset of people who were on the Pile early on relative to the whole group.

DR. LANDRIGAN: Yeah, we tried to do that. We certainly, in our previous paper that you've probably seen, the one that was published in September in Lancet, we saw a very clear gradients in most diseases according to intensity of exposure. The people who were caught in the cloud had the highest rates of pretty much every disease we looked at; the people who arrived in the first 48 hours but missed the cloud were the second highest, and then on down through several more gradations. We saw that for most types of lung disease, most mental health problems, for GERD. It was not so striking for cancer. And it may be because of smaller numbers of cases. Thank you. That's it? Yeah, thank you.

DISCUSSION ON PRESENTATIONS

DR. WARD: So, I guess we're close to the end of our day. And I guess one, it was suggested earlier that maybe we look separately at the question of biologic plausibility and the likelihood of cancer but I think one of the issues I'm struggling with, and I don't know if other members of the committee are struggling with it, too, is that we are -- whatever opinion we come to, we do have to define a scientific rationale, and I know that in a lot of the presentations this morning, you know, it would be more possible to build a scientific rationale around upper respiratory cancer, lung cancer, esophageal cancer, areas of the body where we know that there was direct contact with the carcinogenic substances and we know that there have been other kind of health effects, but I think the difficulties we, we don't -- I mean, I guess, and maybe Dr. Landrigan's study will help with that but with the hematologic cancers and the lymphomas, we don't as yet, I think, have strong epidemiologic evidence, and I'm not sure we have, you know, an exposure -- you know, we have a strong argument in terms of biologic plausibility, and I guess -- so the argument about -- I think we can say that, you know, it's in shorter -- it's observed that they have a shorter latency period but in terms of -- so I guess what I'm seeking is, are that -- do people have thoughts on that. How should we approach the question of the blood cancers given that that seems to be something that people are highly concerned about? Excuse me? Does anyone care to comment on that?

DR. WEAVER: So this is Virginia, and you know, blood cancers are the ones that based on latency alone, we could be seeing now from World Trade Center exposures. You know, ten years out, those would be the first wave of cancers that you would see. Those are also caused, or closely connected, with a number of the VOCs. And if you look at VOCs in combustion products, they ask -- there are a number. So you have an exposure mixture going on there. And so from that point

of view, I can see the biological plausibility and that being an initial concern.
DR. ROM: I think by definition, volatile means volatile, that these compounds probably were very high, right at the beginning with the burning of all the fuels, and they evaporated into the air and they weren't measured, and exposures were probably way higher than any of the standards so that it's biologically plausible that you're going to see non-Hodgkin's, Hodgkin's lymphomas and the acute leukemias, acute myelogenous or non-lymphatic leukemia and probably chronic myelogenous leukemia. I think the ALL and CLL are different biologies, and that may be something totally different 'cause ALL is in children and CLL is in the elderly associated with a lot of genetic mutation defects. But the others, and multiple myeloma, I would add, probably all are very biologically plausible at this time.
DR. MARKOWITZ: Also the firefighters study in fact was positive for non-Hodgkin's lymphoma. It showed a relative risk of 1.58 -- and actually whether you use the corrected one, which tries to take account of the surveillance issue or not, it showed a 50- to 60-percent increase when compared to the general population of men, and when they looked at it compared to the firefighters who hadn't been exposed, it was still elevated; it was 80- to 90-percent increase. Not statistically significant at that point because the numbers are smaller, but when it was compared to the general population it was elevated and that was statistically significant, so there was real epidemiologic evidence that blood cancer was increased.
DR. TALASKA: I think we might want to look more, too, at some of the other compounds that we haven't really spent any time with: the furans, the dioxins; what sort of impact they have, both animals and -- in animal studies for the most part, to see if there is a link between those -- or perhaps an interaction between those. And I don't think anyone has looked at those as hard as maybe we should.
DR. ALDRICH: (Indiscernible) the document that's not biological plausibility (indiscernible). Mesothelioma sometime in the distant future and probably lung cancer in a little bit less distant future, relative to the asbestos exposure. It's hard to quantify but certainly potentially a factor.
The fire department study did not show an increase in lung cancer; it actually showed a decrease in lung cancer possibly related to the health worker effect, but that was seven years of study, and that was probably too early to see the effects.
DR. WARD: So I guess I'm getting a sense. I know some people have not spoken very much today but the sense of the comments I'm getting is that many people on the committee feel that it is certainly biologically plausible that we would be seeing some cancers in excess, either now or in the future, and I guess the question is, is there someone who wants to state, you know, make a statement -- or are there

people who would like to speak to the question who have not spoken on it? Or we can go back to the, you know, the poll, but I guess I'm just trying to get a sense of the committee, of where we stand at this point. Time, again, so we can think about how we want to frame the discussion tomorrow in the maximal -- you know, in a productive way. Valerie?

MS. DABAS: Just from my observation, I understand that the latency period for blood cancers is short. I think we get into a very funny situation when we start piecemealing each part out. Both the fire study and Mt. Sinai seem to indicate that thyroid and prostate, they're seeing increases, and so if we start going by what is easiest and not looking at the whole picture, then I think we may start asking too -- well, I guess you can't ask too many questions but then it gets very confusing.

For me, I've seen, you know, from taking information from responders, I've seen an increase in thyroid, I've seen an increase in prostate. I was told that, you know, thyroid is common, prostate is common, but when we look at the ages people are being diagnosed, it's very uncommon for a 38-year-old man to even be tested for prostate cancer, so when they come up with prostate cancer, I think it's significant. I also have seen an increase -- you know, how do you deal, then, with the blood and liver canc -- kidney cancers that we're seeing? Liver cancers with people that are not hepatitis C and do not have cirrhosis of the liver. You know, we had four cases reported in that instance and, you know, so you have to really look at the whole picture as opposed to just saying well, the blood cancers are a four-to-six year latency period, we're at four to six years. If that's the case, that's just assuming that the dust is the same exposure as we've seen with all these other studies, and I don't think these studies take into effect the concentration of chemicals, metals and so forth, and we keep saying the dust is different than anything that we've seen before, and therefore I think we have to treat it different.

MR. CASSIDY: I just wanted to add that I think it's clear that we need to remember what was highlighted today, which is that this type of exposure to the variety of different things, the concrete, the dust, the metals, the benzene, all the chemicals, really hasn't been -- we haven't seen that anywhere before so when you want to start breaking down studies and say well, exposure to benzene means this. When you add them all together, you really have a toxic stew that, I think, is so biologically plausible to say that blood cancers and these other cancers are a result of that exposure, and I do think the severity of the exposure, you know, bears out clearly that, you know, those who were caught in the dust, in the cloud, in the collapse, those who were there in the 48 hours, those who spent extensive times there, clearly have a more likely coming down with these cancers, but I think it's biologically plausible that anyone that was subject to this is going to have an

increased rate of cancer so that my view now, given everything that I've heard, is that that cancer should be included.

We need a better mic system.

DR. HARRISON: Steve, this is Bob Harrison. Were you saying that we should recommend that all cancers be covered regardless of site?

MR. CASSIDY: I'm sorry? I think to say all is a broad statement; it really is. But I think that clearly the blood cancers, which are showing up early, I think anything related to the lungs, the respiratory system, anything that you can possibly inhale, so the esophageal cancers. You know, the fire department study proves that firefighters lost 12 years' lung capacity in the blink of an eye. That can't be dismissed as -- if that didn't exist people would say well, maybe this dust cloud really isn't going to do anything to us. But it proved what happened. Twelve years lung capacity, so to say all? I'm not saying all but I think we should err on the side of, if there's any evidence, we should err on that side.

MS. FLYNN: I really appreciated **[identifying information redacted]** comments, and I just want to say that I think that this is obviously not a deliberation that should use, you know, scientific certainty; this has been said before.

As his basis, he talked about a 51-percent of, you know, using the phrase that Steve Markowitz used earlier: We can reasonably anticipate that these cancers are linked to World Trade Center exposures, and right now that sounds pretty right to me.

I also want to add that the community cannot be left out of this deliberation, and also that the James Zadroga Act, and I can provide pages to folks if they want them, provides for one list of World Trade Center-covered conditions.

And we all know as erratic and full of gaps as the sampling information was on the Pile, you know, how much more is not known about community exposures. But what we do know is that members of the community, residents, students and area workers have the same respiratory and the same set of aerodigestive 9/11-related illnesses as responders, and it's more than reasonable to anticipate that they would develop the same set of cancers.

MS. HUGHES: I also just wanted to -- I'm not a biology expert, but I did go online and if we could break the body down into different body systems, like respiratory, and then look at the different things that could be impacted, so it is not just necessarily the lungs but it's the throat, so we're looking at a comprehensively wide body system so I just wanted to add that as well.

ADMINISTRATIVE ISSUES AND ADJOURN

DR. WARD: So we do need to leave the building shortly. So again I'm trying to sum up the sense that I'm getting. It seems that many people are in favor of listing at least some cancers of some systems as World Trade Center-related conditions, so I

guess, you know, your homework assignment is to really maybe clarify your own position as much as possible, and try to come up with potential statements that you think the group could agree on, and y'all certainly be thinking about it, but I'd like, you know, others as well to come in with, I think this is the sense of the committee and we can capture it in these words. That would really I think move us along in the morning.

So well, I did want to thank everyone who's here, both those who spoke and those who did not speak. I think, you know, the public comments are very informative. I think the discussion today was very informative, and I hope we've moved towards -- we've moved forward in the process of making a recommendation.

DR. MIDDENDORF: Let me also express my thanks and thanks for NIOSH and the World Trade Center Health Program, for the participation of everyone.

Steve, your wish is our command. We will be in conference rooms A and B tomorrow. And the speaker system will be better. It's not perfect but it will be better. So for any members of the public who intend to come back, we will be at the other end on the same floor. Thank you and good night.

(Meeting adjourned for the day at 5:05 p.m.)

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I, Steven Ray Green, Certified Merit Master Court Reporter, do hereby certify that I reported the above and foregoing on the day of February 15, 2012; and it is a true and accurate transcript of the proceedings captioned herein.

I further certify that I am neither related to nor counsel to any of the parties herein, nor have any interest in the cause named herein.

WITNESS my hand and official seal this the 9th day of March, 2012.

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