BAY AREA
AIR QUALITY
MANAGEMENT
DISTRICT

ADVISORY COUNCIL MEETING

WEDNESDAY
APRIL 13, 2011
9:00 A.M.

7TH FLOOR BOARD ROOM
939 ELLIS STREET
SAN FRANCISCO, CA 94109

AGENDA

CALL TO ORDER

Opening Comments — Ken Blonski, Chairperson
Roll Call — Clerk

PUBLIC COMMENT PERIOD

Public Comment on Non-Agenda Items, Pursuant to Government Code Section 54954.3. *The public has the opportunity to speak on any agenda item. All agendas for Advisory Council meetings are posted at the District, 939 Ellis Street, San Francisco, at least 72 hours before a meeting. At the beginning of the meeting, an opportunity is also provided for the public to speak on any subject within the Council's purview. Speakers are limited to three minutes each.*

CONSENT CALENDAR

1. Approval of Minutes of the March 9, 2011 Advisory Council Meeting.

DISCUSSION

2. Discussion of Draft Report on the Advisory Council's March 9, 2011 Meeting on Ultrafine Particulate: Health Effects Measurement and Analysis

The Advisory Council will discuss the Draft Report on the March 9, 2011 Meeting with Air District staff and finalize the recommendations.

3. Discussion of Advisory Council Members attending the Annual Air & Waste Management Association (A&WMA) Meeting in June.

The Advisory Council will discuss Council Members attending the annual A&WMA meeting in June 21-24, 2011.

OTHER BUSINESS

4. Council Member Comments/Other Business

   Council Members may make a brief announcement, provide a reference to staff about factual information, or ask questions about subsequent meetings.

5. Time and Place of Next Meeting

   Wednesday, May 11, 2011, at 9:00 a.m. at 939 Ellis Street, San Francisco, CA 94109.

6. Adjournment

CONTACT EXECUTIVE OFFICE - 939 ELLIS STREET SF, CA 94109

(415) 749-5130
FAX: (415) 928-8560
BAAQMD homepage:
www.baaqmd.gov

- To submit written comments on an agenda item in advance of the meeting.
- To request, in advance of the meeting, to be placed on the list to testify on an agenda item.
- To request special accommodations for those persons with disabilities notification to the Clerk's Office should be given in a timely manner, so that arrangements can be made accordingly.
- Any writing relating to an open session item on this Agenda that is distributed to all, or a majority of all, members of the body to which this Agenda relates shall be made available at the District's offices at 939 Ellis Street, San Francisco, CA 94109, at the time such writing is made available to all, or a majority of all, members of that body. Such writing(s) may also be posted on the District's website (www.baaqmd.gov) at that time.

BAY AREA AIR QUALITY MANAGEMENT DISTRICT
939 ELLIS STREET, SAN FRANCISCO, CALIFORNIA 94109
(415) 771-6000

EXECUTIVE OFFICE:
MONTHLY CALENDAR OF DISTRICT MEETINGS

APRIL 2011

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Board of Directors Executive Committee *(At the Call of the Chair)*	**Monday**	**11**	**9:30 a.m.**	**4th Floor Conf. Room**
Advisory Council Meeting	**Wednesday**	**13**	**9:00 a.m.**	**Board Room**
Board of Directors Regular Meeting *(Meets 1st & 3rd Wednesday of each Month)* **- CANCELLED**	**Wednesday**	**20**	**9:45 a.m.**	**Board Room**
Board of Directors Mobile Source Committee *(Meets 4th Thursday each Month)*	**Thursday**	**28**	**9:30 a.m.**	**4th Floor Conf. Room**
Board of Directors Budget & Finance Committee *(At the Call of the Chair)*	**Thursday**	**28**	**Immediately Following Mobile Source**	**4th Floor Conf. Room**

MAY 2011

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Board of Directors Regular Meeting *(Meets 1st & 3rd Wednesday of each Month)*	**Wednesday**	**4**	**9:45 a.m.**	**Board Room**
Board of Directors Regular Meeting *(Meets 1st & 3rd Wednesday of each Month)*	**Wednesday**	**18**	**9:45 a.m.**	**Board Room**
Board of Directors Budget & Finance Committee *(At the Call of the Chair)*	**Wednesday**	**25**	**1:00 p.m.**	**4th Floor Conf. Room**
Board of Directors Mobile Source Committee *(Meets 4th Thursday each Month)*	**Thursday**	**26**	**9:30 a.m.**	**4th Floor Conf. Room**

JUNE 2011

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Board of Directors Regular Meeting *(Meets 1st & 3rd Wednesday of each Month)*	**Wednesday**	**1**	**9:45 a.m.**	**Board Room**
Board of Directors Regular Meeting *(Meets 1st & 3rd Wednesday of each Month)*	**Wednesday**	**15**	**9:45 a.m.**	**Board Room**
Board of Directors Mobile Source Committee *(Meets 4th Thursday each Month)*	**Thursday**	**23**	**9:30 a.m.**	**4th Floor Conf. Room**

LH – 4/6/11 (12:30 p.m.)
P/Library/Forms/Calendar/Calendar/Moncal

AGENDA: 1

BAY AREA AIR QUALITY MANAGEMENT DISTRICT
Memorandum

To: Chairperson Blonski and
Members of the Advisory Council

From: Jack P. Broadbent
Executive Officer/APCO

Date: April 6, 2011

Re: Advisory Council's Draft Meeting Minutes of March 9, 2011

RECOMMENDED ACTION

Approve attached draft minutes of the Regular Advisory Council's meeting of March 9, 2011.

DISCUSSION

Attached for your review and approval are the draft minutes of the March 9, 2011 Advisory Council meeting.

Respectfully submitted,

Jack P. Broadbent
Executive Officer/APCO

Prepared by: Lisa Harper
Reviewed by: Rex Sanders

AGENDA: 1

Bay Area Air Quality Management District
939 Ellis Street
San Francisco, CA 94109
(415) 749-5000

DRAFT MINUTES

Advisory Council Regular Meeting
9:00 a.m., Wednesday, March 9, 2011

CALL TO ORDER

Opening Comment: Chairperson Blonski called the meeting to order at 9:06 a.m.

Roll Call: Chairperson Ken Blonski, M.S., Vice Chairperson Stan Hayes; Secretary Robert Bornstein, Ph.D., and Council Members Sam Altshuler, Louise Bedsworth, Ph.D., Benjamin Bolles, Jeffrey Bramlett, M.S., Harold Brazil, Peter Chamberlin, Jonathan Cherry, AIA, Alexandra Desaultels, Stan Hayes, John Holtzclaw, Ph.D., Kraig Kurucz, M.S., Gary Lucks, JD, CPEA, REA I, Liz Lutzker, MPH, Jane Martin, Dr.Ph.D., Kendall Oku, Jonathan Ruel and Dorothy Vura-Weis, M.D., M.P.H.

Absent: Member Jennifer Bard

Public Comment Period: There were no public comments.

Consent Calendar:

1. Approval of Minutes of the February 9, 2011 Advisory Council Meeting

Council Action: Member Holtzclaw made a motion to approve the minutes of February 9, 2011; Member Bramlett seconded the motion; carried unanimously without objection.

PRESENTATION: ULTRAFINE PARTICULATE

2. Ultrafine Particulate: Health Effects, Measurement and Analysis

   A. Measurement and Analysis of Ultrafine Particulate
   Susanne V. Hering, Ph.D.
   President
   Aerosol Dynamics, Inc.

Deputy APCO Jean Roggenkamp introduced and gave a brief background of Susanne Hering, Ph.D., founder and President of Aerosol Dynamics in Berkeley, California.

Dr. Susanne Hering began her presentation on historic perspectives and recent developments of ultrafine particle (UFP) measurement, noting there are both health issues and climate implications. She reviewed global climate issues and presented satellite photo of clouds formed from particulate emissions, their effect, and their magnitude for atmospheric aerosols. One health effect from UFPs that grow large enough to become nuclei for cloud formation is the effects on global climate; the likelihood of droughts. With regard to their distribution with respect to size, they can be presented on a number concentration or mass concentration, and particles differ in their size, chemistry, morphology, optical characteristics, and hyggroscopicity, and she presented examples of each. Size distribution of particles can be presented on a number basis or mass basis, and described each, stating most particles when counted are tiny, while the mass distribution is weighted more towards larger particles.

She provided an overview of ambient number concentration data, and presented particle number concentrations from winter time data at night in Fresno taken as part of an Air Resources Board (Kraypac) study, which show concentrations vary daily from 10,000 to 100,000 particles per cubic centimeter. Dr. Hering presented size distribution of ambient particles during early morning rush hour and nighttime when there are heating and stagnation effects, showing a movie of changes occurring from morning to evening.

The simplest way to characterize the concentration of UFP is to measure the total number of the concentration because the UFPs dominate the particle number. This is an important indicator in regard to health issues, as well as climate and the number of cloud droplets. It is also the oldest measurement in the atmosphere, and Dr. Hering reviewed the first measurements dating back to 1889. She presented John Aitken's work in Scotland on the number of dust particles in the atmosphere, and described his work on measurement. The highest concentrations Dr. Aitken saw were indoors in a room near the ceiling. She presented concentrations measured in a car when going through the Caldecott Tunnel and in a residential kitchen which significantly increase once an oven is turned on. One of the constant sources was found to be pilot lights, especially faulty ones. Combustion sources produce UFPs which are all below 100 nanometers in size. She presented the Kelvin Relation, noting that the elevation required is proportional to the surface tension and it is inversely proportional to the particle diameter or particle radius. Supersaturation increases as one over the particle diameter.

Dr. Hering presented various approaches to creating the vapor supersaturation:

Adiabatic Expansion (water)
- Aitken, GE counter

Turbulent Mixing (various working fluids)
- Kousaka, Flagan, delaMora

Thermally Diffusive – Butanol-CPCs
- Numerous manufacturers
- TSI (3760, 3010, 3025, 3775, 3776)
- MetOne, Grimm

Differentially Diffusive – Water-CPC
- 3781, 3783, 3785, 3786, 3787, 3788

Dr. Hering described the history of particle counting, as follows:

- 1890s: Aitken: first measurements of particle concentrations (by adiabatic expansion)
- 1912: Wilson Cloud Chamber: Applied Aitken's principle to particle physics (Nobel Prize, 1929) Determined precise expansion ratios for avoiding homogeneous particle formation.
- 1950s: Vonnegut: Automated adiabatic expansion counter - recognized particles grow to uniform size, fully automated counter.
- 1970s: Sinclair, Bricard: First Continuous flow particle counters (butanol)
- 2003: First continuous flow water-based particle counter

She then reviewed the Butanol based CPCs where heat transfers to the walls with minimal vapor transport, versus the water based CPCs, where both heat and water vapor are transported from the walls into the flow, but the water vapor moves more quickly resulting in supersaturation, allowing one to run a particle counter without butanol.

When measuring particles and identifying CPC performance characteristics, one should care about:

- Activation Cutpoint:
  - What is the smallest particle that can be detected
  - Does the response depend on particle type of chemistry?
- Concentration Range
  - For ambient sampling, ability to measure high concentrations especially important

Dr. Hering then described the response to traffic aerosol from the Caldecott Tunnel and individual trucks using both water and butanol instruments which give the same response.

Regarding high concentrations, they are a challenge if an instrument is chosen for monitoring. Particles are grown by condensation and they are sent through a light beam. Each particle gives a light scattering pulse, which is then counted. The problem lies in that when there are high concentrations, pulses start to pile up and there several droplets in the light beam all at once and they get much more difficult to count. There is not time for the signal to go back down to zero before the next particle leaves. When instruments shift, there is a calibration gap. She presented an example of a single count vs. photometric mode which reveals a transition gap when instruments shift from single counting to photometric mode. Therefore, the single count mode gives much more consistent data.

To extend the single particle count to high concentrations which are relevant for ambient sampling, one looks for fast, symmetric, uniform pulses, faster electronics, and then accurately account for dead time (the time the particle is in the light beam). Dr. Hering said this was done as an example on the Caldecott Tunnel on a calibration and checking at high dilution factors. It is possible to get rid of this mode and there are instruments that will count reliability up to 1 million counts per cubic centimeter.

In summary, Dr. Hering said when looking at UFPs and asking how to monitor for those, the best single indicator is to measure the particle number concentration. An alternative would be to do a complete science distribution, but there are complications with many numbers to report. She noted that to monitor by number, most ambient particles are "ultrafine." Particle number

concentration is the simplest approach for ultrafine particle monitoring. Particle number concentration measurement is well developed, the oldest air quality measurement, and the continuous flow instruments are more than 40 years old and water based instruments are over 8 years old.

Regarding their operating principle, condensation is used to grow particles to a size that is detectable optically by light scattering. Different instruments use different condensation approaches, but the end result is the same. Important performance characteristics in terms of what one would look for is what is the smallest minimum detectable size and then looking at what the upper concentration limit is because data in ambient air will go to very high concentrations.

Dr. Hering said lastly, it is important to realize that some of the highest concentrations or UFPs are actually not outdoors but indoor where there are close combustion sources from poorly operating furnaces, cooking appliances, pilot lights, and other combustible sources.

Advisory Council members thanked Dr. Hering for her presentation.

A. Health Effects of Ultrafine Particulate
John R. Froines, Ph.D.
Professor, Environmental Health Sciences
School of Public Health
UCLA

Deputy APCO Jean Roggenkamp introduced and gave a brief background of John Froines, Ph.D., Professor of Environmental Health Sciences at UCLA's School of Public Health, a faculty member of UCLA's Institute of the Environment and Sustainability and Director of the Southern California Particle Center, and chairs the State of California's Scientific Review panel.

Dr. John Froines said his presentation would be extremely complicated, noted that considerable research has been done recently where they have identified significant new health effects and toxicity associated with course particles. They see oxidative stress, inflammatory markers, and toxicity. The issue of UFPs versus PM2.5 and course particles is not a simple whatsoever, and it will take a while to sort out.

Secondly, he emphasized that in their research center, they never violate a rule that when measuring emissions or exposure, they always link it with toxicology or human health effects. It is crucial to link measurements with health effects and these cannot be separated. He believes standards are needed for UFPs, but they do not know yet what they want to measure, stating in Europe they measure non-volatile numbers, which the U.S. considers a waste of time and having nothing to do with health effects.

Dr. Froines said in 1997, the National Research Council conducted a report which revealed there is uncertainty in health effects associate with particles; however, he noted that EPA has pulled back funding for this.

Important to recognize is that in the last 12 years, we have seen premature death and lung cancer and he guarantees that cancers will be seen at other sites. Cancer is not a single organ

phenomenon but can be created in organs throughout the body, in lung, breast, and other illnesses. They have seen increases in these, as well as adverse birth outcomes. Diseases are multi-factorial and areas requiring more intense and wide ranging study. He said most important are semi-volatile compounds which start out in a vapor or particle phase, and these are what causes disease—metals and semi-volatiles. So, what we should be measuring are denuded semi-volatiles, metals and secondary organic compounds.

He provided an outline of what their Southern California Particle Center does. The perspective of this research is about characterization of PM for the chemical reactivities associated with adverse health effects, pro-oxidant effects and electrophilic activity. These are irreversible reactions from DNA. They focus on cellular nucleophiles, thiols and amines as targets of the reactive species. Their operating hypothesis is that toxicity of a number of chemicals due to two primary reactions that determine disease outcome:

- Oxidation of sensitive proteins and DNA by reactive oxygen species (ROS) leading to oxidative stress;
- Irreversible alkylation of reactive proteins by electrophiles present in pollutants

Pollution that causes electrophilic reactions leads to disease outcomes. Their research is about what happens when you have chemical reactivity to begin, what happens with the 20 to 30 steps in-between, and the result or outcome. They are interested in chemical reactivity to:

- Assess the magnitude of early changes that would lead to illness and disease exacerbation
- Ability of sample to catalyze electron transfer from oxygen to generate ROS, oxidative stress, impact on signaling pathways and gene expression
- Ability of sample to catalyze generation of hydroxyl radial by Fenton reaction catalyzed by metals
- Ability of sample to irreversibly inactivate biological molecules by covalent bond formation

He discussed chemical assays for pollutant activity:

1. Dithiothreitol (DTT) based redox assay
   Measures thiol oxidation capacity, using DTT as the thiol
2. Dihydroxybenzoate (DHBA) based redox assay
   Measures metal based hydroxyl forming capacity by the hydroxylation of salicylate
3. Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) inactivation.
   Measures electrophile content by the loss of enzymatic activity due to alkylation

These assays provide quantitative measures of the capacity of a given sample to carry out the reactions indicated. They can compare the capacity of different samples to carry out these reactions. Current studies examined:

DEP samples provided by the EPA
Particle and vapor samples collected simultaneously in Riverside, CA.

Dr. Froines presented a slide that shows what happens with particles going a distance from the freeway and as distance progresses, particles tend to grow with the mass staying the same.

Dr. Froines displayed what happens with particles going a distance on the freeway, high levels of ultrafine particles and as distance progresses, particles tend to grow and mass stays the same. He emphasized that concentrations of nanoparticles are much higher in the winter than summer because particles are formed when combusting fuel; however, what comes out of the tailpipe are hot vapors which then cool and form particles. These have very high semi-volatile concentrations, and one could argue that those particles that do not have significant carbon cores potentially have more vio-availability and be more toxic.

Dr. Froines emphasized that if technology is used to control particles and put filters on tailpipes, this will not cut it. What will cut it is if you can put filters on and also dealing with particles formed by nucleation condensation.

He presented a slide showing three emission sources—1) emission sources of air pollutants (traffic, freeways, ports, power plants); 2) Atmospheric dilution where particles are diluting and new particles are formed, some of which are ultrafines and others with different chemical constituencies. One of the greatest crimes he thinks in this field is that we have emphasized PM and we have ignored vapors which should not have been done. 3) Atmosphere aging and photo-chemical reactions (photo-chemical reactions of gas phase SVOC with O3 and oxidant gases and formation of secondary organic aerosol formations.

He then presented a study measuring the rate of air across the basin and certain chemicals going across the basin. What happens is that there is phenanthroquinone content; naphthoquinones and phenanthroquinone in Santa Monica, and high levels in Riverside. The two compounds produce oxidative stress.

He presented a chart of median pollutant concentrations by roadway and urban area, noting that as PM2.5 goes down, numbers of particles skyrocket. Similarly, as the number goes down, the particle mass goes up. He presented a Caldecott tunnel study by Kirchstetter in 1997 and UCLA's work in 2004 showing that as PM2.5 goes down, particle numbers go up. Therefore, he questioned whether we were measuring the right thing in PM.

He then presented a graph of redox activity (DTT assay) of semi volatile and total PM from newer diesel trucks. Particles are heated and semi volatiles come off and are captured in XAD resin traps. Particles contain significant amounts of semi-volatile particulate matter and when heated, this is lost. The point he makes is, should we forget size as the measure of what we regulate or should we be regulating organic compounds in metals. He would advocate measuring organic compounds in metals.

He presented a study of the Los Angeles Port, pointing out that black are ultrafine particles, grey are fine particles, and the darker grey are course particles. He said there is no question that when it comes to oxidative stress and the toxicity associated with it, ultrafines are the highest.

Dr. Froines gave concluding remarks, stating they are very concerned with goods movement in both southern and northern California ports:

- Newer vehicles have increased emissions of semi volatile organic compounds (SVOC) ultrafine PM; redox activity much higher than nonvolatile PM;

- These particles may pose a greater risk to public health;
- Dilution of air parcels from the point of emission drives SVOC species off the PM phase into the gas phase;
- A better understanding of the linkages between the SVOC phase, chemistry and toxicity in order to adopt regulatory strategies that protect the public

He then showed mitochondria: an important subcellular target of PM and a source of ROS generation on slide 11. Ultrafine particles not only enter the cell, but they enter the mitochondria which mean they are having a negative impact of the electron transport process in the mitochondria. The slide shows that ultrafine particles not only are small but they are small enough that they enter the cell by diffusion, which means they can go through membranes.

He presented results of comparison of three sites; Riverside, USC and Westwood. Important is that the electrophilic activity is 10 times higher in the vapor phase. The irreversible finding of chemicals with proteins and DNA are 10 fold higher than in the vapor phase while the redox activity is 7 to 10 fold higher in the PM 2.5 particle phase. Therefore, there is a contradiction. We are saying that highly toxic chemicals are demonstrating toxicity in the vapor phase. As suggested earlier, we have been guilty of not paying attention to the vapor phase. They find iron (Fe) and copper (Cu) are metals important in terms of hydroxyl radical formation. Copper is 60 times more toxic than iron. Electrophiles have been shown to activate a transcription factor; protein Nrf2, which actually results in the formation of enzymes that are protective. They have also seen electrophiles activate the epidermal growth factor receptor which activates signaling pathways leading to transcription factor activation of inflammatory pathways. Therefore, in looking at what is and is not toxic, there are protective processes and adverse processes, and the relative rates of competition between those processes must be looked at, which he said is done now.

He then reviewed cell response to air pollutants: oxidative and electrophilic stress, actions of prooxidants and electrophiles, prooxidant content in particles and vapors, and the point he made was that ultrafine particles will get into the epithelial cell and chemicals can react with lung-lining fluid. Important is that lung-lining fluid can contain high quantities of ascorbic acid or vitamin C, which actually then creates reactive oxygen species and other qualities. Ascorbic acid produces electrons, which react to oxygen to form reactive oxygen species, and reactive oxygen species are part of the toxic problem.

Dr. Froines then discussed a study on atherosclerosis disease showing mice are susceptible to heart attacks. Mice were kept in non-exposed, filtered air, PM2.5 and PM0.1 (UFP). They did a 5 hour, 3 times a week session for 5 weeks and were interested in aortic atherosclerosis assessment. He said in looking at metals and organic carbon in comparing PM2.5 and UFPs. In UFPs there is 52% organic carbon. With PM2.5, there is 25% organic carbon. Therefore, for disease processes, this shows a prediction that PM2.5 will be toxic but not as toxic as UFPs.

He presented histologic slides of aortic lesions in the heart resulting in the most significant impact. One important thing about low density cholesterol; good and bad cholesterol is that low density cholesterol is bad cholesterol for more reasons than one realizes. It gets oxidized by reactive oxygen species and it impacts the high density cholesterol. So, air pollution has an impact on low density cholesterol oxidation which facilitates the atherosclerotic process. This is why emissions and exposure can be studied, but most important is to study what happens from it.

He presented the number of aortic atherosclerotic lesions, and UFPs demonstrate much more in the way of aortic lesions.

Regarding adjuvant (or exacerbated) effects of UFPs resulting in allergic airways disease including asthma, Dr. Froines said they looked at all regions in the mouse from the nasal passages down to the Alveolar region and toxicity effects were found in every region. He described primary versus secondary immune responses when providing antibodies. OVA and UFPs produce allergic airways disease and asthma. When a second and third dose of UFPs are given, the same kinds of histology are seen. The inflammatory process is immense and UFPs will cause exacerbation of allergic airway disease and exacerbation of asthma. The bottom line is that humans get cancer, asthma and heart disease, and he feels it is important to understand the complexities of those processes.

While academic, the District needs to determine what it must regulate and if not enough is known about mechanism, it will not regulate the right material.

Dr. Froines then briefly reviewed and discussed other assays in cells they study, displayed chemical pathways for cellular effects. They reviewed ultrafine particles and elderly people in assisted living facilities who had had heart attacks and looks at their biomarkers of inflammation, ambulatory blood pressure, electrocardiographic ST and airway inflammation. What they saw is air pollutant particles are associated with increased ambulatory blood pressure with seniors citizens exposed to UFPs and they saw circulating biomarkers.

Dr. Froines then presented a children's health study done in 12 cities in Los Angeles. As elemental carbon, which is a surrogate for diesel, goes up the amount of air you can breathe out in one second becomes more difficult. This shows that diesel exhaust has an impact on lung function in children. It gets less difficult away from major roads but the problem with epidemiology is that this does not indicate what causes it. They must go back and identify what is causing the problem, and it will not be the number of UFPs or the mass of UFPs. He reiterated that PM 2.5 is not the causal agent for disease in air pollution, but rather it is a surrogate for health impacts.

Children's lung growth over time is dramatically affected by air pollution. This leads to the fact that when the child reaches 65 or 70, there will be a problem with cardiovascular disease because lungs have been impacted because they never grew to begin with. He said important to understand is there are upstream and downstream effects one should be concerned with. He stated they took mice and based on work done at the University of Rochester that UFPs are trans-located to the brain where they stimulate brain cells to produce inflammatory markers. It turns out that those markers are crying and it does not go away overnight. Every day when one is commuting and getting UFPs, the implication is any number of central nervous system disorders.

Dr. Froines added there is a whole new field of science emerging called "upstream downstream science" and what it is trying to do is to identify upstream events like the ones presented and work to find relevance to downstream apical disease. So, if this transcription factor for inflammation he presented has relevance to the exacerbation of asthma, then he questioned why the transcription factor cannot be regulated. If we understand mechanisms, we can regulate and do risk assessments on upstream assays like transcription factors which are things that lead to disease. Therefore, there is no reason why epidemiology must be done and always do human

based studies, and cellular biological assays can be done and the District could regulate on that basis. This would cause an extraordinary improvement in our understanding of what is going on.

Conclusions:

- To determine the contribution of PM sources (both primary and secondary) to exposure and evaluate their relative toxicity.
- The SCPC has addressed the effect of vehicular emissions for the nation.
- This is clearly a national problem ranging from goods movement to global warming
- Pro-oxidant and electrophilic chemistry play a crucial role in health effects
- Advanced understanding of mechanism dramatically
- Demonstrated multiple endpoints heretofore unrecognized
- Important role of ultrafine particles vis a vis health effects
- Important role of ultrafine particles vis a vis health effects
- The role of vapors is vitally important;
- Speculations that non-familial neurodegenerative diseases are related to exposures to environmental toxins
- Pathways of inflammatory responses to primary and secondary organic aerosols (POA & SOA) in elderly populations
- Air pollutant particles are associated with increased ambulatory BP in elderly subjects with coronary heart disease.
- There is strong evidence for adjuvant effects in respiratory/allergic illness.
- Upstream biological and chemical events have been identified with relevance for assessing risk

Going back to 1998, he presented an article headline from the Los Angeles Times which names diesel exhaust as posing a strong cancer risk, which amazingly, is still being debated. Diesel exhaust was proven a carcinogen in 1775 and he believes the burden should be on the industry, which he reviewed along with their associated health impacts. He said several studies dating back to 1998 all demonstrate an increased risk of lung cancer from diesel, and biodiesel is not the answer. Vegetable oil becomes rancid and therefore produces toxic chemicals.

Dr. Froines then reviewed a USC study of downtown London, and he displayed people walking down Oxford Street getting heavy doses of diesel from buses, taxicabs, and cars. He then showed a picture of Hyde Park where one would conclude there is not a lot of diesel. They took people with light asthma and heavier asthma. One group walked through Hyde Park for 2 hours and the other group downtown. He presented the results, stating lung function dramatically declines for individuals in the downtown as compared to lung function at the park.

The final slide he presented shows the risk of low pre-term and low term birth babies as a function of trucks and heavy duty vehicles. The risk is elevated if one is in proximity to trucks. This demonstrates what they call "developmental effects" and spontaneous abortion, miscarriage, birth defects, toxicity, and developmental effects. No one has done a study of neurodevelopmental effects, or what happens to the fetus' brain, and he said this should be done.

PANEL DISCUSSION

3. Health Effects, Measurement and Analysis of Ultrafine Particulate

Mr. Kendall noted that Advisory Councilmember Sam Altshuler will serve as the lead author, with Dr. Dotty Vura-Weis and Liza Lutzker also serving. Mr. Kendall indicated he would like to meet with these individuals after the meeting.

Chairperson Blonski noted that the following questions submitted to speakers:

a. Are ambient air quality and/or emission standards for ultrafine particulate necessary?
b. If so, at what level and form:
    i) level
    ii) measurement units (mass, particle count, surface area)
    iii) averaging time (one hour, 24 hours, annual, other)
c. What would be the best way to measure ultrafine particulate for ambient air quality and/or emission standards?
d. Should ultrafine particulate monitoring be conducted in the Bay Area and, if so, what locations should monitoring focus on?
e. What are the implications of ultrafine particulate for the Air District's regulatory and legislative agendas, and programs?

Dr. Holtzclaw referred to the semi volatiles and asked how much do they change during measurement, from gaseous state to particulates or vice versa? Secondly, do they grow during measurements; is there aggregation during the measurements themselves? When they are condensing water around them, does this keep them from aggregating or do they condense together?

Dr. Hering said she talked about measuring concentrations as a practical way to assess ultrafine levels. It is a surrogate and it would need to be accompanied by some of the more complex forms of measurement on a study basis so that you are always looking at the correlation of toxicity, the reactive oxidant stress level. The question must address whether or not the changes in combustion sources have changed that toxicity relative to the number of concentrations. ROS measurements cannot be done to achieve measurements 24 hour at 50 sites in the basin; therefore, one would look at the concentration measurement.

Regarding whether or not that measures semi volatiles, you are just counting particles, so if you lose some of the semi volatile material off the particle in the counting process, you do not see it. So, you will still count that particle even if the measurement process causes a little bit of loss.

Regarding coagulation, whether or not there is a practical problem with particles conglomerating together in the measurement process, at any reasonable concentration level, these measurements move quickly. One would enhance the coagulation probability by making them all larger, but it is measured in a second or half second, so there is not enough time for much coagulation.

Dr. Froines agrees that the Europeans are using particle number not knowing or ignoring all the science. It could be that you could decide to do particle number as the District's regulatory monitoring element, but what worries him is that what it boils down is a dose response curve. Doing it by numbers may give a steeper slope and regulations will be more rigid. He noted that 80% of UFPs people are breathing are during commute. He said they found lots of toxicity is lost if they use a filter based sampler as opposed to an impinger-based sampler, and we must be careful that we are not losing material that we should be measuring. The issue of losses on filters is 40-50 years old.

Executive Officer/APCO Jack Broadbent said it is appropriate to ask both speakers that, given the breadth and depth of the District's control and monitoring program, he asked for comment on the sufficiency of what the District does, keeping in mind that the Advisory Council is charged with providing recommendations to the governing board who makes ultimate policy, rules and resources decisions. He asked for comment about where the organization should go, as there is much more to be done.

Dr. Hering said in thinking about measurement of criteria gases versus where the measurements of particles have been on a monitoring basis, particulate measurements are in the dark ages. Ozone is a specific molecule and this is measured with minute or five second resolution. It is reported as hourly averages. It is a specific compound with health impacts and one to regulate. The same thing could be said for carbon monoxide. She described first monitoring devices and their performances by pulling air through filters for 24 hours and then performing measurements.

The first advance was PM10; it was collected on a sample. For PM2.5, it is smaller, but it is a surrogate and does not even address what is the health effect, but rather something correlated to it. She thinks whatever is monitored, there is a need before a robust monitor but there is the danger that because it is being monitored, it is the end thing that needs to be controlled, which is the pitfall to avoid.

On the other hand, Dr. Hering said she would also push to get particulate surrogate or a handful of surrogates on the same time resolution and the same data completeness as done for the gases. Getting it every 3-6 days gives holes, so getting a consistent time base on something is important to establish geographical and seasonal differences and for identifying problems. However, unlike ozone, this is not going to be true for what is being measured with particles.

Dr. Froines said he slightly disagreed, stating they have discovered that no one practically in the world knows—if you take ozone and expose it in the presence of polycyclic aromatic hydrocarbons, which is what we do when we breathe, the ozone oxidizes the polycyclic hydrocarbon and that produces quinones which generate reactive oxygen species. This will cause heart defects as well as respiratory effects.

Therefore, they have to be very careful that they are working to figure out what is going on because we are not there yet. It is much more complicated and it may mean the District will have to regulate ozone to prevent reactive oxygen species formation, and this is a different thing than worrying about somebody's lungs.

He said in his view, the District should monitor close to transportation sites. The District should look at the issue of dilution, which is complicated. Secondly, he would go 300 meters away from a freeway to have a background, and the last thing he would say is the District needs to deal with the receptor site issue. Quinones are formed and you must deal with the secondary organic species that are formed which will have different characteristics than primary emissions or dilution. The District should deal with secondary emissions, primary emissions, transportation, and then metals, which cannot be ignored. The last one is vapors. The District cannot ignore them because they are more toxic than the reactive toxic species. The District needs to figure out what vapors it wants to measure, such as Acrolein, which is super-toxic; however, there is very little of it in the air and not a good surrogate.

We need to look at chemicals in a broader issue. If vapors are proposed, the ARB should assist the District in determining which are most appropriate.

Mr. Broadbent said the Bay Area struggles because there are a number of policies that will continue to densify the Bay Area, including creating livable communities and getting people out of their cars. Often many land use decisions are those that put people at greater risk. Dr. Froines suggested holding a series of workshops over time where these issues get discussed and over time, some answers begin to emerge that cannot all be resolved in a two-hour session. He felt it will take some work and he knows government can sometimes be hard to move. He would say that the most progressive forces in the State on air pollution are the air districts and the bottom is EPA at this point. He discussed the $40 million in research funds; the EPA has seems to have lost its interest in PM which he felt was absurd.

Sam Altshulter said over the years there has been the issue of asthma, and as criteria pollutants have dropped, asthma rates have gone up. He asked if the UFPs are the missing link to explain this. Dr. Froines said in his review, ultrafines is not the answer but whether it is considered from the point of view from mass or number, is a surrogate that one can decide on whether or not to move ahead on that level. He showed the steps that occur in the cell that causes asthma. It is not a simple thing like ozone, but the chemistry leading to asthma. Therefore, the question of whether we should take this transcription factor that binds with DNA and then produces inflammatory effects--is this enough to regulate? He would say yes, but others might say no.

Mr. Altshuler said a number of years ago there was a study reported at the Air and Waste Management Conference that showed the I-110 freeway in Los Angeles that does not have diesel traffic on it, which showed a high concentration. He presumed it was gas powered vehicles and issues were lube oil, and he asked if the same level of effort was going on to understand the effects of lube oil and emissions occurring relative to diesel or is it silently off to the side? Dr. Hering said when looking at the chemistry of the particulate matters, from diesel trucks there is a lot of polycyclic aromatic hydrocarbons associated with that PM and has its origins in the lube oil. In a broader sense, knowing the chemistry of these particles is critical. Dr. Froines agreed, and said this is why caution should be taken in relying on CNG buses because it may be the oil is just as an important factor.

Dr. Hering added that even though there is a huge difference in the mass of particulate matter that comes from diesel engine as compared to a gasoline engine, there are still a huge number of particles that come from spark emission engines, so their composition is fairly different but also in question.

Dr. Froines said in looking at cars in Europe years ago versus now, the number of particles has gone up dramatically. The issue of numbers versus mass is not something recognized. When they did the Caldecott Tunnel study, they found the toxicity of the gasoline particles was greater than those from diesel vehicles.

Mr. Altshulter said in trying to evaluate this and add value, he questioned what is different in the Bay Area than Los Angeles or other areas, as there are different indoor exposures, the environment allows for windows to be opened differently, and different heating systems. Dr. Hering said in the East Bay, people are crammed between I-80 and the hills. So, it is the combustion sources that provide ultrafine particles. Cooking, furnace, cigarette smoking,

candles, and outdoor sources are traffic. In looking at land use areas, there have been comments that given concerns about lung development, schools should not be located near freeways, and Dr. Froines agreed that schools and parks should not be located near freeways, and he discussed a situation in Long Beach and a school site.

Mr. Broadbent noted the Bay Area has similar sources but less of them. What the Bay Area does have is a much more dense population base which is somewhat unique and is the reason why there is so much discussion on land use regulation and decisions, in addition to the control program. He added there is also a significant amount of public transit used by children as well.

Mr. Hayes referred to current federal and state standards, which are written in terms of PM2.5 and in terms of mass. The District must develop a control program to achieve those and will have to pick from a menu of control measures. Knowing everything Dr. Froines knows about measurement and health effects of UFPs versus PM, he asked what the best advice is about how the District ought to take UFPs into account in the selection of control measures for the PM plan.

Dr. Hering said ultrafine concentrations are basically uncorrelated with PM2.5 mass. So, it becomes a problem that PM2.5 mass is itself a surrogate because it is now the pitfall of having now to control it, and there is an EPA regulation based on it. In controlling it and looking at PM2.5 mass, the District you will look at secondary nitrates, sulfates secondary organic matter, and probably none are correlated with the ultrafines. Therefore, they are two different questions. In developing strategies, the District must look at chemical composition first because it identifies where they came from. But the answer not achieved is polycyclic aromatic hydrocarbins, which are associated with ultrafines, because they are so miniscule.

Mr. Hayes said, therefore, it is likely that in designing a control program for PM2.5 mass, the District does not have a handle on the ultrafine piece at all, to which Dr. Hering agreed. She said this is why she thinks having a surrogate for ultrafines in the monitoring effort is perhaps justified because they are almost unrelated quantities. Dr. Froines agreed. He further elaborated on a study where, in using reactive oxygen species and calling it a measure of toxicity, for example, they compared mass-based ultrafines with numbered-based ultrafines, and he firmly could not choose between the two. Neither would be perfect, but there would be no major difference between the two, so the District would have to make a terrible decision, and this is why the District must link measurements with toxicity.

Dr. Hering said with regard to PM2.5, there are other implications other than direct health effects. The majority of the particles that comprise PM2.5 mass are the ones that affect visibility, have impacts on global climate, and it ultimately has effects on droughts, crops succeeding, which has its own set of worldwide issues.

Ms. Lutzker said she comes back to the indoor and outdoor concentrations and asked if it was 3 to 5 times higher UFPs indoors. Dr. Hering said it depends; if there are no indoor sources, they are lower. If cooking or if you have a furnace or hot water heater, the emissions get into the house.

Ms. Lutzker said looking at concentrations may not be the best way of looking at things as it is merely a surrogate, if there is a regulation of UFPs and it does succeed in reducing ultrafines outdoors by a certain percentage, she asked what contribution would it make to human health

given there are indoor exposures. Dr. Hering said the District can do a lot to control by getting rid of pilot lights and by replacing old furnaces which has a huge effect. She thinks it is a question largely ignored, given the fact that people spend 85% of their time indoors.

Dr. Froines reiterated that 80% of UFPs people breathe are those when commuting and he guessed that the chemistry and toxicity indoors may not be as significant as outdoors.

Mr. Kurucz said in making sure what gets measured gets managed and making a positive impact, using the envitro testing, he asked if there is a possibility to identify a set of indicator species that if measured, would lead the District to regulate something that would make an impact on all pollutants. He asked if there was a possibility to use Dr. Henry's methodology at USC using regression from the receptor model back to sources by fingerprinting pollutants that would lead to a strategy for regulating something that would make a positive impact. Dr. Froines said yes; while stretching it a bit, one could regulate air pollution on the basis of the activation of upstream assay transcription factor. It is not necessarily enough because the electrophiles and reactive oxygen species are different and research must be done to sort out the differences and competition with the Nrf2 which is adaptive and protective. He said work can be done, but he strongly believes we are at a place in the United States that we know enough cellular biology and genetic toxicology that we can say we can use an upstream event. We do not have to do epidemiology before regulating. We can go upstream and look for events like that.
He thinks that what we have done by looking at mechanism of disease is that we have created the possibility for identifying upstream assays you can use for policy purposes.

Dr. Vura-Weiss referred to the identification of NFkB, this is related to development of asthma, and she asked if there were other markers that would be related to some of the cancer causing effects or others. Dr. Froines said he believes this. They may be wrong, but he thinks prooxidant and electrophilic steps are the first 2 steps. He said there are different mechanisms and differences at the first two steps, and he discussed a chemical called Acrylamide which causes terrible peripheral neuropathy, it causes synapses to not function effectively, and this is a neurotoxic event. But, acrylamide causes pancreatic cancer, and he does not think the pancreatic cancer has the same mechanism that the peripheral neuropathy does and this is where one of the great scientific challenges is. Chemicals can cause lots of different diseases by different pathways. They believe they know the first two steps, but it can then become very complicated, and this is why he said a better understanding of Nfr2 and NFkB.

Dr. Froines said in looking at the State's Office of Environmental Health Hazards Assessment document on environmental tobacco smoke and look at the number of diseases that ETS causes, it is 20 to 25 diseases. To look at these things in simplistic terms does no one any good, and this should be about people valuing research and not just a question of getting the regulatory rules set so the EPA approves them. These are policy, regulatory, and risk assessment issues, so there is a need to link the science with the regulatory process and not have that regulatory process be so rigid the way it is now.

Mr. Lucks said in following up with Mr. Kurucz's question, he said the District's mission is to take science and turn it into regulation to the extent it is warranted. He said Dr. Froines was talking about looking upstream for the downstream effect and the idea of the transcription factor and using health risk assessment and cell biology as a mechanism. For the District's purposes, he asked to elaborate on what that would translate into in thinking of strategies. He asked if this

means a permit applicant would perform a health risk assessment. Dr. Froines said he was not fully confident in answering that question and suggested having a second workshop in which this was the subject matter. He said if he did an epidemiologic study similar to the study that looked at asthma incidents up to 300 meters or, if an asbestos study was done, the Council would be comfortable with that. Then if he took a mouse and a rat in a national toxicology program and they did two-year chronic animal bio assays and got positive results for cancer, there are some who would not accept this. So, for the most part we believe them and the sooner we get work done to start identifying what needs to be identified, the sooner the District will be able to translate this to policy and regulation. He discussed his chairing of a committee that did a study on the use of methyl iodine which was very controversial. They argued with the Governor over whether it should happen and the committee worked hard to try and identify the steps that would give them the upstream and downstream process. It can and should be done, the science exists for it, but agencies must buy into it. His view is that not only is it possible, but it is essential.

Dr. Bornstein summarized that the only possible way forward is that right now, we are observing surrogates and both speakers pointed out many times that the District needs to be more specific. It is not only we have many specific chemicals that do damage, but they also have synergistic effects, which makes things worse. So this leads us to depression, but we must move forward, and we come back to surrogates. We now have to get an optimum number and need to identify those things that stand in and capture the sources and health effects. Then government agencies can think about specific standards of indicators at what kind of sites, which we talked about as being where we should measure. There must also be some specificity as the Bay Area is different than other areas. Therefore, the only thing the District can do is keep up with the science so that when science provides the information, the District can attempt to pressure the government agencies to come up with surrogates to make more intelligible measurements that can control something that will have a health impact.

Dr. Hering added that having a consistent surrogate so there is a historical record is important, but the danger is having that surrogate be your end point of what you are going to control. At the same time, as different scientists develop different upstream measurements, NFkB may be what is viewed today as the best upstream measurement, but it could be something different. She said it is also a very expensive measurement as compared to setting up a particle counter. Therefore, science studies are needed and the ideal would be to do upstream measurements in coordination with surrogate measurements on a fairly routine basis.

Dr. Froines said he has said on many occasions about the competition between NFkB and Nrf2. Nrf2 produces protective enzymes. He added they did a study using a genetically altered mouse that was Nfr2 deficient. In other words, they made the mouse genetically incapable and they got stronger allergic inflammation, and the study knocked out one piece and reinforced the importance of the other side. Science can be done to differentiate things and he discussed a study using potentiation.

Mr. Kendall referred to slide 15 and voiced concern when looking at the slide. He said the control of diesel began in 1987 and later it started focusing on PM and NOx. There are people living by freeways with a lot of diesel traffic with health effects. From a policy perspective, the work has been successful in reducing PM mass and NOx coming from heavy duty diesel vehicles, but the particle count has increased by a factor of 10 when worrying about the PM and

by a factor of 100 when the District started worrying about reducing the NOx. He questioned if public health has been improved.

Dr. Froines said he showed results from three separate studies, and he said the health effects are real, and we should get over this notion of being locked into mass based measurements. The answer is, we do not know and a high priority research agenda would be to answer your question, as the answer to the question does not yet exist. He would say that we do not have to prove that people are dying to be able to say changes are needed to be made. While agreeing in principle, we have to get past epidemiology studies because they are too long and past animal studies and other studies because they are expensive and take too long. Epidemiology and animal chronic anobioassay studies can measure risk of 1 in 100 at best. Therefore, he said mathematical extrapolation must be done called risk assessment.

Mr. Kendall asked if diesel emissions can be controlled without increasing the number of particles. Dr. Froines said he thinks we need to deal with vapors and control that, and while he is not an engineer, we need catalytic converters, oil, primary emissions and vapors that come out of the tailpipe. If these three can be addressed, significant progress and strides would be made.

OTHER BUSINESS

4. Council Member Comments/Other Business

Chairperson Blonski recognized Dr. Holtzclaw and Jenny Bard for presenting the Advisory Council's recommendations from its February Council meeting at the March 2, 2011 Board of Directors meeting.

Chairperson Blonski reminded members on the need to complete Ethics Training, stating the District will hold a workshop on March 24, 2011 from 1:00 p.m. to 3:00 p.m. Individuals can also complete on-line training.

5. Time and Place of Next Meeting - 9:00 a.m., Wednesday, April 13, 2011, 939 Ellis Street, San Francisco, CA 94109.

6. Adjournment: The meeting adjourned at 12:18 p.m.

Lisa Harper
Clerk of the Boards

AGENDA: 2

BAY AREA AIR QUALITY MANAGEMENT DISTRICT
Memorandum

To: Chairperson Ken Blonski and
Members of the Advisory Council

From: Jack P. Broadbent, Executive Officer

Date: April 6, 2011

Re: Discussion of Draft Report on the Advisory Council's March 9, 2011 Meeting on Ultrafine Particulate: Health Effects, Measurement and Analysis

The attached draft report on the March 9, 2011 Advisory Council Meeting on Ultrafine Particulate: Health Effects, Measurement and Analysis was prepared by Advisory Council members Sam Altshuler, Dorothy Vura-Weis and Liza Lutzker.

The draft report will be discussed by the Advisory Council at its April 13, 2011 meeting.

Respectfully submitted,

Jack P. Broadbent
Executive Officer/APCO

Prepared by: Gary Kendall
Reviewed by: Jean Roggenkamp

DRAFT REPORT ON THE MARCH 9, 2011 ADVISORY COUNCIL MEETING ON ULTRAFINE PARTICULATE: HEALTH EFFECTS, MEASUREMENT AND ANAYSIS FOR DISCUSSION BY THE ADVISORY COUNCIL AT THE APRIL 13, 2011 MEETING

SUMMARY

The following presentations were made at the March 9, 2011 Advisory Council meeting on Ultrafine Particulate: Health Effects, Measurement and Analysis:

1. ***Research findings on particulate air pollution from the Southern California Particle Center*** by John R. Froines, Ph.D., Professor, Environmental Health Sciences, School of Public Health, UCLA. Professor Froines joined the faculty of the School of Public Health in 1981. He received a B.S. in chemistry from UC Berkeley (1963), M.S. in chemistry (1964) and Ph.D. in physical-organic chemistry (1967) from Yale University. Before coming to the UCLA School of Public Health, Dr. Froines was Assistant Professor of Chemistry at the University of Oregon and later served as Director of Toxic Substances at the Occupational Safety and Health Administration and Deputy Director of the National Institute for Occupational Safety and Health. Dr. Froines the Director of the Southern California Particle Center and Supersite. Dr. Froines' area of expertise is toxicology and exposure assessment. His research interests are in the qualitative and quantitative characterization of risk factors in environmental and occupational health. Dr. Froines chairs the State of California's Scientific Review Panel and the central review panel at the State level for identifying toxic air contaminants.

2. ***Ultrafine Particle Measurement: historical perspectives and recent developments*** by Susanne V. Hering, Ph.D., President, Aerosol Dynamics. Dr. Hering is the founder and President of Aerosol Dynamics, Inc. has over thirty years of experience in the field of atmospheric aerosols. Her primary interest is the in-situ, automated characterization of the size and chemical composition of atmospheric particles. Dr. Hering holds a PhD in Physics from the University of Washington, with postdoctoral work in Environmental Engineering at the California Institute of Technology. After many years at UCLA, she left the academic world to form Aerosol Dynamics Inc. She has served as a Board Member and as President of the American Association for Aerosol Research, and in 2007 was honored as the recipient of the BYH Liu Award, presented by the Association in recognition of contributions to aerosol instrumentation. Dynamics.

DISCUSSION MEETING

At the April 13, 2011 meeting, the Council discussed the presentations and the materials received at the March 9, 2011 meeting and the draft report.

KEY POINTS

Dr. John R. Froines

- Semi-volatile compounds go back and forth between vapor phase and condensation into (and onto) particles, interact more with cells in the vapor phase, can cross from alveoli into bloodstream and travel to other organs outside of lungs.

- UFP (acting as adjuvants) make the body more susceptible to developing allergies to various substances and to reacting more strongly when re-exposed to them. As particle mass decreases, the number of particles increases. So, PM mass based standards may not be measuring the right thing.

- Oxidative stress most associated with UFP (vs. other particle size). UFP enter mitochondria via diffusion (because they're so small) and disrupt the electron transport process.
- Chronic inflammation in the brain caused by UFP may have serious chronic neurodegenerative effects. This is an area for more study.
- We should not have to wait until multiple epidemiologic studies show that UFP are associated with adverse health effects. First, the UFP are not the true problem, only a surrogate, so we don't know what it is about UFP that is problematic. Second, epidemiology studies take a very long time, and simply amassing more and more studies takes a long time and is overkill in terms of asserting causality. Lastly, by looking at health outcomes (downstream), we miss the mechanisms that connect the particles (or other molecules) to the disruptive actions at a cellular level. In vitro toxicology studies can do this, so we should rely more heavily on toxicology studies (downstream) when deciding what to measure and/or regulate, especially if we already understand the connection between the downstream cellular mechanism and upstream health effects. You can actually regulate based on the cellular mechanism.
- Health effects include cancers in the lungs and elsewhere, asthma, cardiovascular disease, and probably neurological degenerative diseases

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Dr. Susanne V. Hering

- Ultrafine Particles (UFP), <0.1 microns in diameter, account for the largest number of particles in PM, but only a small proportion of the total mass of PM; they also have a much higher surface area on which to carry other molecules
- UFPs are more numerous in urban areas, especially near freeways, than in other areas, and during rush hour than overnight
- The technology exists to count the number of these tiny particles, regardless of their composition, but not necessarily to characterize their components
- Concentration does not say anything about particle composition

- UFP also have extremely high concentrations indoors (in fact often higher than outdoors), especially in kitchens and with furnaces, and especially when pilot lights run constantly

EMERGING ISSUES FROM THE ADVISORY COUNCIL

- High concentration of ambient UFP within 100 meters of freeways is cause for concern
- Indoor exposure to UFP can be significant relative to outdoor exposure to UFP.
- Chemical characterization of UFP is needed to assess potential health effects and sources of UFP. The actual cellular-level changes are probably more specific to the component of the UFP than to the overall particle concentration, but this needs to be studied.
- Specific causes of health effects due to exposure to UFP nitrates and sulfates are not well known or defined. Both are in need of more research as they can constitute a significant fraction of ambient PM2.5, likely also in the UFP size range.
- Concentrations and health effects of vapors of organics and metals need to be better understood.
- Particle size distributions are affected by diesel PM emissions control systems. Where significant diesel PM reductions have occurred (some funded by BAAQMD grant funds) in sensitive areas (Port of Oakland) studies are needed to evaluate the potential increases of UFP concentrations and potential impacts on nearby residents.
- UFP may be emitted by from engines as a result of lube oil consumption. These UFP may be laced with metals (iron, copper, and zinc) and organics in both the particulate and vapor phases.
- High UFP may occur disproportionately from gross emitting vehicles.

ADVISORY COUNCIL RECOMMENDATIONS

The following Advisory Council recommendations to the Board are based on: the above presentations, and subsequent discussions among Advisory Council members. The Air District should:

1. Set up a UFP monitoring program in the Bay Area near traffic sources (and away from them to get background) to monitor these identified substances. Concentrate monitors in environmental justice neighborhoods, perhaps near the Port of Oakland or in conjunction with EPA required NO2 monitors.

2. Track continued research on health effects and mechanisms for causing health effects regarding UFP and its various constituents. Pay particular attention to vapors, metals, organics, sulfates, and nitrates.

3. Look more closely at the UFP emissions from all internal combustion engines (gasoline, diesel, CNG, propane, hydrogen, ethanol, etc.) as well as the contribution to UFP emissions due to lube oil. Pay attention to the role of gross emitters.

4. Compare more closely the relative UFP exposures occurring indoors and outdoors and provide summaries for public education.

GLOSSARY