Bay Area Air Quality Management District

COUNCIL MEETING

October 30, 2017

BAY AREA
AIR QUALITY
MANAGEMENT
DISTRICT

Protecting Refinery Communities from Air Pollution

BAY AREA
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AGENDA: 4

Protecting Refinery Communities from Air Pollution

Advisory Council Meeting
October 30, 2017

Greg Nudd, Acting Rules and Strategic Policy Officer
Office of Rules and Strategic Policy

Overview

- Three-pronged approach:
  - Direct regulation of criteria pollutants (20% by 2020)
  - Capping risk from toxic air contaminants (Rule 11-18)
  - Monitoring crude oil inputs and refinery air pollution (Rule 12-15)
- Next steps
  - Risk-based controls on emergency diesel generators
  - Risk values for fine particulate matter
  - Assembly Bill 617 impacts:
    - Best Available Retrofit Control Technology Review
    - Community Emission Reduction Plans

Direct Regulation of Criteria Pollutants

BARCT Rules Impacting Refineries

Number	Source Category	Pollutant	Estimated Reductions [tons/year]	Date Approved
Rule 6-5	Fluid Catalytic Cracking Units	$PM_{2.5}$	223	Dec. 2016
Rule 8-18	Equipment Leaks	ROG	1,228	Dec. 2016
Rule 11-10	Cooling Towers	ROG	861	Dec. 2016
Rule 9-14	Coke Calcining	SO_2	430	Apr. 2017
Rule 9-1	*Refinery Fuel Gas Sulfur Limits*	SO_2	*245*	*Q1 2018*
Rule 9-1	*Gas Turbines*	NO_X	*211-277*	*Q2 2018*

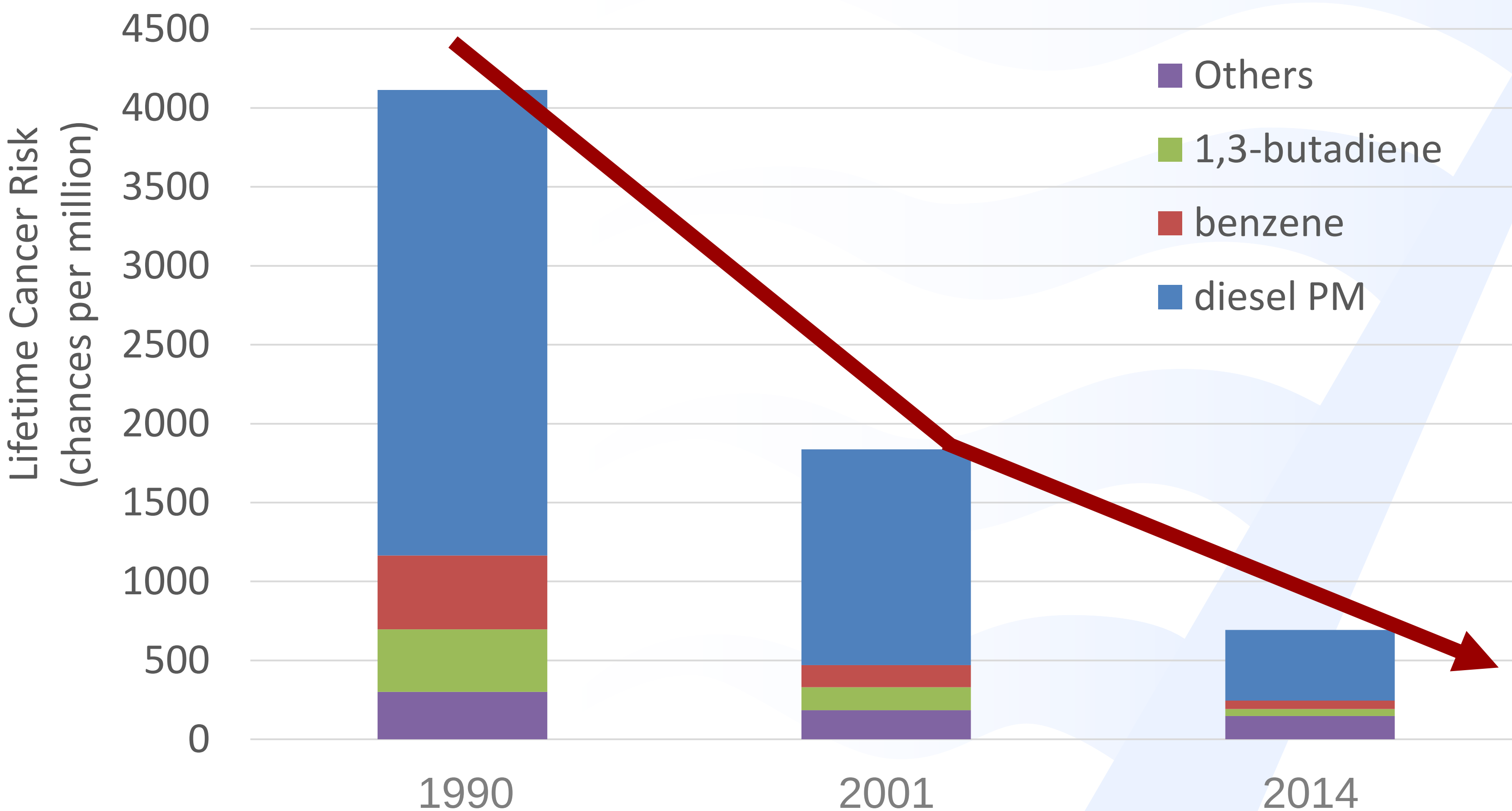
Direct Regulation of Criteria Pollutants

<u>Next Steps</u>

- Follow-up rulemaking for Fluid Catalytic Cracking Units (Rule 6-5):
  - Reviewing results of optimization studies to see if $PM_{2.5}$ can be substantially reduced by operational changes
  - Additional controls may be needed to further reduce $PM_{2.5}$ and SO_2 from these sources
- Assembly Bill 617
  - Control technology review of thousands of sources in the refining sector (roughly 1/3 of the sources being reviewed)
  - May require additional reductions to meet BARCT or community emission reduction requirements

Capping Risk from Toxic Air Contaminants

Bay Area risk levels decline since 1990



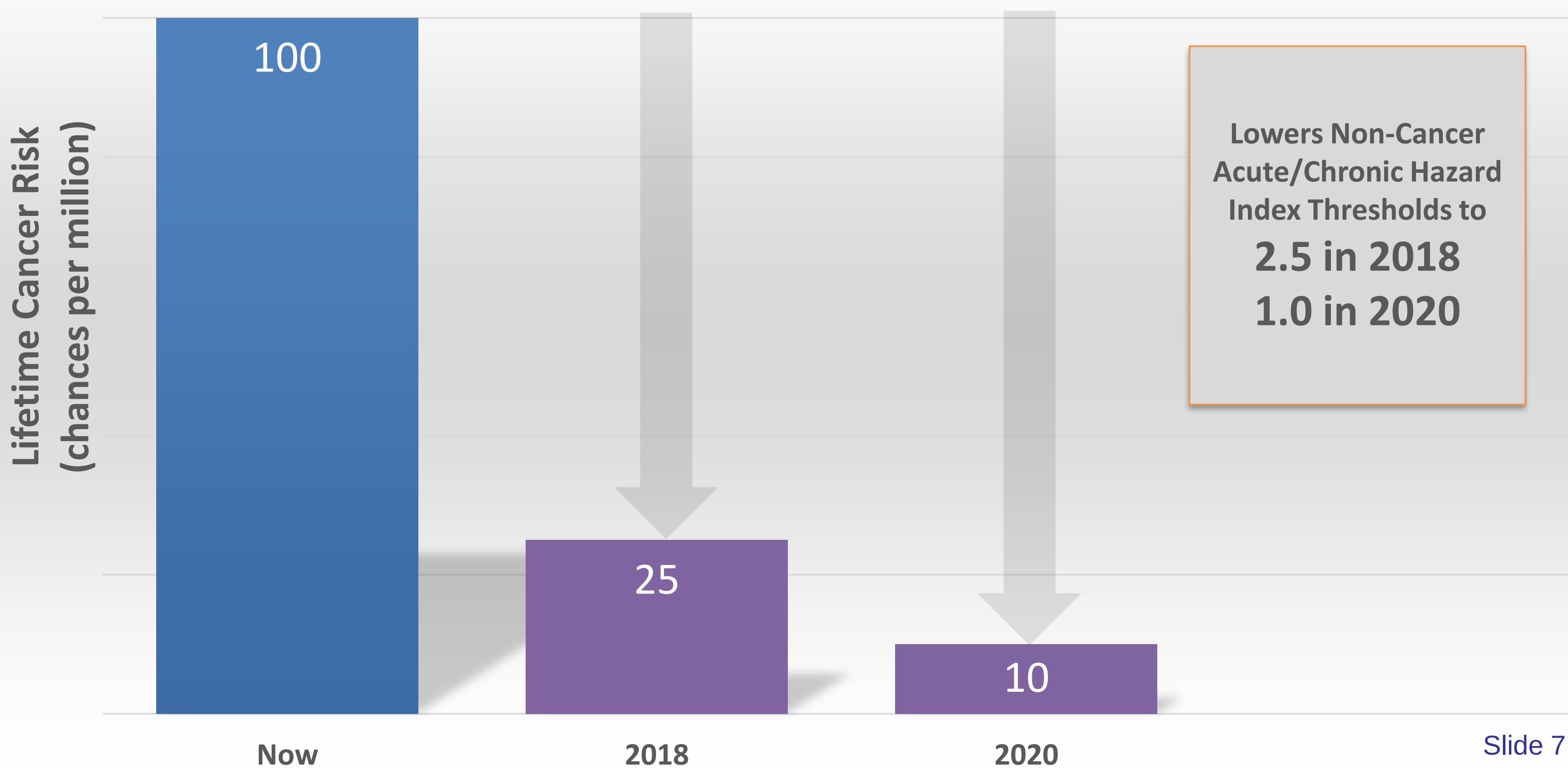
Capping Risk from Toxic Air Contaminants

Rule 11-18

- Reduces Risks from TACs to the lowest achievable levels
- Particularly benefits CARE areas
- Incorporates latest health risk methodologies
- Promotes continuous improvement
- Ensures public transparency
- Provides greater flexibility

Capping Risk from Toxic Air Contaminants

Rule 11-18: Risk Action Thresholds



Capping Risk from Toxic Air Contaminants

- **Air District staff will conduct health risk assessments**
- **Facilities above Risk Action Levels must:**
  - Develop a risk reduction plan for Air District approval
  - Plan must get down below 2020 Risk Action Levels (10/million)
  - Execute plan according to plan schedule
- **Risk reduction measures include:**
  - Installation of **B**est **A**vailable **R**etrofit **C**ontrol **T**echnologies for **T**oxics (TBARCT)
  - Modification of operating hours and activity levels
  - Modification of emissions point characteristics

Capping Risk from Toxic Air Contaminants

Next Steps

- Board consideration of proposed rule Nov. 15, 2017
- Implement Rule 11-18 starting Jan. 2018
  - Highest emitting facilities first (included refineries)
- Develop rule for backup generators at lower risk facilities
- Assembly Bill 617
  - Expanded community monitoring
  - May require additional reductions to meet community emission reduction requirements

Monitoring Refinery Inputs and Air Pollution

Rule 12-15

- Approved April 2016
- Monitors crude slate changes and refinery emissions
- Rule requirements:
  - Reporting of crude oil properties for baseline period and every month
  - Improved emissions inventories
  - Fence-line monitoring systems

Monitoring Refinery Inputs and Air Pollution

Rule 12-15: Crude Slate Data

- Air District engineers have reviewed crude slate data from 2013-2016
- Initial findings:
  - Heavier crude oil is correlated with higher CO_2 emissions, but not strongly correlated
  - Some refinery crude slates are consistent, others are highly variable
- Ongoing monthly reports will detect significant changes in key crude properties
- Canadian crude imports are not significant in the Bay Area
  - Historically less than 2% of crude input
  - Air District staff will continue to coordinate with the California Energy Commission to monitor this issue

Monitoring Refinery Inputs and Air Pollution

Rule 12-15: Fence-Line Monitoring

- Important for monitoring toxic emissions
  - Benzene is a key driver of toxic risk in refinery communities.
  - Benzene usually comes from equipment leaks or tanks, these emissions can't be directly measured at the source
  - Monitoring confirms accuracy of emissions calculations, and identifies significant unknown leaks
- Complements community monitoring
  - Community monitors measure pollution from a mix of sources (refineries, cars, trucks, ships, cooking)
  - Fence-line monitoring directly address refinery pollution

Recap: Three-Pronged Approach

Direct regulation of criteria pollutants

- On track for 20% reduction
- More regulations for further reductions in development

Capping risk from toxic air contaminants (proposed Rule 11-18)

- Risk reduced to lowest feasible level
- Start with refineries and other high emitting facilities

Monitoring crude oil inputs and refinery air pollution (Rule 12-15)

- Ongoing review of crude oil characteristics
- Better emissions estimates
- Fence-line monitoring to verify emissions and find leaks
- Regular reports to Board on crude slate changes and impact on emissions

Next Steps

- Two more criteria pollutant rules in 2018
- Possible further controls on Fluid Catalytic Cracking Units
- Work with Office of Environmental Health Hazard Assessment (OEHHA) on risk values for fine particulate matter
  - Incorporate into Rule 11-18 or
  - Develop $PM_{2.5}$ risk cap rule
- AB 617 Requirements:
  - Expanded BARCT review
  - Enhanced Community Monitoring
  - Community Emission Reduction Plans

Air Monitoring and Attainment Status of Criteria Pollutant Standards in the San Francisco Bay Area

BAY AREA AIR QUALITY MANAGEMENT DISTRICT

AGENDA: 5

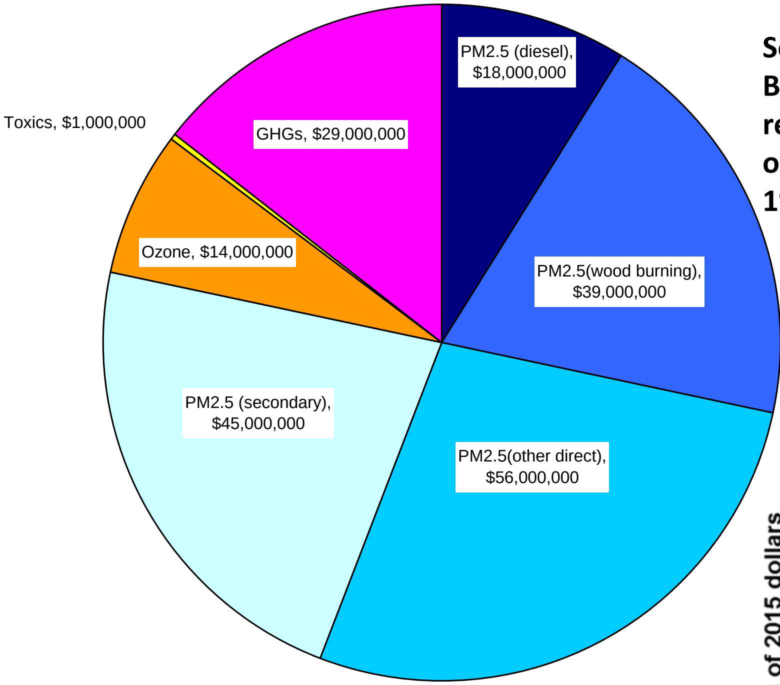
Air Monitoring and Attainment Status of Criteria Pollutant Standards in the San Francisco Bay Area

Eric Stevenson
Director of Meteorology and Measurement

Advisory Council Meeting
October 30, 2017

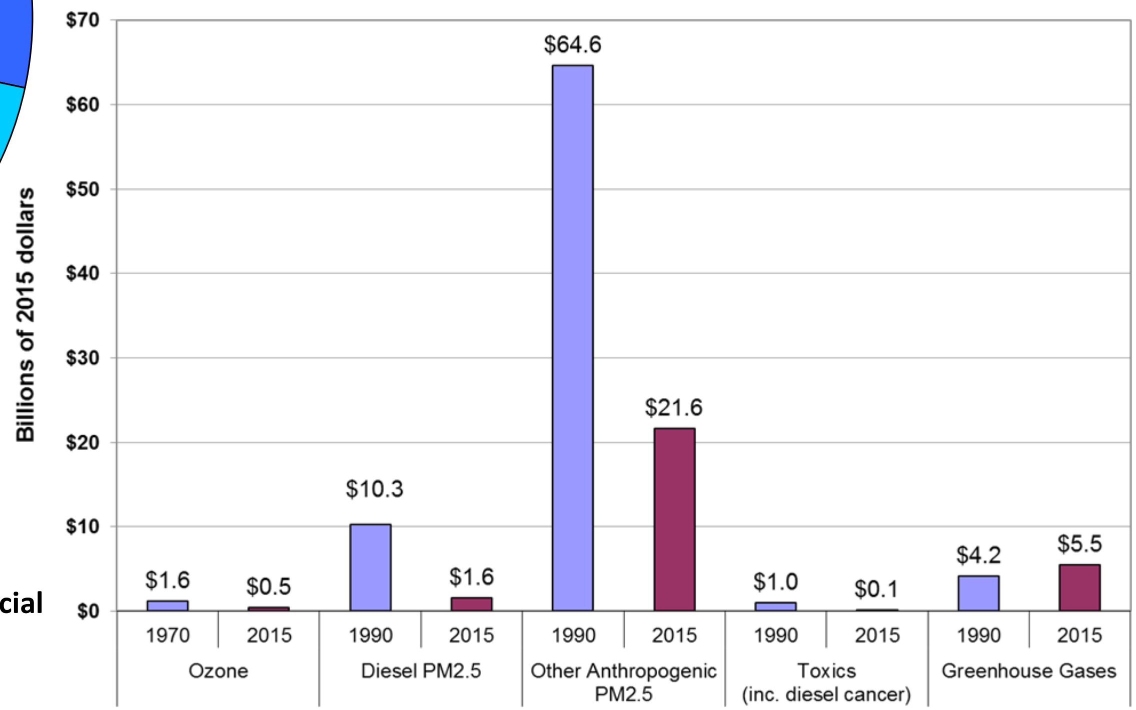
Pollutant	Constituents/ Precursors	Key Anthropogenic Sources	Scale of Impact	Peak Levels	Health Impacts	Other Impacts
Ozone	Reactive Organic Gases (ROG)	Mobile sources (cars) Evaporation of petroleum & solvents Consumer products	Regional & beyond	Summer	Aggravated asthma Acute bronchitis Chronic bronchitis Respiratory symptoms Decreased lung function Heart attacks Premature mortality	**Property damage: tires, paints, building surfaces** **Damage to crops** **Nitrogen deposition to land & waterways**
	NOx	Mobile sources (cars & trucks) Other combustion				
$PM_{2.5}$	Direct emissions from combustion	Wood-burning Diesel engines Gasoline engines Burning natural gas Commercial cooking	Local & Regional	Winter	Aggravated asthma Respiratory symptoms Increase blood pressure Decreased lung function Heart disease Stroke Premature mortality	**Regional haze** **Acid deposition** **Water pollution**
	ROG	See ROG above				
	NOx	See NOx above				
	Ammonia (NH_3)	Landfills, livestock, wastewater treatment, refineries				
	SO_2	Petroleum refining, Ships				
Air Toxics	Diesel PM Benzene 1,3 Butadiene Formaldehyde Acetaldehyde	Diesel engines Gasoline engines Construction equipment Ships & boats Fuel production & Storage	Local	Year-Round	Acute non-cancer Chronic non-cancer Lung cancer Leukemia Premature mortality	**Water pollution**
Green House Gases	**Carbon dioxide (CO_2) Methane (CH_4) Nitrous oxide (N_2O) Hydroflourocarbons Perflourocarbons Sulfur hexafluoride**	**Fossil fuel combustion Mobile sources Industrial/commercial Electricity generation**	**Global**	**Year-Round**	**Potentially increase ozone levels Disease vectors Effects from prolonged heat waves**	**Climate change Rising sea levels Acidification of oceans Species extinction Drought Wildfires**

Bay Area Health Impacts



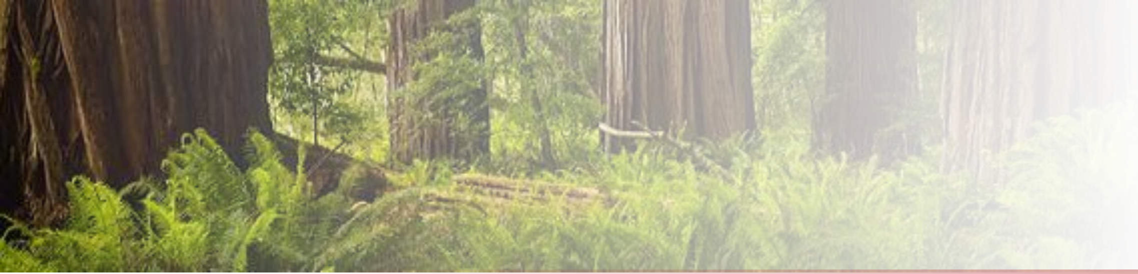
Source: *Bay Area 2010 Clean Air Plan*

Social benefits of a 1% reduction of air pollutants in the Bay Area. The estimated social benefits are based on reductions of 1% of anthropogenic emissions, except for ozone. For ozone, the estimated benefit is based on a 1% reduction in exposures above 0.50 ppm.



Estimated current annual health and other social costs of Bay Area air pollution: prior years compared with 2015

Source: *Bay Area 2017 Clean Air Plan*



Clean Air Act Requirements

State and National Ambient Air Quality Standards (AAQS) are set to protect human health and the environment.

- Primary National and State standards are set to protect public health, including “sensitive” populations.
- Secondary National standards are set to protect public welfare, including protection against visibility impairment, damage to animals, crops, vegetation and buildings
- Monitoring requirements for both siting and measurement have been developed to ensure comparability and accuracy

Pollutants Impacting Bay Area Air Quality

- Ozone (O_3)
  - Summer (May through Sep)
  - O_3 Standard is 0.070 ppm (8 hour average)
    - California has a 1-hour standard of 0.09 ppm
- Particulate Matter ($PM_{2.5}$)
  - Winter (Nov through Feb)
  - $PM_{2.5}$ standard is 35 µg/m^3 (24 hour average)
    - California currently does not have a 24-hour standard
    - There is no hourly standard for $PM_{2.5}$

National and State Standards

Pollutant	National Standard	National Secondary Standard	State Standard	Averaging Time
Carbon Monoxide	9 ppm	None	9.0 ppm	8-hour
	35 ppm	None	20 ppm	1-hour
Lead			1.5 µg/m³	30 Day Average
	0.15 µg/m³	Same as Primary		Rolling 3-Month Average
Nitrogen Dioxide	53 ppb	Same as Primary	0.030 ppm	Annual (Arithmetic Average)
	100 ppb	None	0.18 ppm	1-hour
Sulfur Dioxide	None	None	0.04 ppm	24-hour
		0.5 ppm		3-hour
	75 ppb	None	0.25 ppm	1-hour
Particulate Matter (PM_{10})	150 µg/m³	Same as Primary	50 µg/m³	24-hour
			20 µg/m³	Annual (Arithmetic Average)
Particulate Matter ($PM_{2.5}$)	12.0 µg/m³	15.0 µg/m³	12 µg/m³	Annual (Arithmetic Average)
	35 µg/m³	Same as Primary		24-hour
Ozone	0.070 ppm **	Same as Primary	0.070 ppm	8-hour
			0.09 ppm	1-hour

** Current Ozone NAAQS of 0.070 is being reviewed – Older 0.075 ppm standard may be reinstated
There are additional State Standards for visibility, sulfates, hydrogen sulfide and vinyl chloride

Form of the Standards

National Ozone

- 4^{th} highest 8-hour concentration for the year, averaged over three years is $\leq$ 0.070 parts per million (ppm)

National PM_{10}

- Expected number of days per calendar year with a 24-hour concentration above 150 $\mu g/m^3$ $\leq$ one

National $PM_{2.5}$

- 98% of the daily concentrations, averaged over three years are $\leq$ 35 $\mu g/m^3$, Annual mean averaged over 3 years

State standards are values that are not to be exceeded

Attainment Status

	State Standard	Federal Standard
Ozone	Non Attainment	Non Attainment*
PM_{10}	Non Attainment	Unclassified
$PM_{2.5}$	Attainment (Annual Average)	Non Attainment** (24-hour Average)

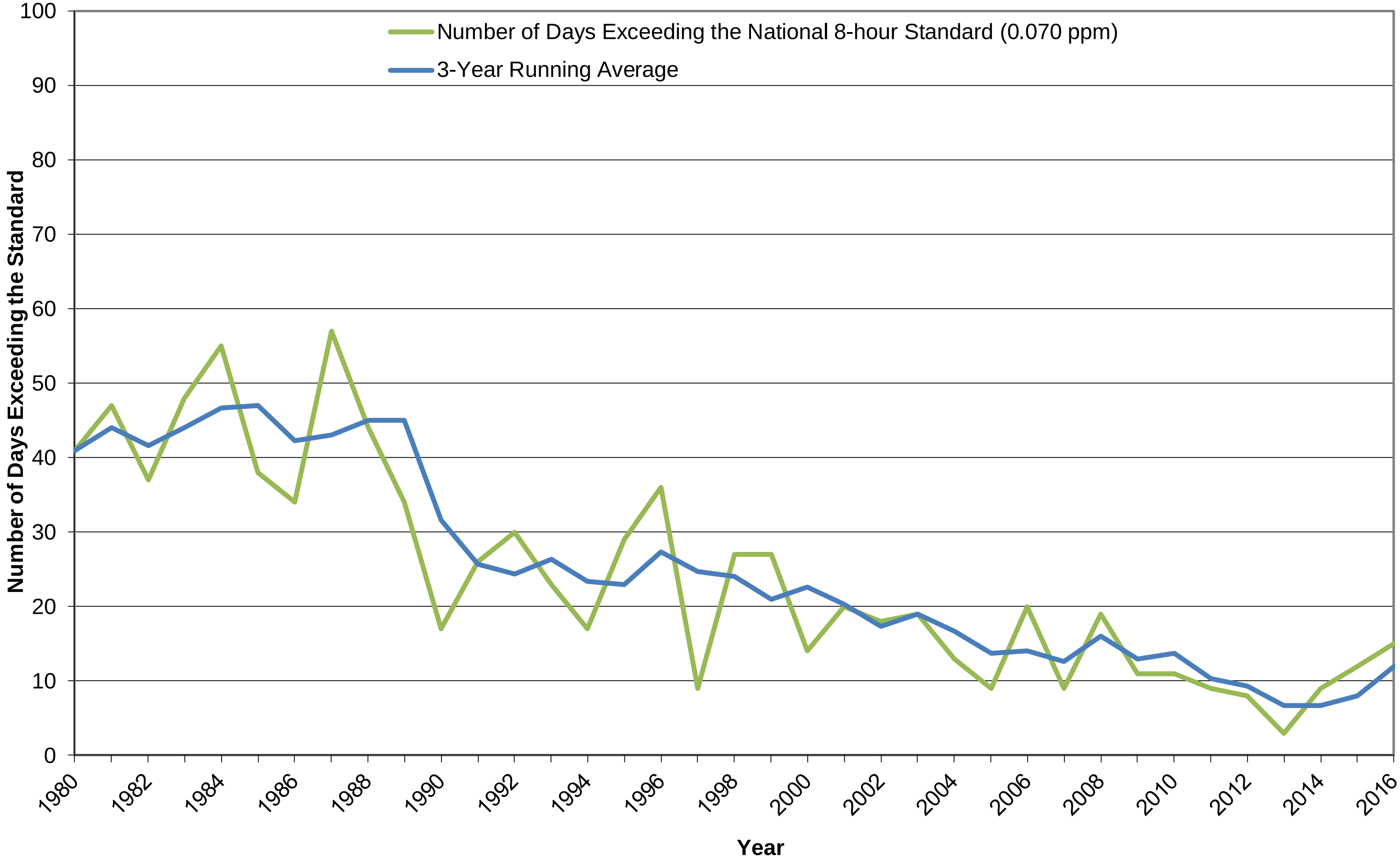
* The current design value for ozone is 0.074 ppm (2016)

** The EPA has recognized the Bay Area has a "Clean Data" for $PM_{2.5}$ – meaning the data confirm that the standard has been reached

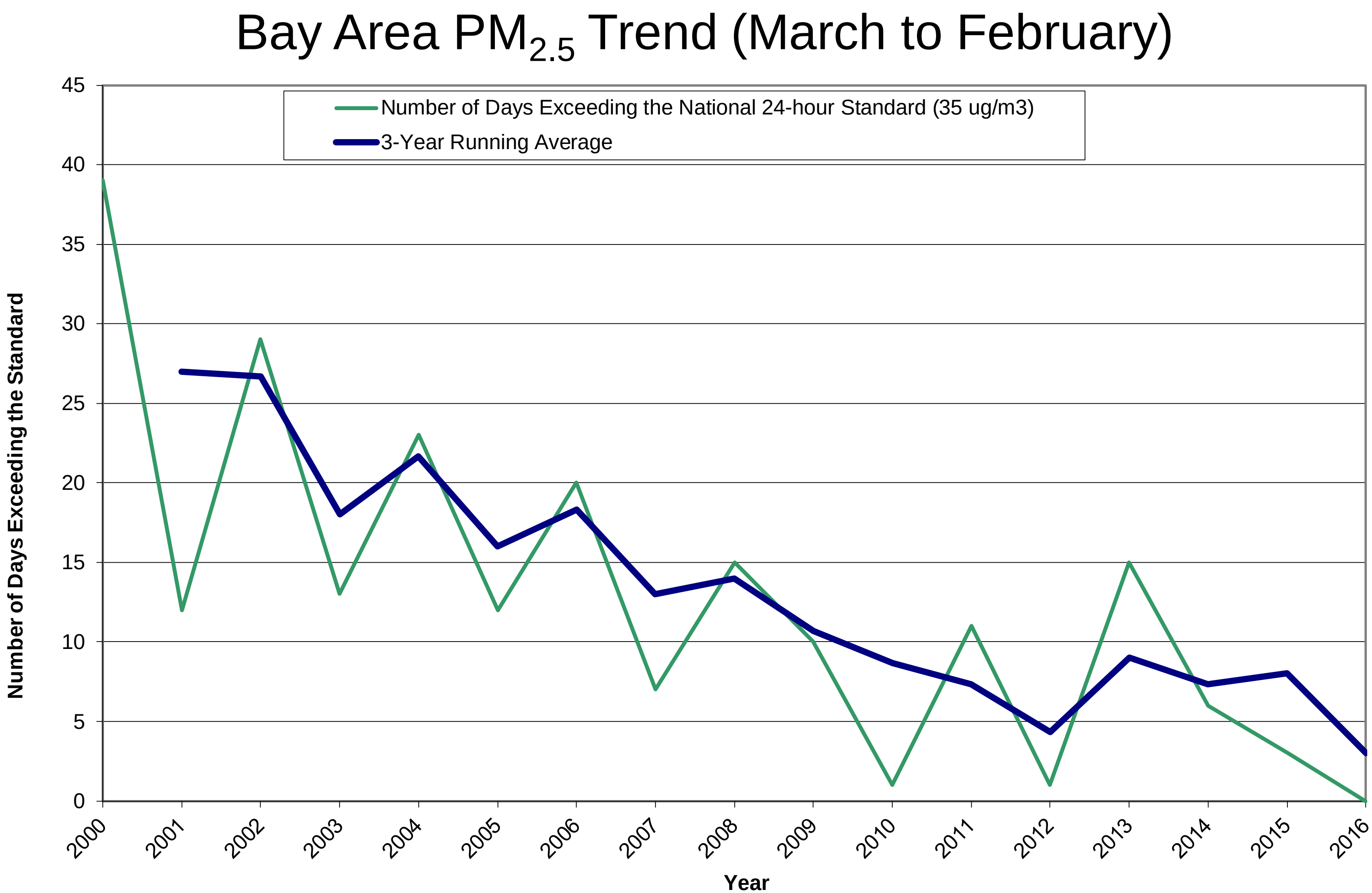
Air Monitoring Network

Ozone Exceedance Trends

Bay Area Ozone Trend



$PM_{2.5}$ Exceedance Trends



Changes Needed for AB 617

Community monitoring is required

- Commence monitoring in identified areas by July 2019
- Pivot from NAAQS focused network to a network more focused on localized impacts (while maintaining NAAQS compliance

What will this entail?

- Effective screening of localized impacts
- More permanent monitoring to determine trends

Staff is investigating methodologies/equipment that will aid in more localized monitoring

Advisory Council Next Area of Focus: Beyond Attainment – Prioritization for Local Health Impacts

Beyond Attainment: Prioritization for Local Health Impacts

AGENDA: 6

- The Bay Area is near attainment for Criteria Pollutant Standards
- Characterize Local Exposures
- Understand Health Impacts of Local Exposures
- Prioritize Actions based on Health Impacts
- Local Particulate Matter is at the Nexus of:
  - Probable Serious Impact, but
  - Much to Learn about Mechanisms

Introduction to the OEHHA in Anticipation of Future Discussion Regarding Particulate Matter Health Effects

OEHHA Role at CalEPA

- OEHHA consists primarily of toxicologists, epidemiologists, and related health and environmental scientists
- Primary mandates related to assessing hazard and risk of environmental chemical exposure
- Legislative mandates across the environmental media spectrum

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- Legislative mandates across the environmental media spectrum

Mandates Related to Air Pollution

- Make health-based recommendation for the level of the California Ambient Air Quality Standards
- Conduct epidemiological studies to ascertain health effects of air pollutants and determine sensitive subpopulations
- Health effects assessments of Toxic Air Contaminants
- Risk Assessment Guidelines under the Air Toxics Hot Spots Act

Presentations Today

- Dr. John Budroe, Chief, Air Toxicology and Risk Assessment Section
- Dr. Keita Ebisu, Research Scientist, Air and Climate Epidemiology Section

Air Toxics Hot Spots Program

- Stationary sources in CA are required to report emissions of specified chemicals to ARB and Air Districts
- Facilities are prioritized by Districts (high, medium, low concern)
  - Based on emissions estimates, distance to nearest receptor, information on potency of emitted toxicants
- High concern facilities must conduct risk assessments to estimate public health impacts to surrounding populations from facility emissions

Hot Spots: OEHHA's role

- OEHHA is required by statute to develop the Guidelines used to conduct Hot Spots risk assessments
- Guidelines Technical Support Documents:
  - Acute Reference Exposure Level (REL)
  - Chronic REL
  - Cancer
  - Exposure Assessment and Stochastic Analysis

Hot Spots: OEHHA's role

- Guidance Manual for Preparation of Health Risk Assessments – application of information in TSDs to facility health risk assessments (HRAs)
- All Hot Spots documents receive public comment, and external peer review by the Scientific Review Panel (SRP)

Noncancer Reference Exposure Levels

- RELs: Usually based on adult animal toxicology or human epidemiology (worker) data.

- REL:

$$\frac{\text{NOAEL or BMDL}}{\text{UF}}$$

Noncancer Reference Exposure Levels

- RELs: chemical air concentrations not expected to produce adverse health effects
- Acute RELs: meant to protect against infrequent 1-hour exposures
- 8-hour, chronic RELs: meant to protect against 8-hour or 24-hour (respectively) long-term exposures. Eight-hour RELs meant to protect off-site workers, students, teachers, day-care centers

Cancer Slope Factors

- Tumor dose-response data is fit to model (usually Multistage-Cancer) using Benchmark Dose Software
- For "genotoxic" carcinogens, data support the assumption of low-dose linearity.
- For carcinogens of unknown mechanism, low-dose linearity is assumed as a policy default.
- For both these cases, potency slope is estimated by linear extrapolation from the point of departure to zero.

Toxic Air Contaminant Program

- Air Resources Board (ARB) and OEHHA determine if a substance should be identified as a Toxic Air Contaminant (TAC)
- ARB and OEHHA staff draft a TAC document that serves as the basis for identification
- ARB staff assess the potential for human exposure
- OEHHA staff evaluates the health effects – RELs and/or cancer inhalation unit risks are developed where appropriate

Toxic Air Contaminant Program

- Draft TAC receives public comment.
- Draft TAC document is peer-reviewed by the SRP
- The SRP approves the document and submits scientific findings to the ARB board
- ARB staff prepare a hearing notice and a draft regulation to formally identify the substance as a TAC

Toxic Air Contaminant Program

- The ARB Board then votes whether to identify a substance as a TAC
- TAC listing triggers ARB development of Airborne Toxics Control Measures (ATCMs) to reduce risk if necessary

Characterizing Particulate Matter (PM) Dose-Response with Epidemiology Study Data

- Overview of association between $PM_{2.5}$ and health
  - Epidemiological Studies
  - Epidemiological evidence of $PM_{2.5}$ effects on health
  - Meta-Analysis

- Case study
  - Forest fire
  - $PM_{2.5}$ chemical constituents and sources
  - Acute $PM_{2.5}$ exposure and health

Epidemiological Studies
~ $PM_{2.5}$ and Health ~

- Most $PM_{2.5}$ epidemiological studies are observational studies
- Health Outcome
  - Mortality/ ER visit/ hospital admission
    - Cardiovascular related disease
    - Respiratory related disease
  - Reproductive outcomes
    - Low birth weight
    - Preterm birth
- Exposure Period
  - Short-term effect
    - Same day - previous 1 week exposure
  - Long-term
    - Annual exposure
- Relationship
  - Linear
  - Non-linear
- Advantages and Disadvantages
  - Advantages
    - Cover a wide range of people and areas
    - Identify susceptible population
  - Disadvantages
    - Confounding: socio-economic status
    - Association is uncertain outside range of observed level: very low/high $PM_{2.5}$ level

Example: Short-term $PM_{2.5}$ exposure and Cardiovascular Disease

Study (Journal, Year)	Study Area	Study Period	$PM_{2.5}$ Average (SD or IQR)	Health Outcome	**Percent Excess Risk** per 10 μg/m^3 increase (95% Confidence interval)
Bell et al. (AJE, 2010)	U.S. (48 state)	1999 - 2005	12.92 (SD = 2.65)	Cardiovascular Hospital Admission (Same day)	0.80 (0.59, 1.01)
	Northeast U.S.		13.60 (SD = 1.99)		1.08 (0.79, 1.37)
	Northwest U.S.		9.29 (SD = 0.75)		0.74 (-1.74, 3.29)
	Southeast U.S.		12.57 (SD = 2.36)		0.29 (-0.19, 0.77)
	Southwest U.S.		12.70 (SD = 3.95)		0.53 (0.00, 1.05)
Bravo et al. (EHP, 2017)	Eastern U.S.	2002 - 2006	12.28 (N.A.)	Cardiovascular Hospital Admission (Same day)	0.79 (0.62, 0.97)
Ostro et al. (EHP, 2007)	California, U.S.	2000 - 2003	19.28 (IQR = 14.63)	Cardiovascular Mortality (Exposure 3 days before)	1.09 (0.08, 2.11)
Ma et al. (STOTEN, 2011)	Shenyang, China	2006/8 - 2008/12	75.0 (IQR = 47.0)	Cardiovascular Mortality (Average of last 2 days)	0.53 (0.09, 0.97)

* SD: Standard Deviation/ IQR: Interquartile Range (75th percentile - 25th percentile)

- Models assume linear relationship.
- Associations were found same day exposure to exposure 3 days before.
- Per 10 μg/m^3 increase of $PM_{2.5}$, 1.09% (95% Confidence Interval: 0.08, 2.11) increase in risk of cardiovascular-related mortality.

Example: Short-term $PM_{2.5}$ exposure and Respiratory Disease

Study (Journal, Year)	Study Area	Study Period	$PM_{2.5}$ Average (SD or IQR)	Health Outcome	**Percent Excess Risk** per 10 μg/m^3 increase (95% Confidence interval)
	U.S. (48 states)		12.92 (SD = 2.65)		0.41 (0.09, 0.74)
	Northeast U.S.		13.60 (SD = 1.99)		0.28 (-0.17, 0.72)
Bell et al. (AJE 2010)	Northwest U.S.	1999 - 2005	9.29 (SD = 0.75)	Respiratory Hospital Visit (Exposure 2 days before)	0.19 (-2.52, 2.98)
	Southeast U.S.		12.57 (SD = 2.36)		0.35 (-0.44, 1.14)
	Southwest U.S.		12.70 (SD = 3.95)		0.94 (0.22, 1.67)
Bravo et al. (EHP, 2017)	Eastern U.S.	2002 - 2006	12.28 (N.A.)	Respiratory Hospital Admission (Same day)	1.16 (0.88, 1.45)
Ostro et al. (AJE, 2016)	California, U.S.	2005 - 2009	16.5 (IQR = 11.4)	Respiratory ER Visit (Same day)	0.88 (0.18, 1.58)
Ma et al. (STOTEN, 2011)	Shenyang, China	8/2006 – 12/2008	75.0 (IQR = 47.0)	Respiratory Mortality (Average of last 2 days)	0.97 (0.01, 1.94)
Strickland et al. (EHP, 2016)	Georgia, U.S.	1/2002 – 6/2010	N.A. (IQR = 8.12)	Pediatric Asthma ER Visit (Same day)	1.30 (0.30, 2.30)

* SD: Standard Deviation/ IQR: Interquartile Range (75th percentile – 25th percentile)

- Models assume linear relationship.
- Per 10 μg/m^3 increase of $PM_{2.5}$, 0.88% (95% Confidence Interval: 0.18, 1.58) increase in risk of respiratory-related ER visit.

Meta-Analysis

- Meta-Analysis is the process of combining the results from a number of studies for the same association to produce a pooled estimate.
- In the meta-analysis by Atkinson et al. (Thorax 2014), 21 studies across the world were combined.
- A 10 μg/m^3 increment in short-term $PM_{2.5}$ exposure was associated with a 1.51% (95% CI 1.01, 2.01) increase in the risk of respiratory related death.
- Estimates vary by disease and age.

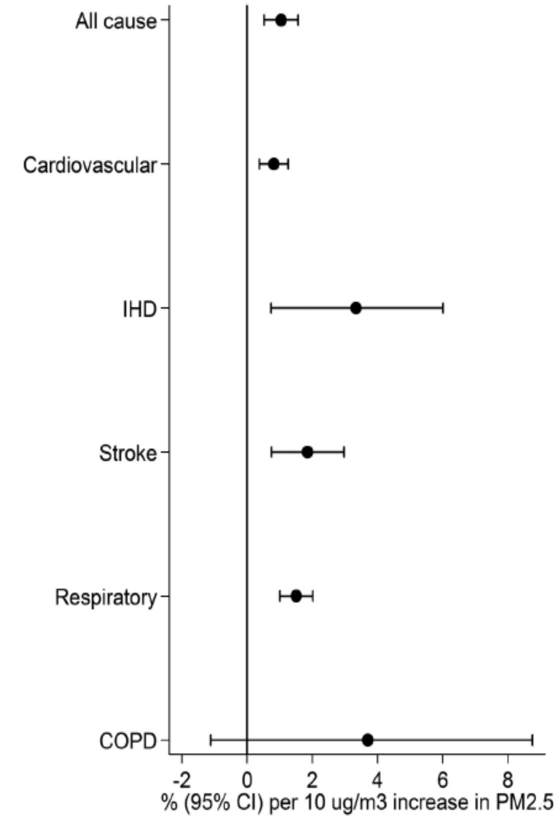


Figure 1 Summary estimates (95% confidence intervals) for all-cause and cause-specific mortality.

Atkinson et al. (Thorax, 2014)

$PM_{2.5}$ from Wildfire and Health Outcomes

- $PM_{2.5}$ levels during wildfires are extremely high.
- On days $PM_{2.5}$ > 35 μg/m^3, 71.3% of total $PM_{2.5}$ could be attributed to wildfires.

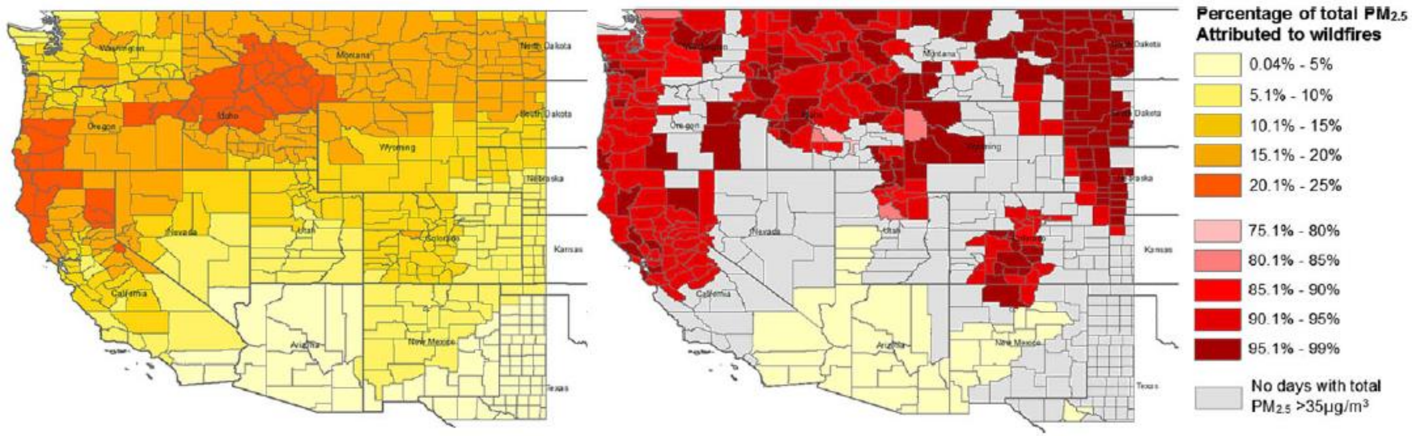


Fig. 1 Fraction of $PM_{2.5}$ attributable to wildfires by county during fire seasons (May-October) in the present day (2004–2009), on all days (left panel), and on the subset of days that had total $PM_{2.5}$ > 35 μg/m^3 (The National Ambient Air Quality Standards (NAAQS) threshold; right panel)

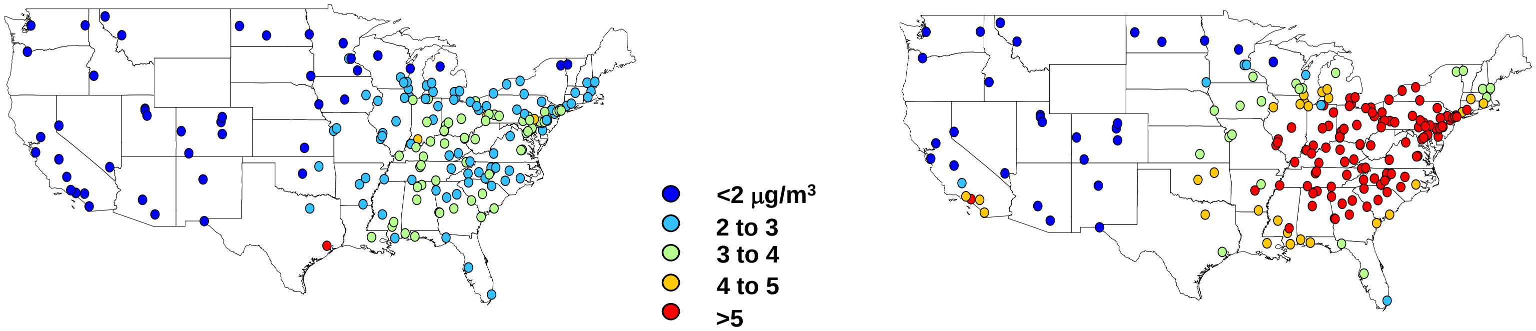
Liu et al. (Climatic Change, 2016)

- Relationship is uncertain outside usual level.

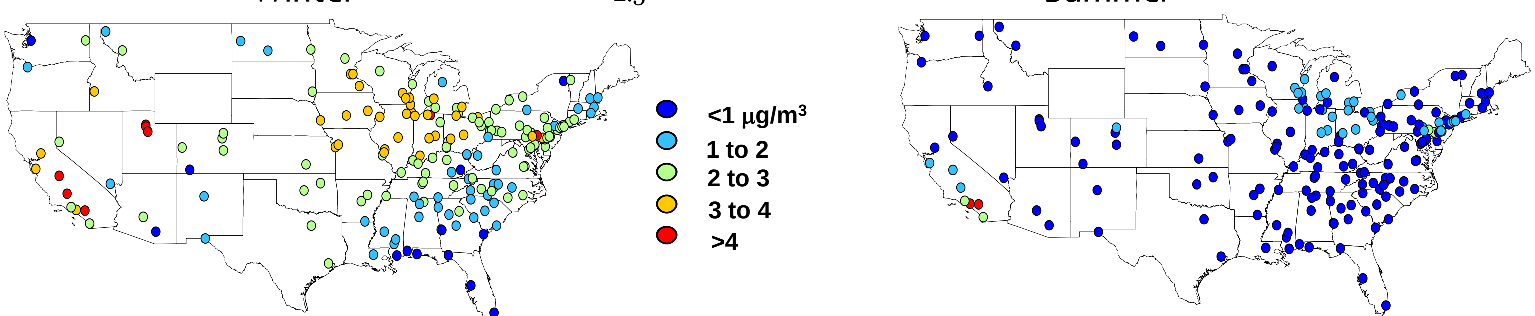
$PM_{2.5}$ Chemical Constituents and Sources

- $PM_{2.5}$ consists of many chemical constituents.

Winter $PM_{2.5}$ sulfate Summer



Winter $PM_{2.5}$ nitrate Summer



Bell et al. (*Environmental Health Perspectives*, 2007)

Where are $PM_{2.5}$ chemical constituents coming from?

- $PM_{2.5}$ EC: Biomass Burning, Vehicular Emissions etc.
- $PM_{2.5}$ Na^+: Sea salt, Biomass burning etc.
- $PM_{2.5}$ NH_4^+: Secondary Ammonium Nitrate, Secondary Ammonium Sulfate etc.
- $PM_{2.5}$ NO_3^- : Vehicular Emissions, Secondary Ammonium Sulfate etc.
- $PM_{2.5}$ OC: Oil Combustion, Vehicular Emissions etc.
- $PM_{2.5}$ $SO_4^=$: Oil Combustion, Secondary Ammonium Sulfate etc.
- $PM_{2.5}$ Si: Resuspended soil, Road Dust etc.

- Most $PM_{2.5}$ chemical constituents come from multiple sources.
- Using source apportionment method, source-specific $PM_{2.5}$ level can be estimated.

Potential Sources of $PM_{2.5}$ Chemical Constituents

- Aged Sea Salt: Na^+, NO_3^-, $SO_4^=$
- Biomass Burning: EC, OC, Na^+
- Oil Combustion: EC, Na^+, OC,
- Road Dust: Al, Si, Zn
- Resuspended Soil: Al, Si, Fe
- Secondary Ammonium Nitrate: NH_4^+, NO_3^- , $SO_4^=$
- Secondary Ammonium Sulfate: NH_4^+, NO_3^-, $SO_4^=$
- Vehicular Emissions: EC, OC, Zn

- Ostro et al. found that exposure to vehicular emissions, EC, and Zn are associated with cardiovascular related ER visits
 (American Journal of Epidemiology 2017)

Acute $PM_{2.5}$ Exposure and Atrial Fibrillation
~ Exposure Timing ~

- Critical time of exposure could be less than 24 hours.
- A study conducted in Boston found that associations between $PM_{2.5}$ and onset of atrial fibrillation were stronger in shorter exposure period.
- Few studies have investigated associations between acute exposure (or peak exposure) and health outcomes.

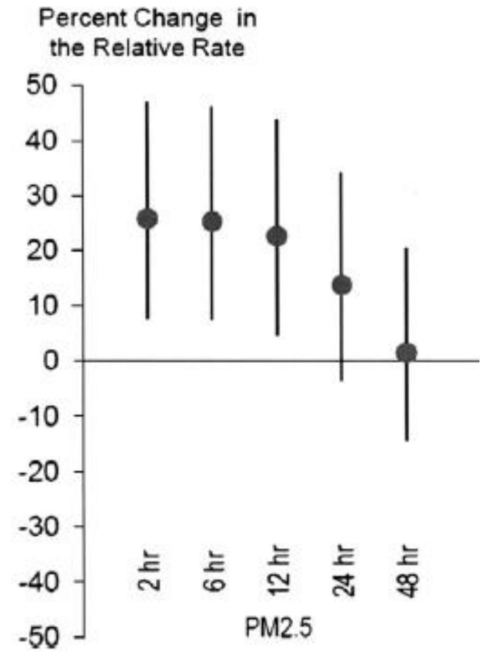


Figure 2 Risk of Atrial Fibrillation With Air Pollution

Link et al. (Journal of the American College of Cardiology, 2013)

Summary

- Many studies indicate associations between $PM_{2.5}$ and adverse health outcomes.
- Several studies focused on $PM_{2.5}$ chemical constituents and/or sources to identify toxic chemical constituents and sources.
- Further studies are warranted for
  - Critical time of exposure (exposure-response relationship) could be more acute
  - Associations outside range of observed level
- $PM_{2.5}$ monitoring of finer spatio-temporal resolutions could help further investigations.

Complexity of Particulate Matter and its Effects on Human Health

Air Pollution and Human Health
Particles are the Heart of the Matter

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Bay Area Air Quality Management District
Advisory Council Meeting
October 30, 2017

1 in 6 deaths, worldwide, is attributable to Pollution

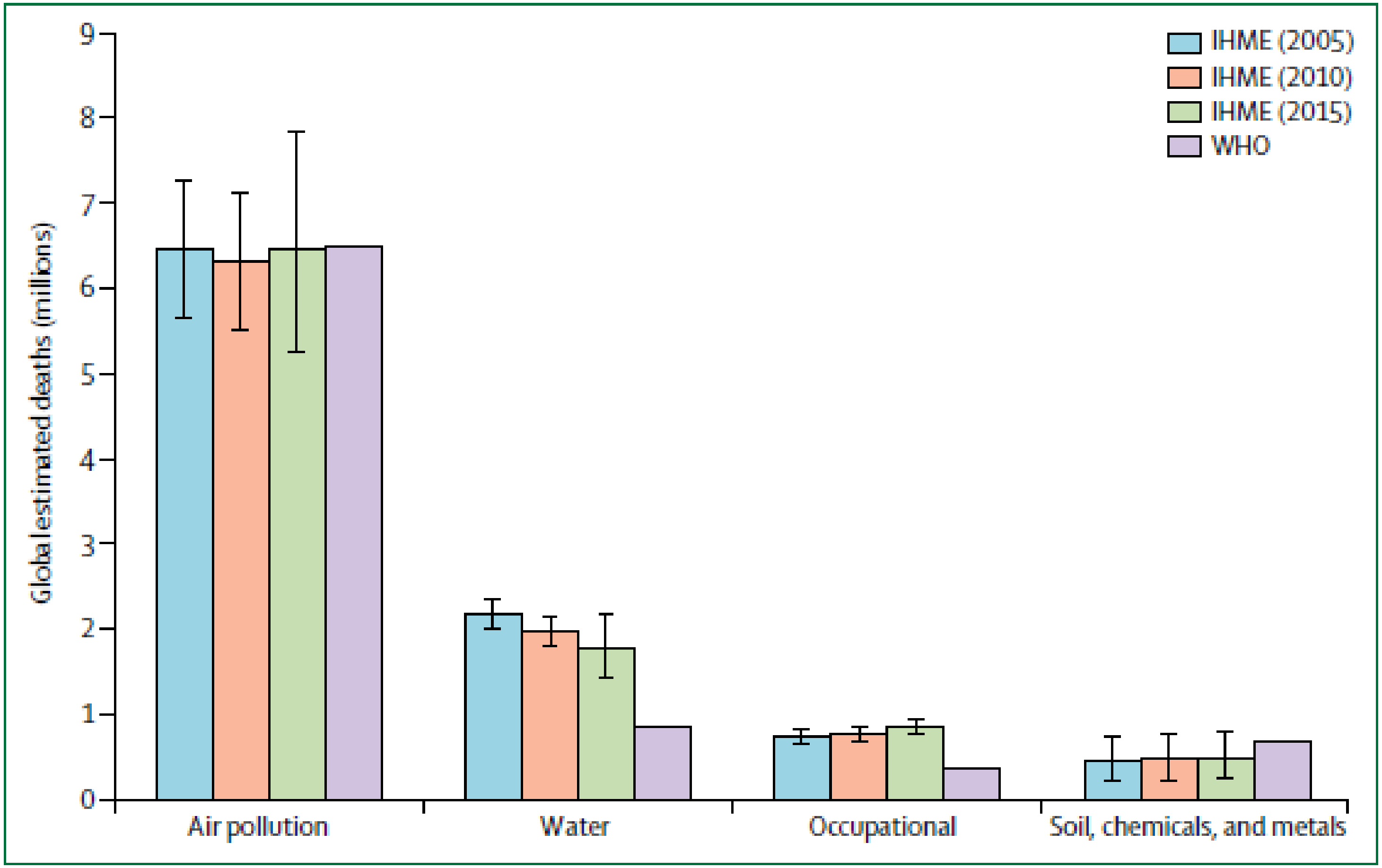


Figure 4: Global estimated deaths (millions) by pollution risk factor, 2005–15
Using data from the GBD study[47] and WHO.[99] IHME=Institute for Health Metrics and Evaluation.

Air Pollution Contributes to Multiple Diseases

The *Lancet* Commission on pollution and health, Lancet, October 2017

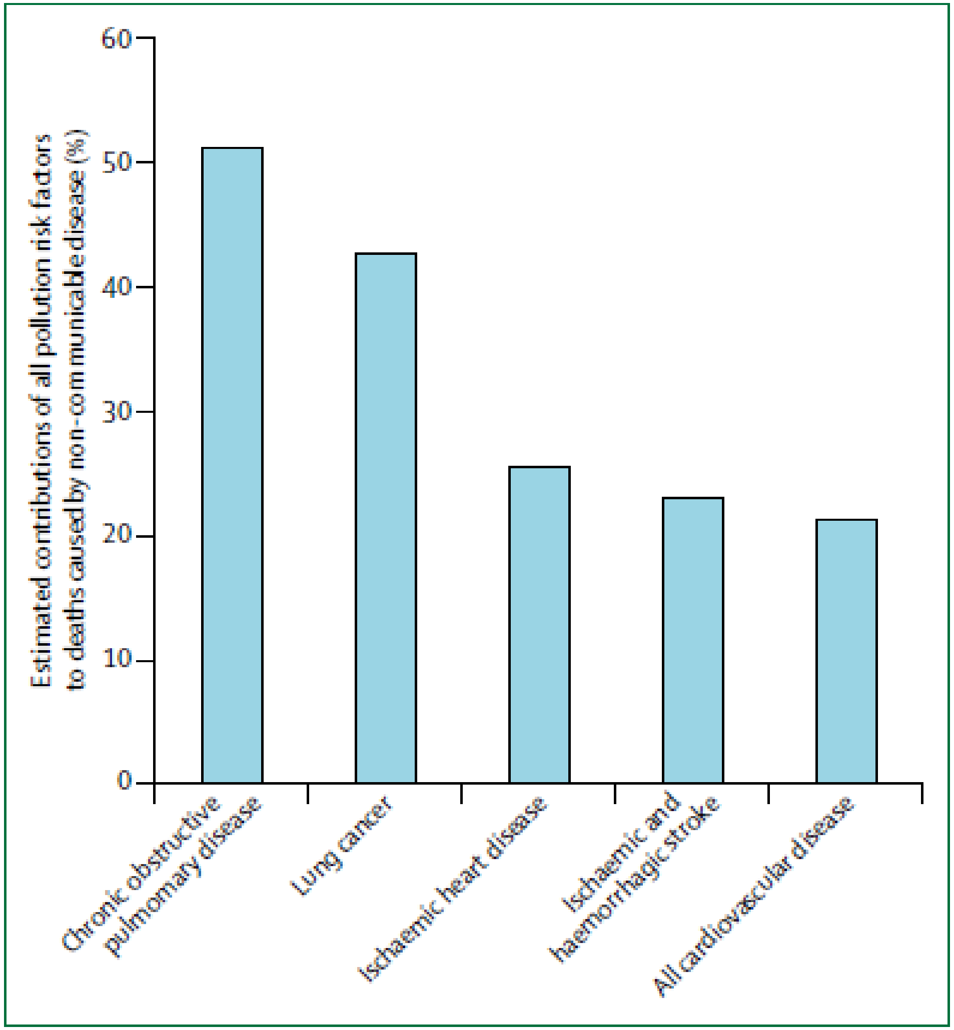


Figure 6: Estimated contributions of all pollution risk factors to deaths caused by non-communicable diseases, 2015
GBD Study, 2016.[47]

Age is a major factor with respect to susceptibility.

The *Lancet* Commission on pollution and health, Lancet

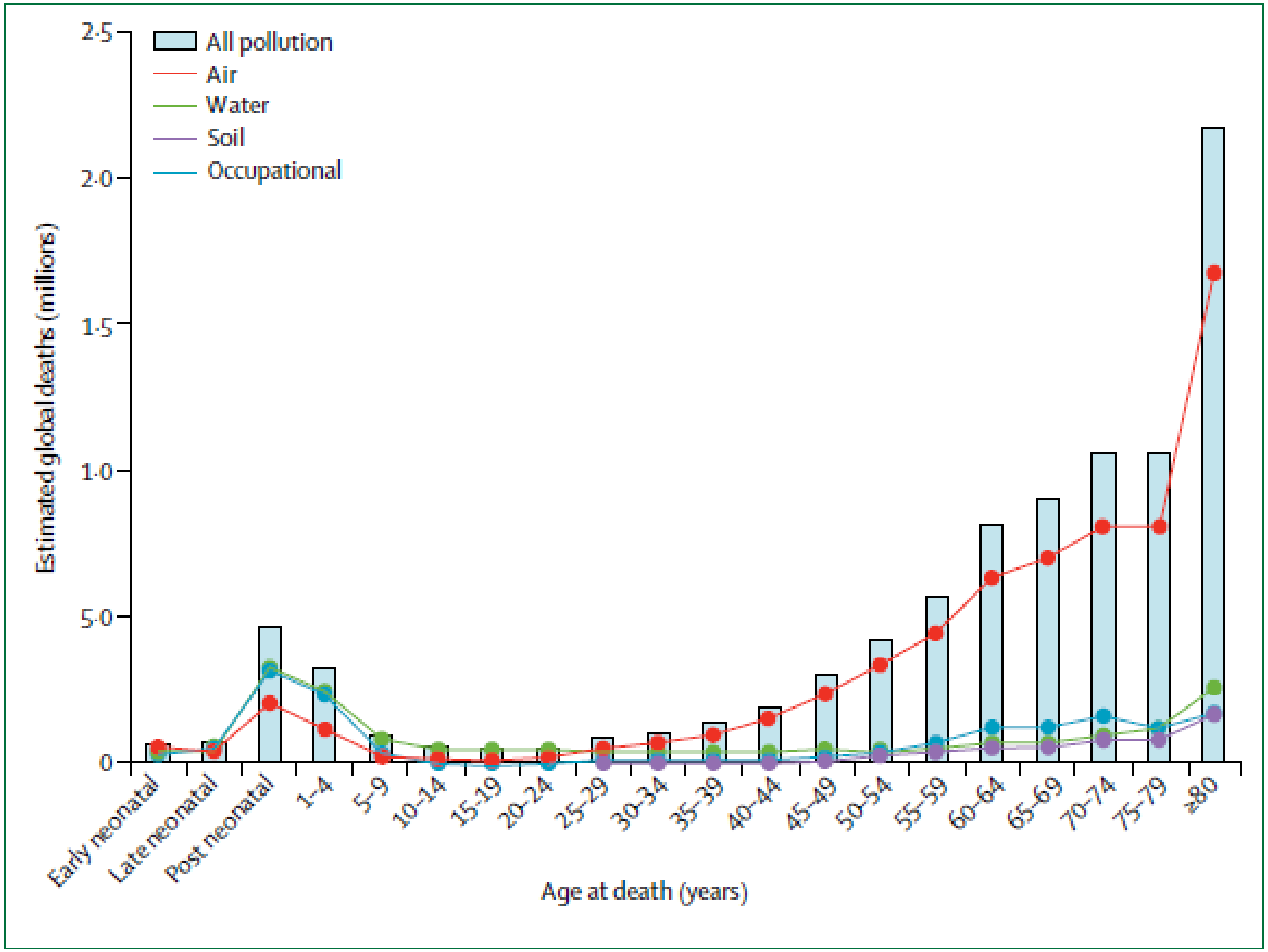


Figure 10: Estimated global deaths by pollution risk factor and age at death, 2015

GBD Study, 2016.[42]

Epidemiological Studies Link Air Pollution to Cardiovascular Disease

- An increase in air pollutants leads to increased mortality and hospital admissions because of cardiovascular diseases (Analitis A. et al. 2006, Zanobetti et al. 2003, Dominici et al. 2006, Peel et al. 2007)
- Exposure to elevated levels of particulate matter (PM) in ambient air leads to an increased heart rate (HR) and a decreased heart rate variability (HRV) in elderly patients (Dubowsky Adar S. et al. 2007, Luttmann-Gibson et al. 2006)
- These associations suggested that controlled studies could help identify causal agents and mechanisms of toxicity.

Ambient Pollutants that are Associated with Adverse Cardiovascular Effects

Particles:
Definition according to the particle size:
PM: Particulate matter, measuring unit: $\mu g/m^3$
PM10: Particles with an aerodynamic diameter ≤ 10µm
PM2.5: Particles with an aerodynamic diameter ≤ 2.5µm
UFP (ultrafine particles): Particles with an aerodynamic diameter between 0.01 und 0.1µm, measuring size: $count/cm^3$

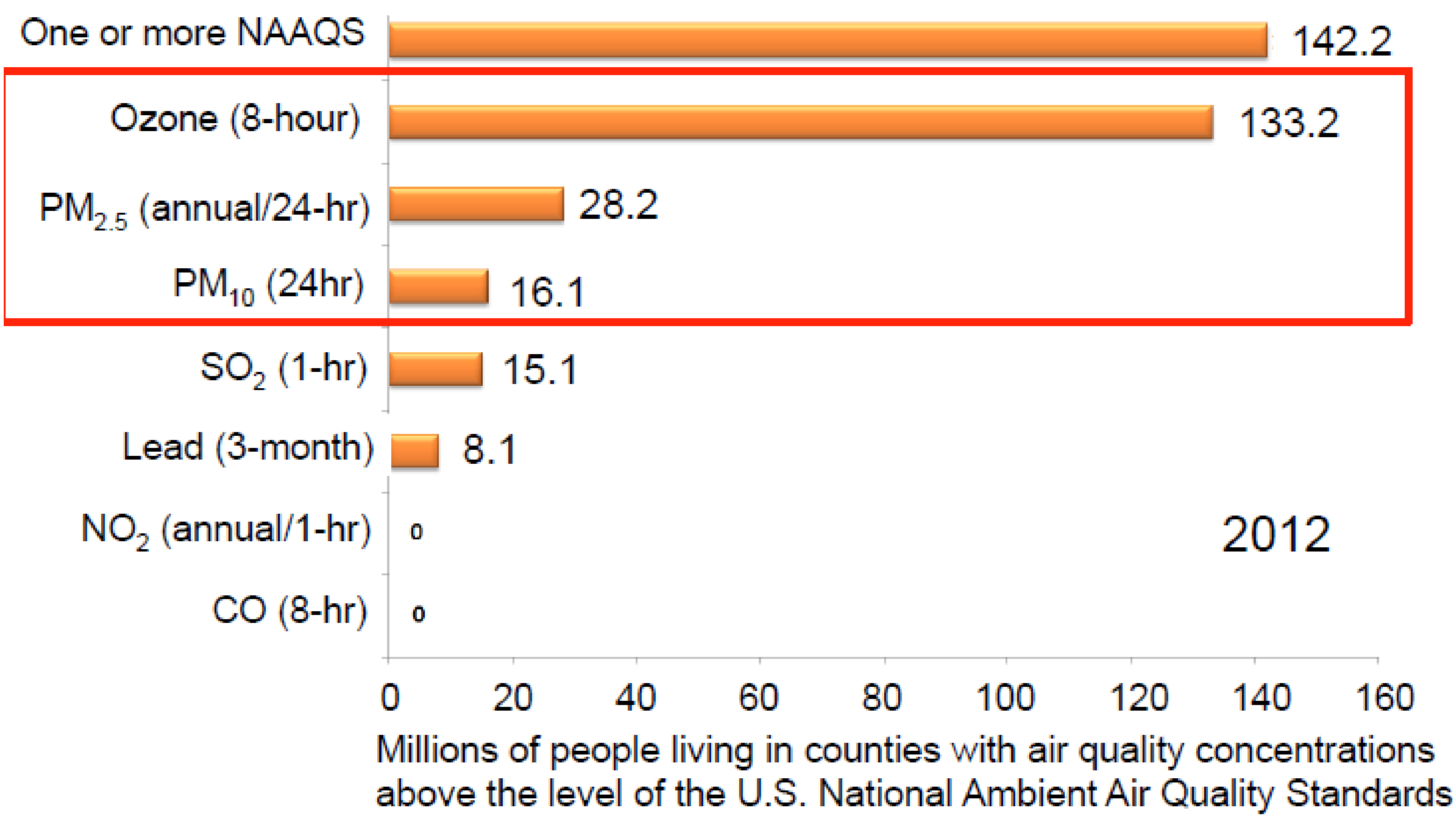
Definition according to chemical composition:
OC, EC: organic and elemental carbon

Gases:
NO, NO2: nitrogen monoxide, nitrogen dioxide
CO: carbon monoxide

What is in Polluted Air?

- Emissions from power plants, motor vehicles, dust.
- Some pollutants are gases:
  - Ozone and NO_2 are major problems in California.
  - SO_2 and organic gases are also important.
- Some pollutants are particles:
  - Particles are associated with increased deaths during air pollution episodes.

Ozone and Particulate Matter (PM) are the top two U.S. air pollutants



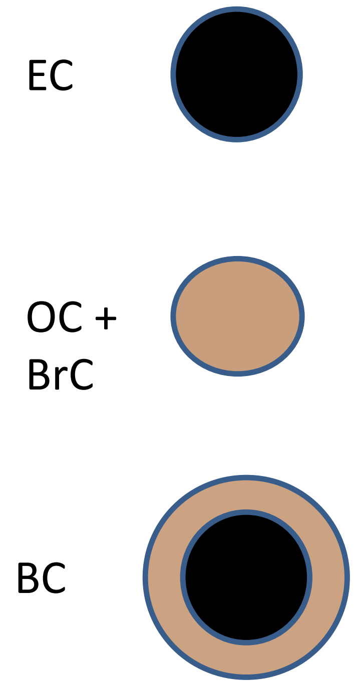
EPA, 2014: http://www.epa.gov/airtrends/aqtrends.html#comparison

The Role of PM Constituents on Health

- Based on epidemiological studies and mechanistic studies, air pollution health effects are more strongly associated with PM and Ozone than with other pollutants, and the bulk of potential monetary benefits can be accrued by mitigation of PM and ozone exposures.
- Our current studies have identified carbon-containing compounds that are components of PM as agents that can impact the heart.

Black Carbon and Brown Carbon?

- Black carbon (BC) is a major component of “soot”, a complex light-absorbing mixture that comprised of a mixture of Elemental Carbon (EC) and Particulate Organic Carbon (OC).
- BC is the most strongly light-absorbing component of particulate matter (PM), and is formed by the incomplete combustion of fossil fuels, biofuels, and biomass.
- BC is emitted directly into the atmosphere in the form of fine particles ($PM_{2.5}$) and ultrafine particles ($PM_{0.1}$). These are also considered nanoparticles.
- BC is the most effective form of PM, by mass, at absorbing solar energy: per unit of mass in the atmosphere, BC can absorb a million times more energy than carbon dioxide (CO_2).
- Organic carbon aerosols are a significant absorber of solar radiation. The absorbing part of organic aerosols is referred to as "brown" carbon (BrC).



http://www.epa.gov/blackcarbon/basic.html

MOST Black Carbon and other contaminants are produced during FOSSILE FUEL OR BIOMASS COMBUSTION

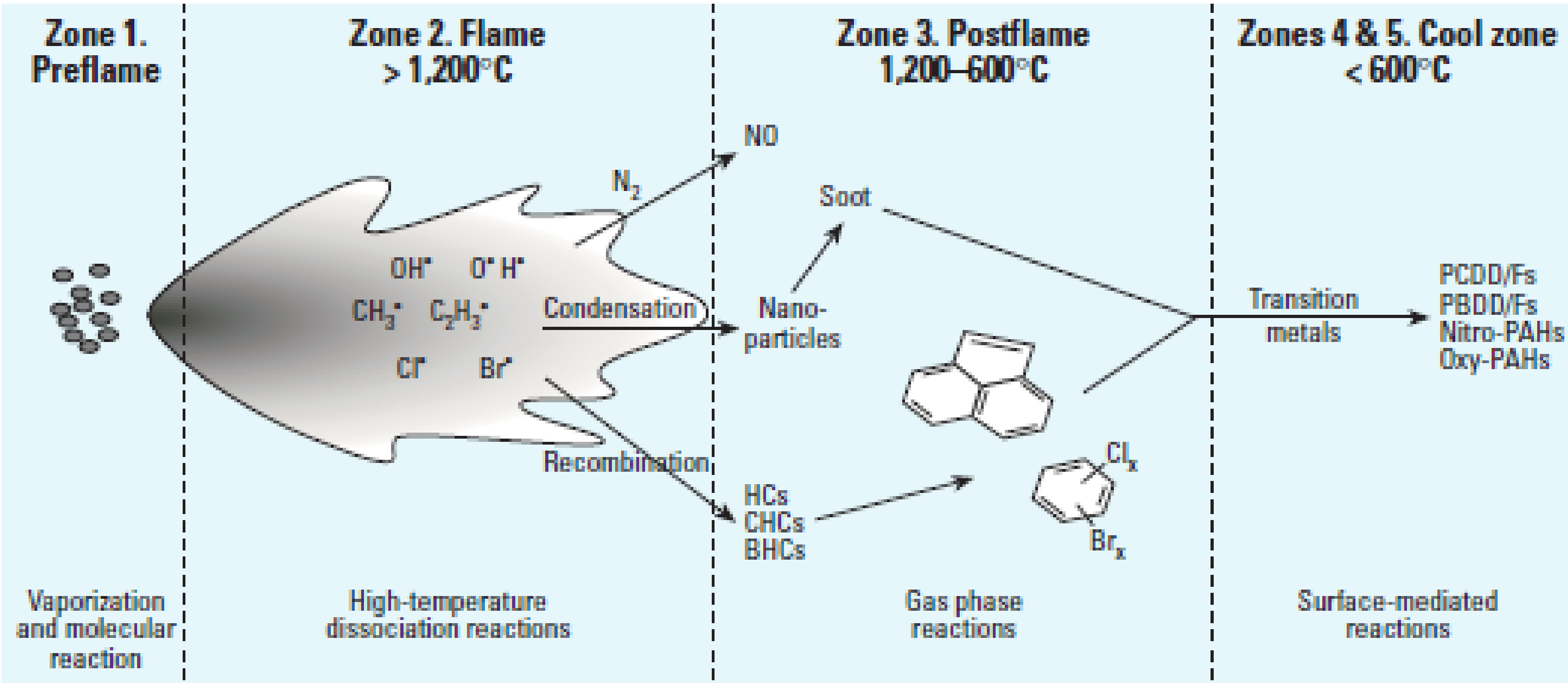


Figure 1. Combustor reaction zones. Zone 1, preflame, fuel zone; zone 2, high-temperature, flame zone; zone 3, postflame, thermal zone; zone 4, gas-quench, cool zone; zone 5, surface-catalysis, cool zone. PBDD/Fs, polybrominated dibenzo-*p*-dioxins and dibenzofurans. Reaction products from upstream zones pass through downstream zones and undergo chemical modifications, resulting in formation of new pollutants. Zone 2 controls formation of many "traditional" pollutants (e.g., carbon monoxide, sulfur oxides, and nitrogen oxides). Zones 3 and 4 control formation of gas-phase organic pollutants. Zone 5 is a major source of PCDD/Fs and is increasingly recognized as a source of other pollutants previously thought to originate in zones 1–4.

Origin and Health Impacts of Emissions of Toxic By-Products and Fine Particles from Combustion and Thermal Treatment of Hazardous Wastes and Materials

Stephania A. Cormier,[1] Slawo Lomnicki,[2] Wayne Backes,[3] and Barry Dellinger[2]

[1]Department of Biological Science, and [2]Department of Chemistry, Louisiana State University, Baton Rouge, Louisiana, USA; [3]Department of Pharmacology, Louisiana State University Health Sciences Center, Baton Rouge, Louisiana, USA

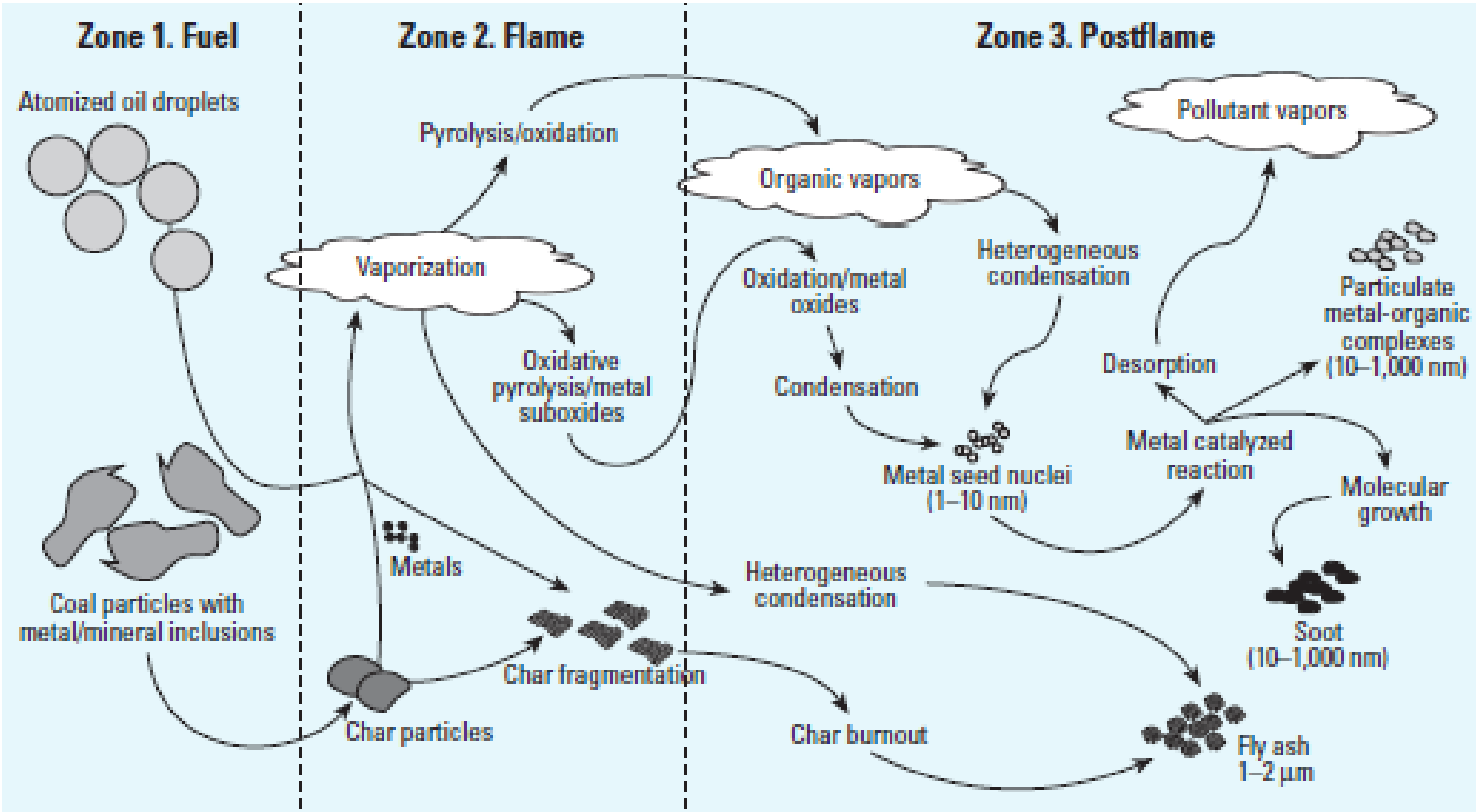


Figure 2. Nanoparticle formation/growth and mediation of pollutant-forming reactions in combustion systems. The combustor reaction zones described in Figure 1 effect particle formation as well as gas-phase pollutant formation. Metals and other refractory compounds are vaporized in the flame zone. They can recondense as cluster or seed nuclei in the postflame zone, where they catalyze further particle growth and pollutant formation in the cool zones.

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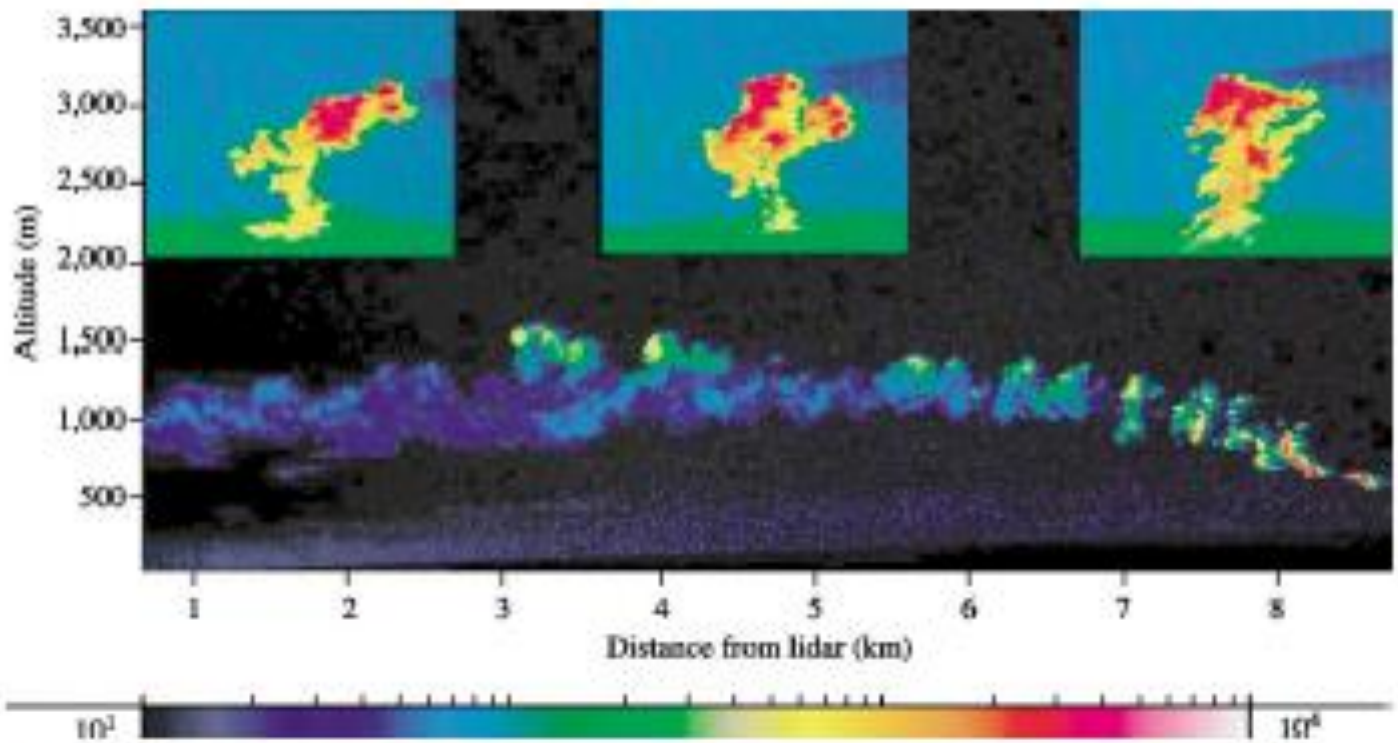

Figure 20 Lidar images of an aerosol-rich plume emitted from a power plant. The plume, viewed lengthwise from the ground, is above the PBL at an altitude of ~0.3 km. The inserts are enlarged cross-sections of a similar plume, taken at 1 min intervals. The shapes of such sections change continuously with both position along the length of the plume and with time. Nanticoke power plant, Ontario, Canada. The lengthwise and cross-section images were taken on January 22 and 19, 2000, respectively at 1,064 nm, with scan speeds adjusted depending on distance from source and proximity of mobile lab to produce a full-scan image in less than 1 min. The images were obtained by and are courtesy of K. Strawbridge, Meteorological Service of Canada.

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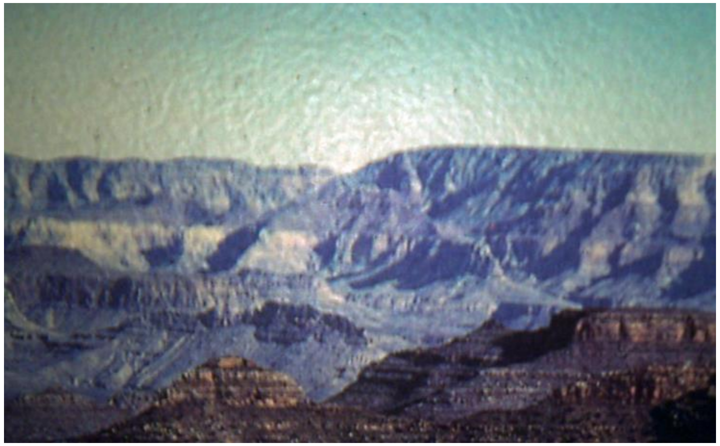
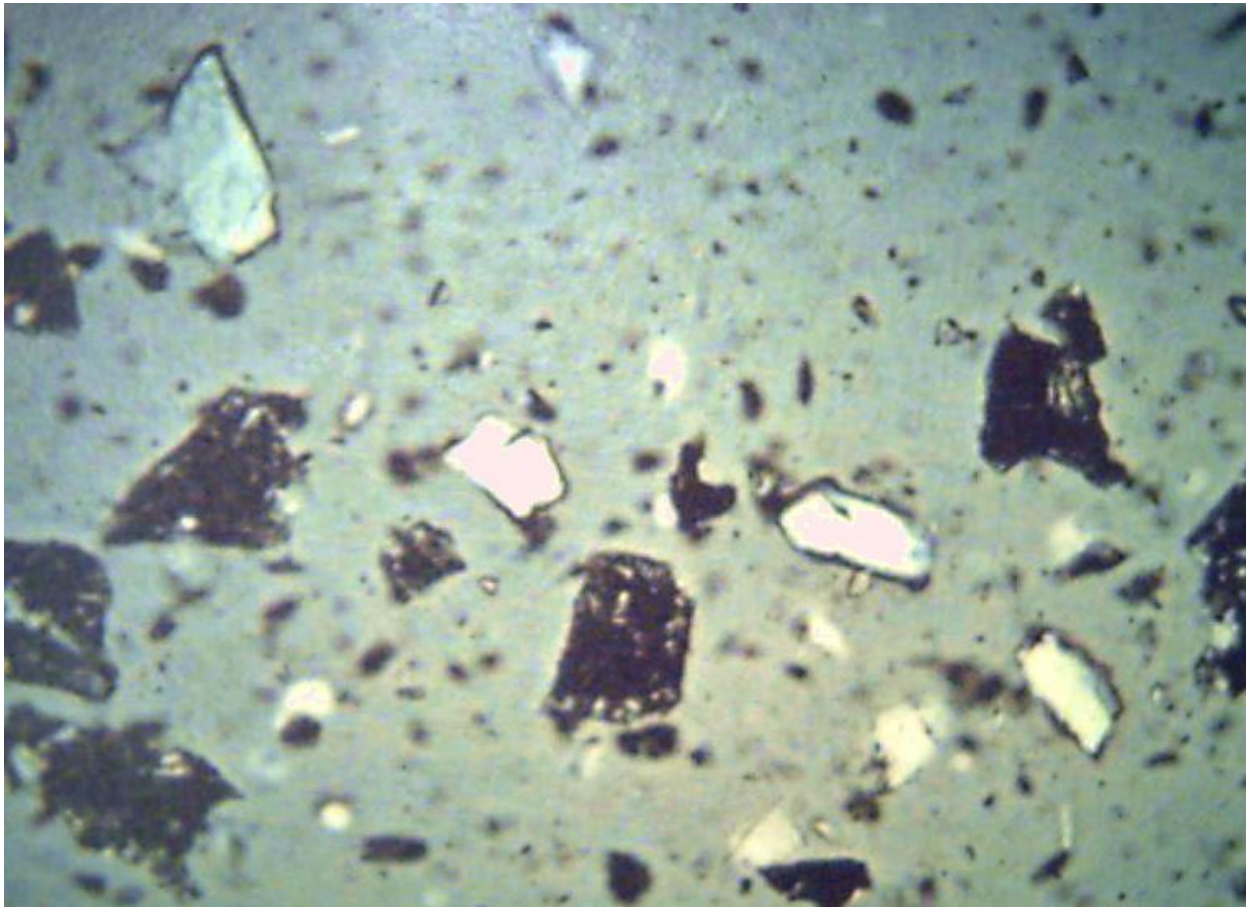
Low Levels of Particles Alter Visibility



9/11/2003

Adapted from JAWMA Sept. 1984

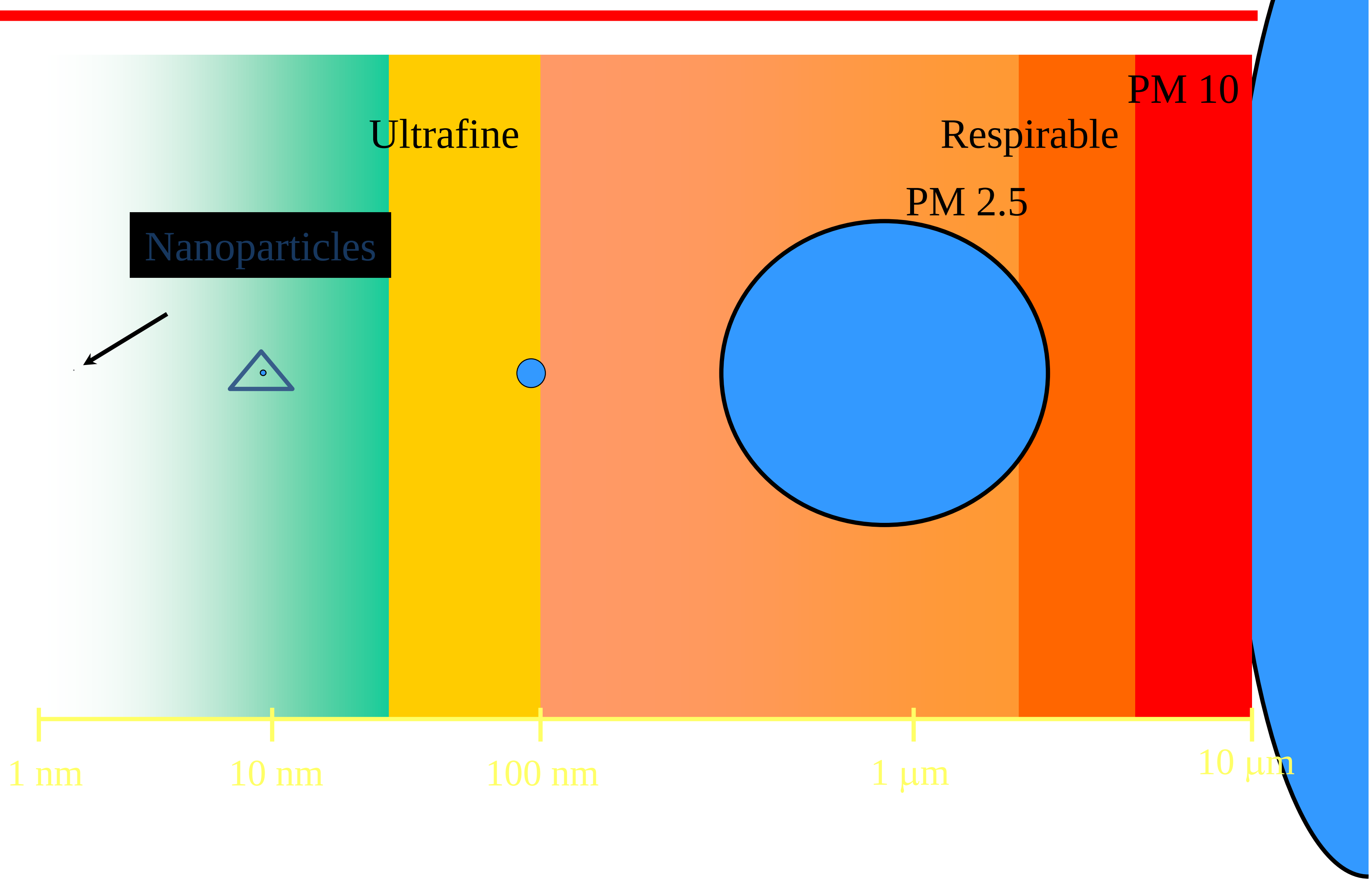
Particles Vary in Size and Shape



9/11/2003

Adpted from The Particle Atlas, McCrone and Delly, 1973

Particle Scale



PM2.5 and UFP are heterogeneous and a large fraction of these particles are combustion generated.

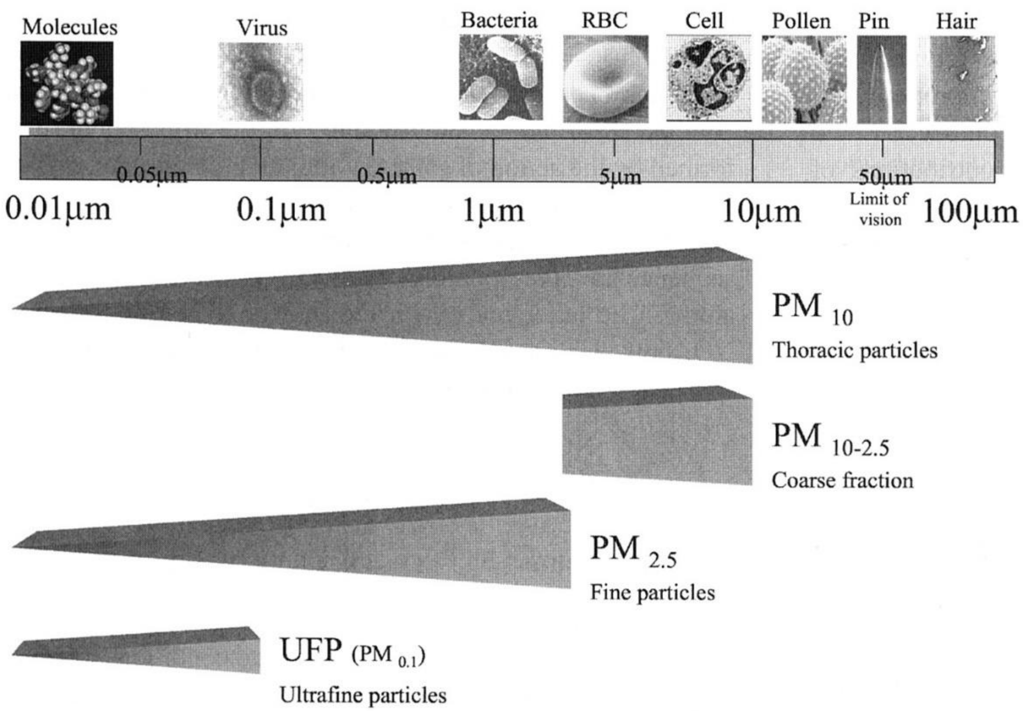


Figure 1. Particulate matter air pollution size distribution.

Most Black Carbon and the particles that seem to be the most biologically active are in the Nanoparticle Size Range

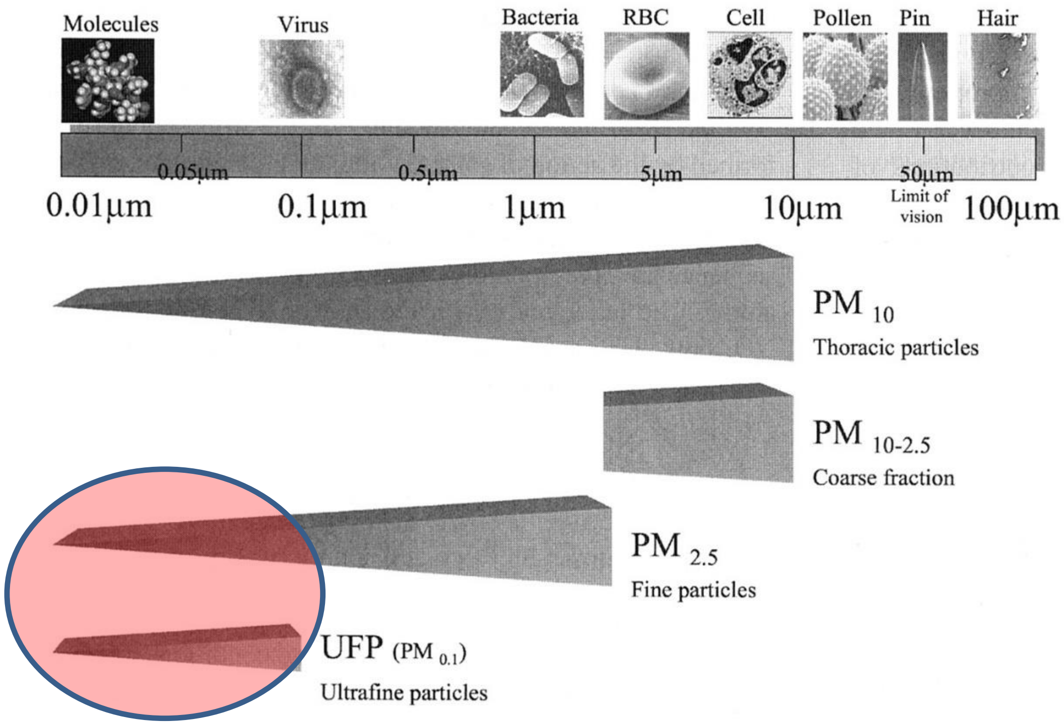
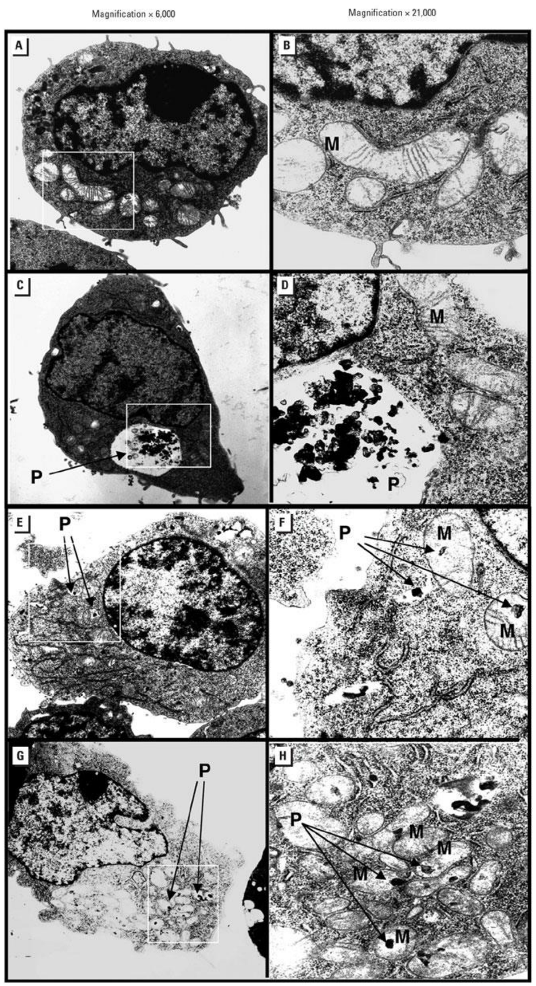


Figure 1. Particulate matter air pollution size distribution.

Nanoparticles Can Penetrate Cell Membranes

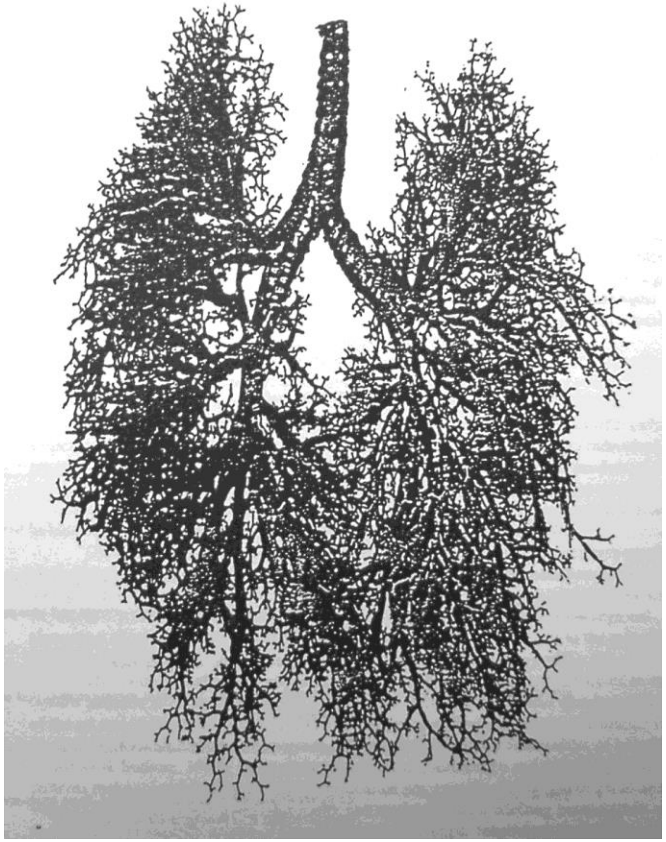


- Normal Macrophage (A, B).
- Coarse particles collected in large cytoplasmic vacuoles (C, D).
- Mitochondrial architecture remained intact after coarse PM incubations
- UFPs frequently lodged inside mitochondria (G, H).
  - Cells incubated with UFPs showed extensive mitochondrial disruption (H).

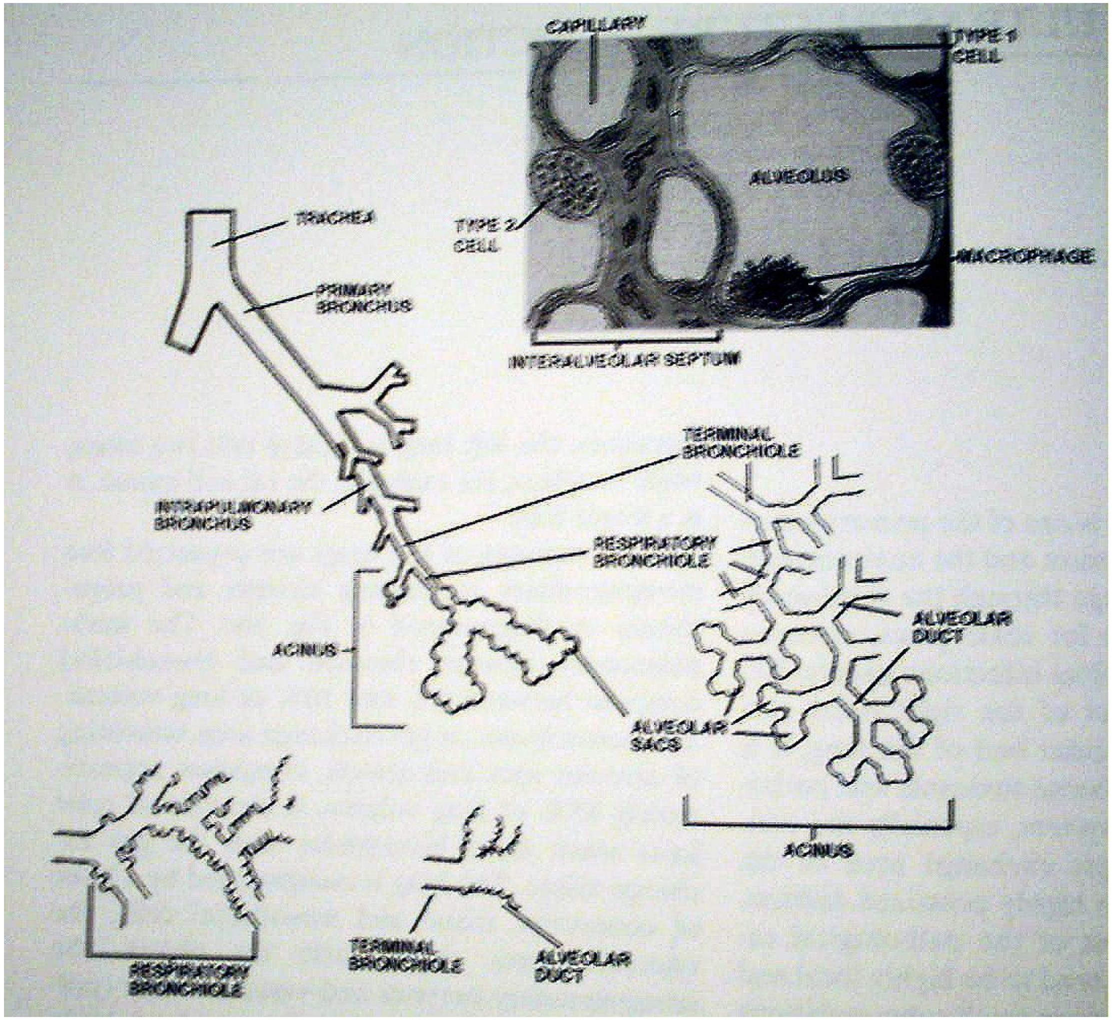
From Li et al., Env. Health Perspect. 2003

Let's start by looking at the Lung

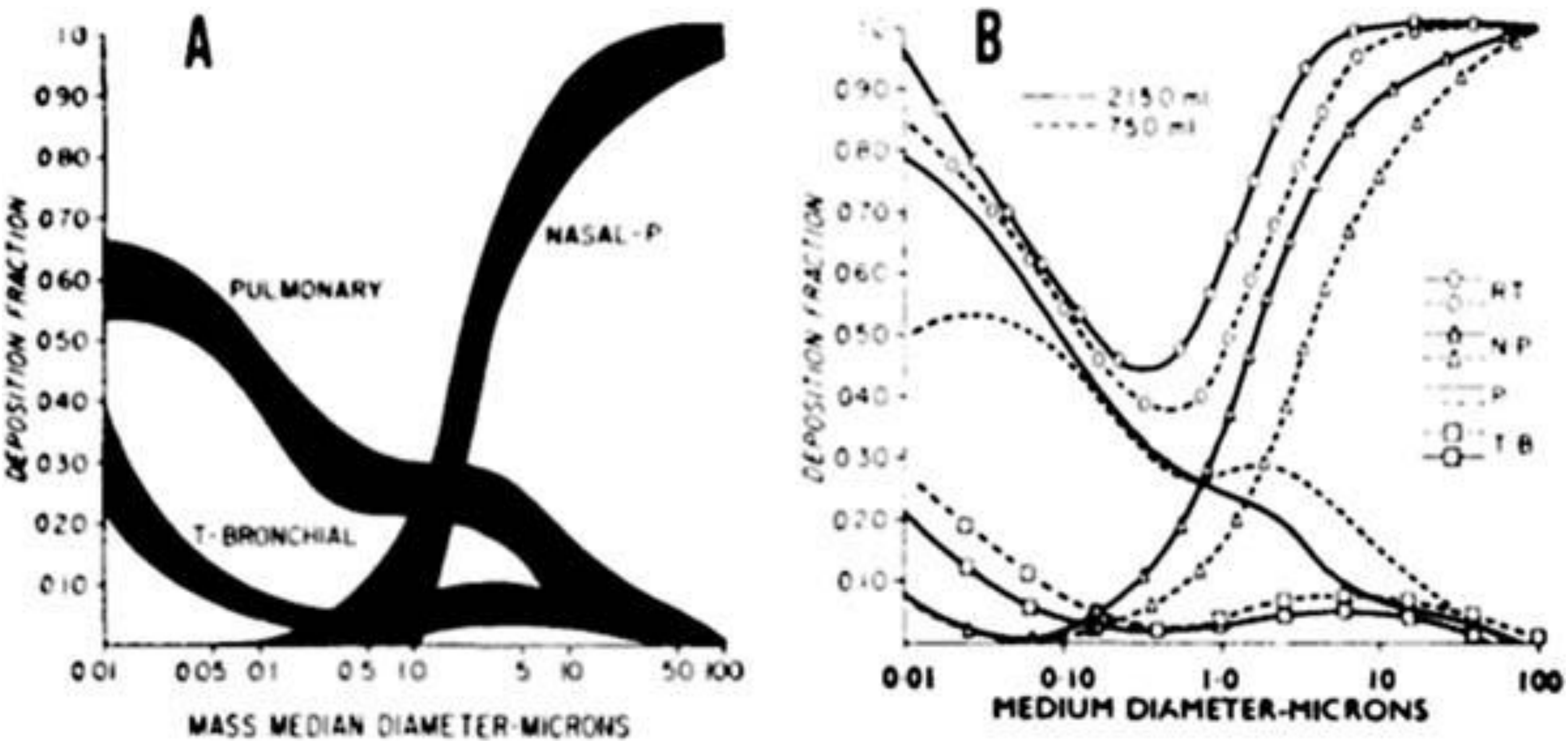
- The human lung is a complex, branching structure.
- The structure is also complex at the cellular level .
- This complexity means that different parts of the lung have different sensitivities to particles.



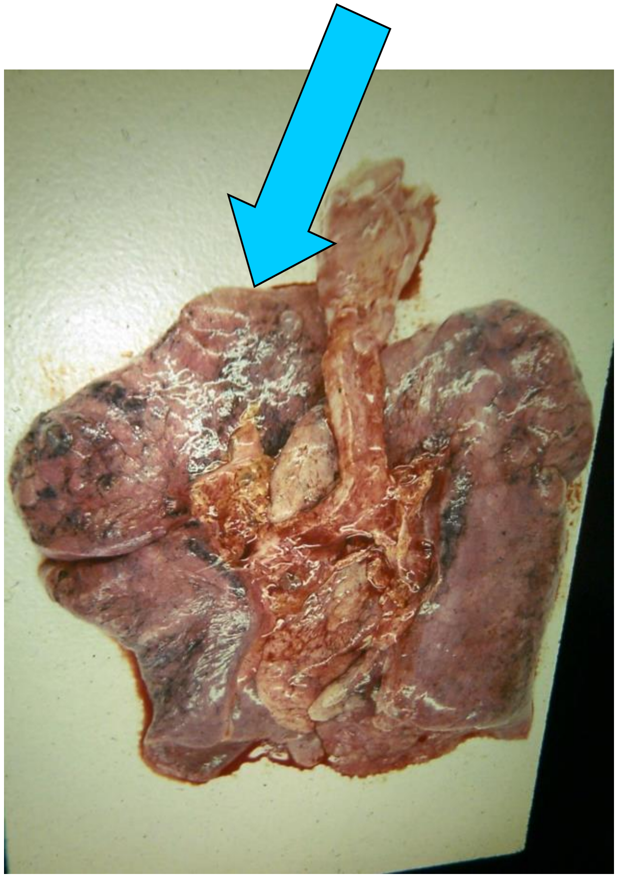
Airways are Complex Structures



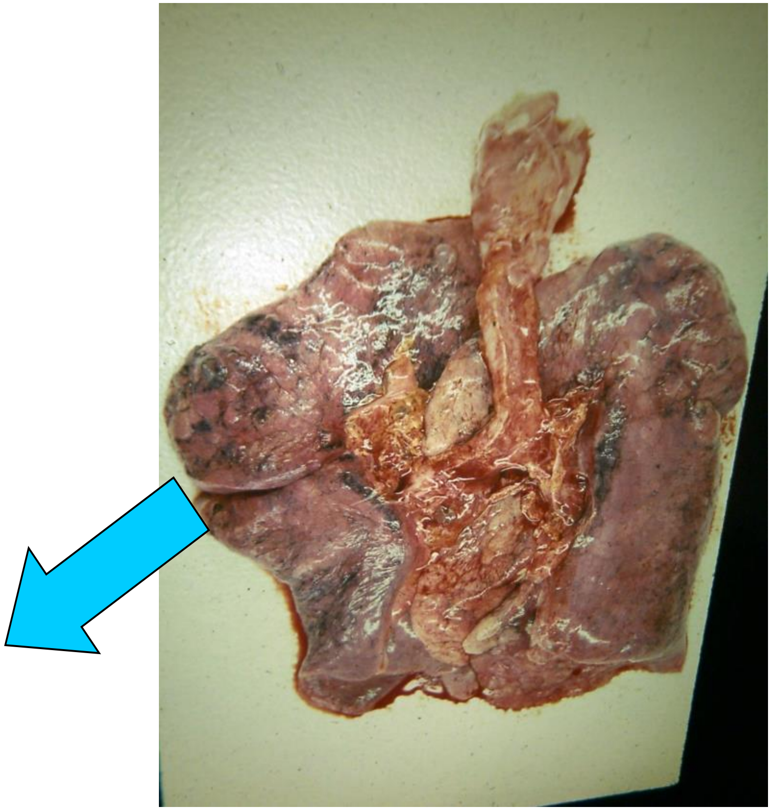
Regional deposition predictions based on model proposed by ICRP Committee II Task Group on Lung Dynamics, indicating effect of variations in σ_g and flowrate. A: each of the *shaded areas* (envelopes) indicates the variable deposition for a given mass median (aerodynamic) diameter in each compartment when the distribution parameter σ_g varies from 1.2 to 4.5 and the tidal volume is 1,450 ml. *B*: two ventilatory states, i.e., 750 ml and 2,150 ml tidal volume (~11 and ~32 liters/min volumes, respectively) are used to indicate the order and direction of change in compartmental deposition which are induced by such physiological factors.
From Task Group on Lung Dynamics



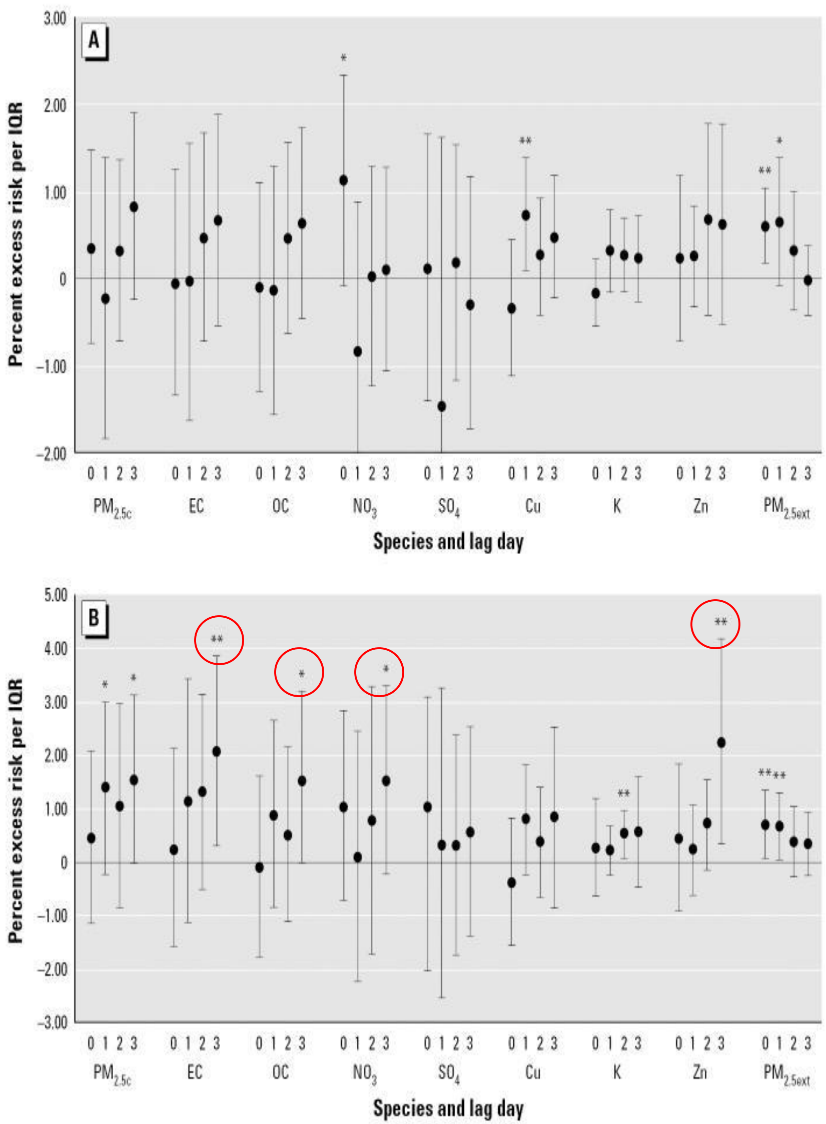
Soluble Particles Clear Quickly Although soluble components can readily enter circulation the less soluble components are retained.



Sequestered particles can still slowly dissociate and reach other organs.



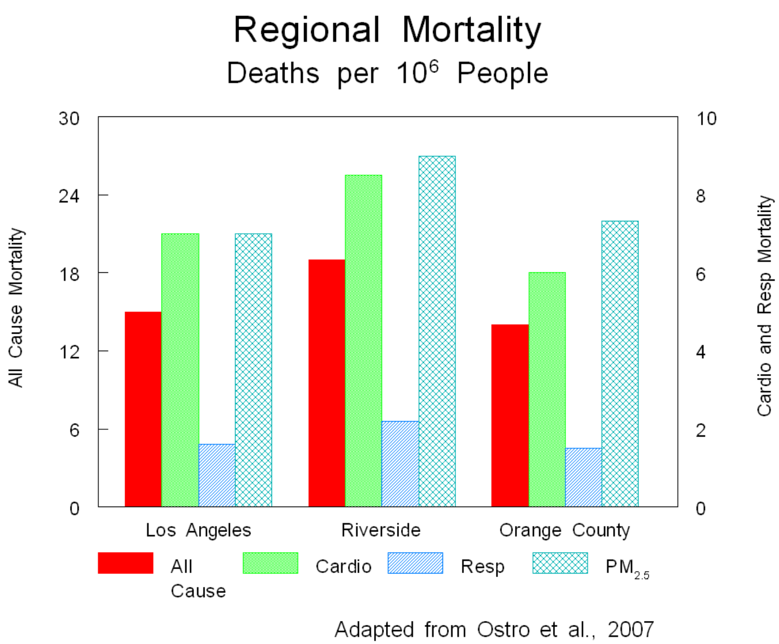
Components of PM could be Causal Agents (or Surrogates)



- Cardiovascular mortality more tightly linked to PM exposure than is total mortality.
- EC, OC, nitrate and Zn may be important based on the epidemiological findings.

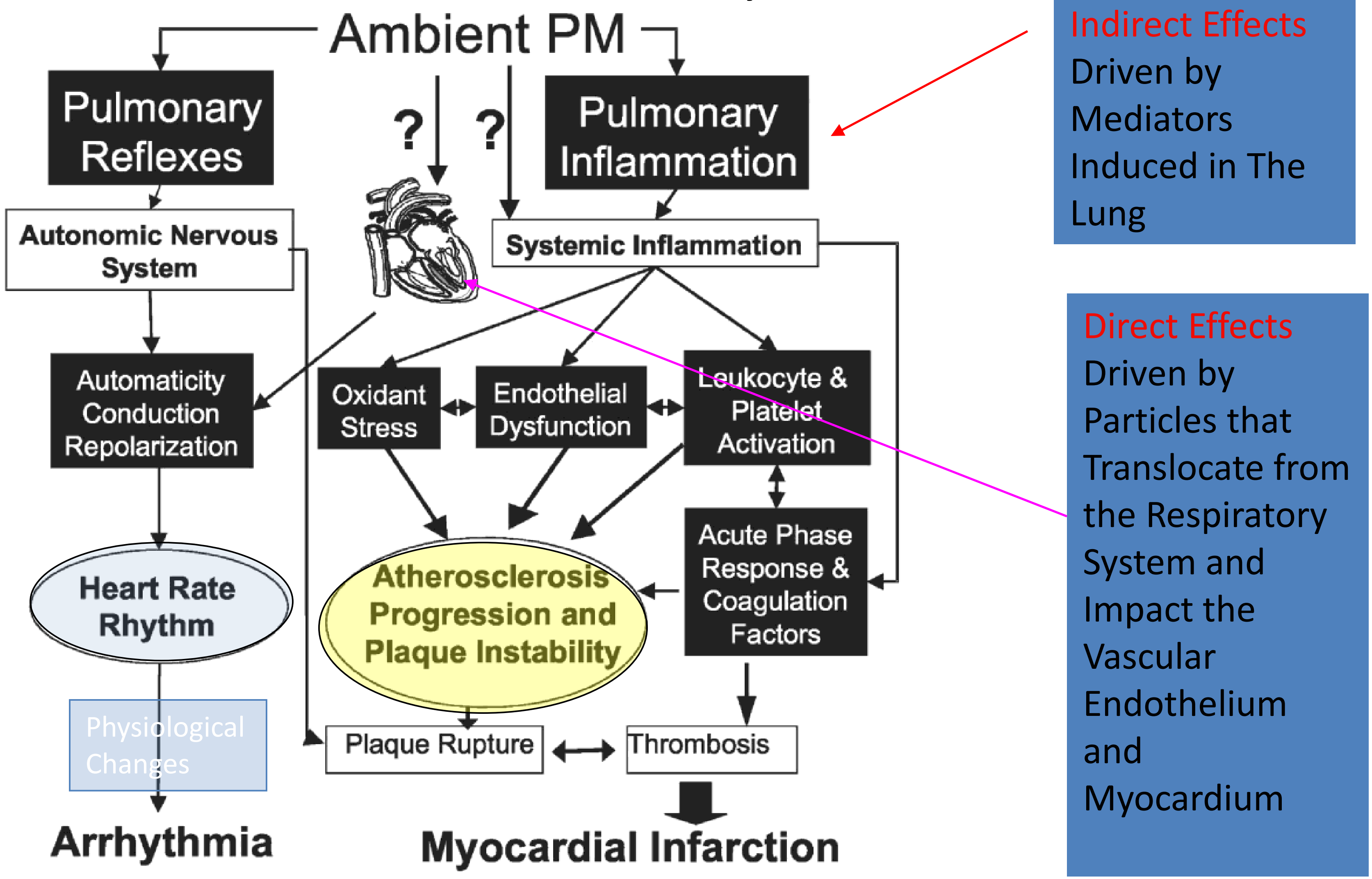
From Ostro et al., Environ Health Perspect. 2007 January; 115(1): 13–19.

California Air Pollution is Strongly Associated with Deaths



- Mortality data are consistent with PM exposures on a regional basis in California.
- Cardiovascular Mortality Represents about ½ of all Deaths.

Inhaled Pollutants can Induced Effects Through Multiple Pathways



Adapted from: Pope, 2006, Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution,. Circulation :114, 2443 -8

Controlled Studies Using Models of Diseases Can Be Used to Identify Causal Agents and Mechanisms

- Rats
  - Aged (geriatric)
  - MI
  - Hypertensive (SHR)
- Mice
  - ApoE-/- (3, 4 and 6 month studies)
  - Nrf2-/- (in progress)
  - Diabetes (proposed)

Various Endpoints Can Be Measured Before, During and After Exposures

- Biochemistry
  - Blood Samples
    - Acute phase proteins
    - Cytokines
  - Expired Breath
- Cardiac Physiology
  - Heart Rate and HRV
  - Blood Pressure
    - Systolic
    - Diastolic
    - Mean
  - Developed Pressure
  - Contractility
- Molecular Biology
  - Gene/protein expression from lung, heart, brain
  - In-situ hybridization for effects localization

SIZE IS IMPORTANT BUT SIZE ISN'T EVERYTHING

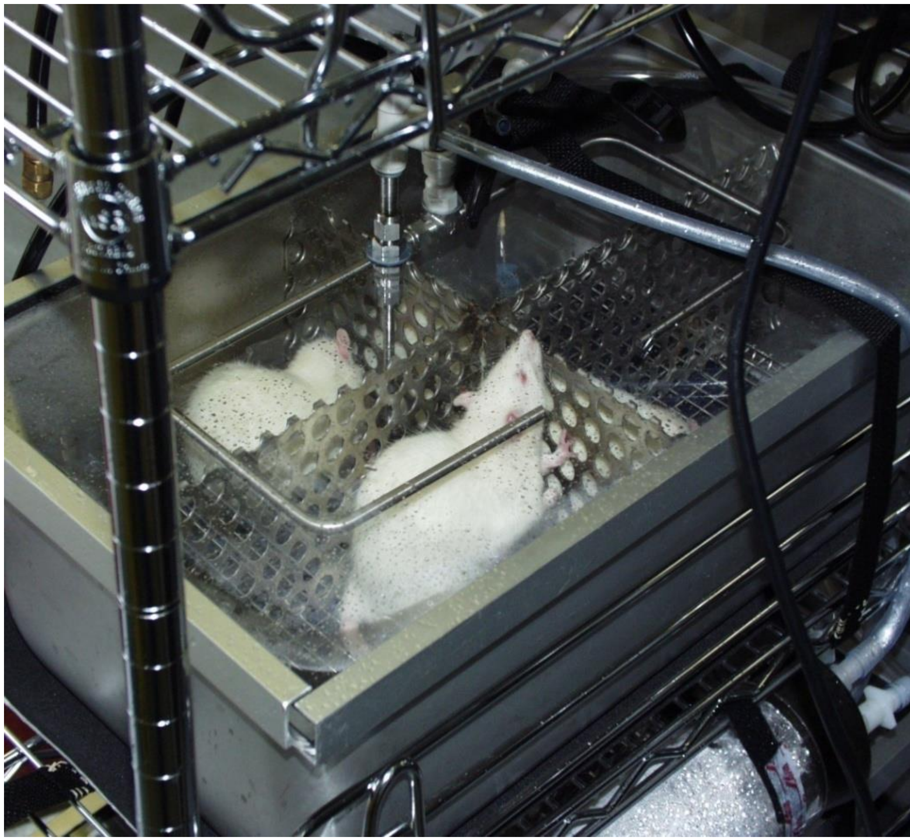
- The composition of the particles should affect the way that particles interact with biological systems.
- UFP have high surface to mass ratios which makes the good 'carriers' for adsorbed substances.
  - Can carry toxins to areas which they couldn't ordinarily reach
  - Can become activated by adsorbing reactive substrates
- It has become well-established that the organic constituents on particles play an important role in development or worsening of cardiovascular diseases.
  - Atherosclerosis
  - Hypertension
- Some of our data indicated worse outcomes after exposures during summer (high photochemical activity, O_3) than during fall or winter.

Experimental

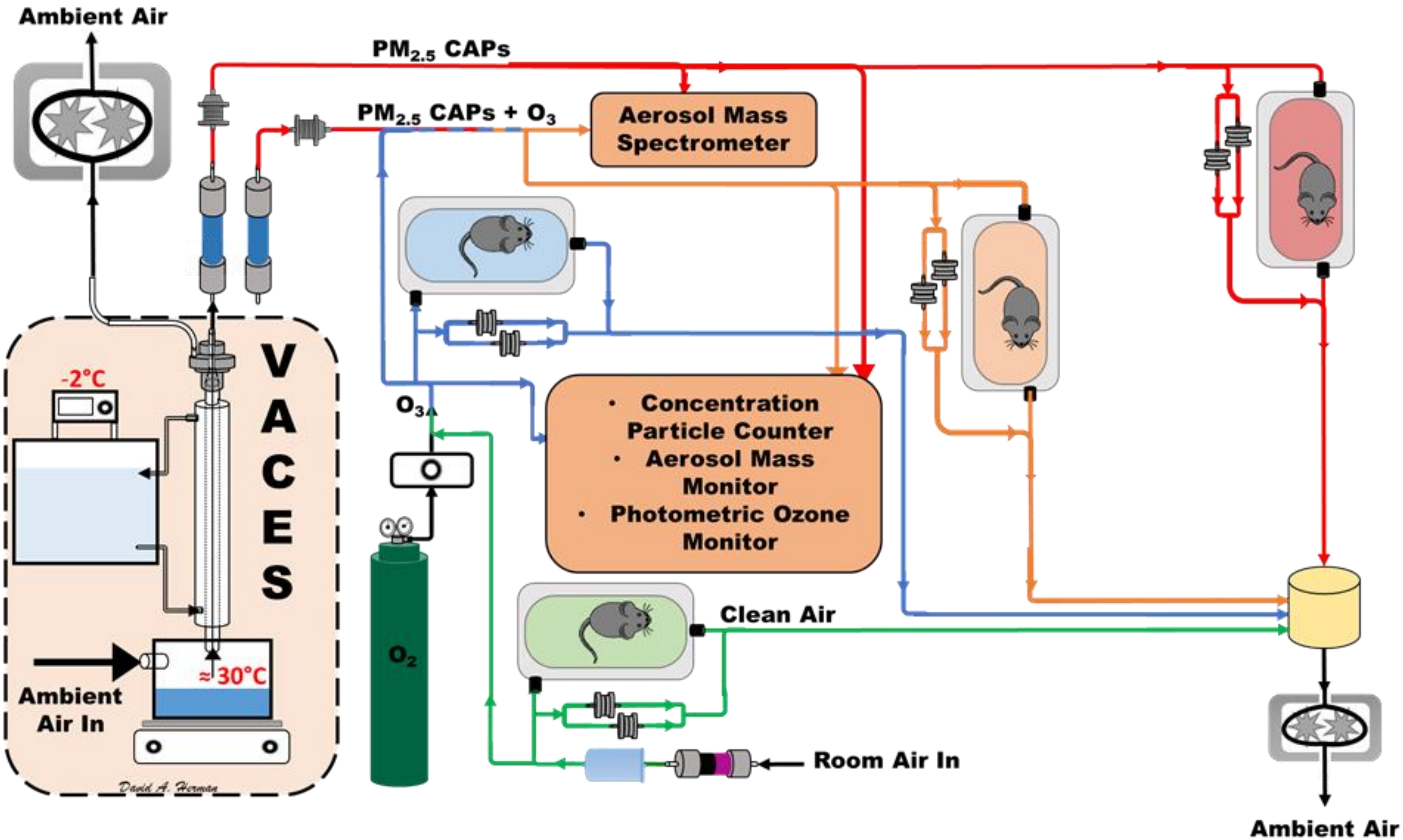
- Rats were exposed to Air or CAPs
- Vapor- and particle-phase samples collected in parallel to animal exposure
- Mean arterial pressure and heart rate were measured
  - during exposure
  - at night after exposure

Rats or Mice Can Be Exposed to Purified Air or CAPs in Sealed Chambers

The Sealed Chambers Can Be Placed Onto Racks to Facilitate Transport



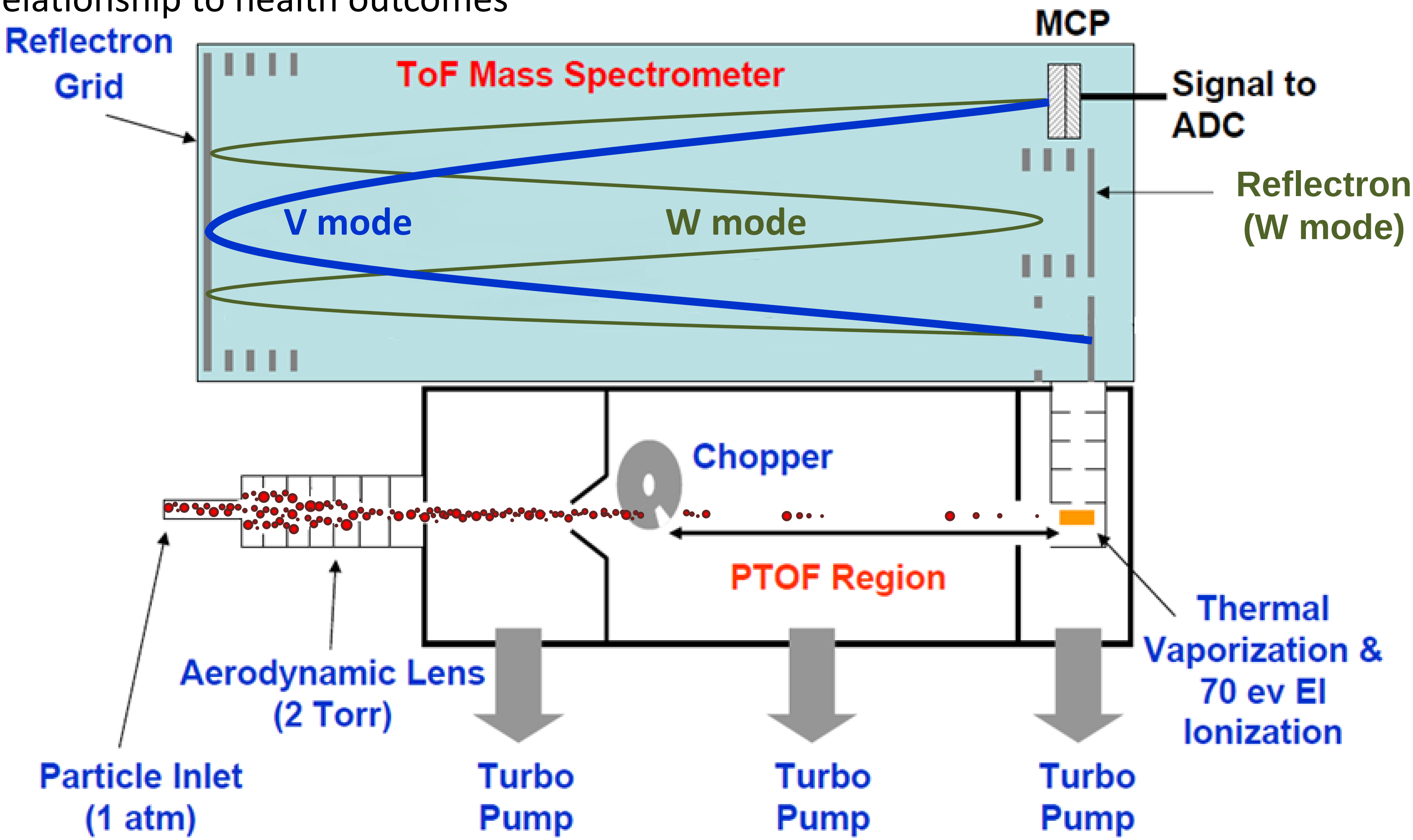
Airborne particles were concentrated using a Versatile Aerosol Concentration Enrichment System (VACES) in conjunction with a Dakati Thermal Denuder. O_3 was metered into the Concentrated Ambient Particles (CAPs) to reach a final concentration of 0.2 ppm.



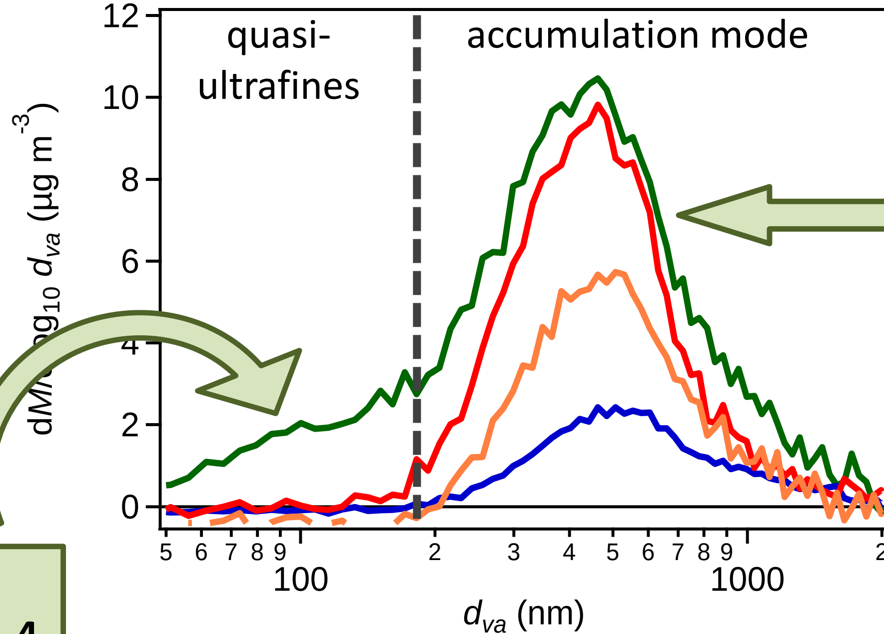
Exposure Protocol

- ApoE-/- mice were surgically implanted with ECG telemetry devices.
- Mice were exposed 5 hr per day (8AM to 1 PM) 4 days per week for 8 weeks at UC Irvine and were housed in filtered air-supplied caging systems between exposures.
- ECG data were monitored during exposures and while the mice were in housing (21 hr / day).
- All animal protocols were approved by the Institutional Animal Care and Use Committee.

- We are going to look at data generated using the Aerosol Mass Spectrometer
- AMS provides real time high resolution mass spectra of particles as well as particle size distributions (aerodynamic diameter).
- We will specifically look at oxygen to carbon ratios in organic constituents and their relationship to health outcomes



Health-related characteristics of Ultrafine PM



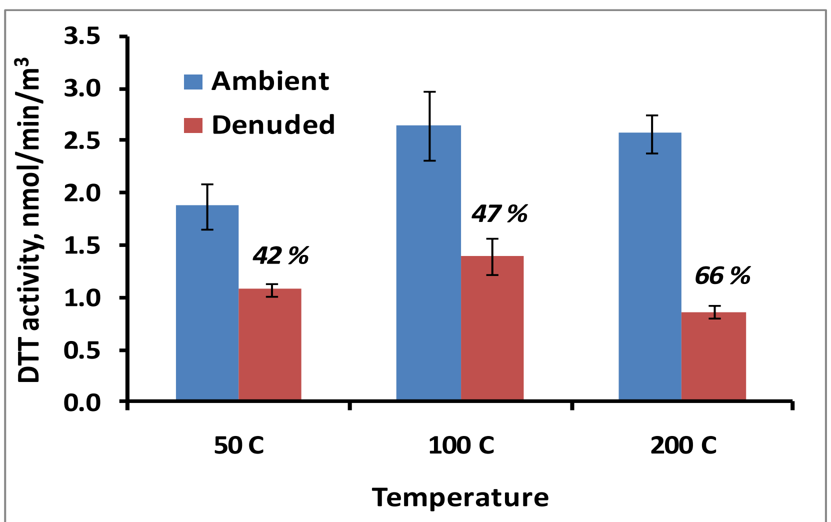
ultrafines
less oxygenated
(to denuder)

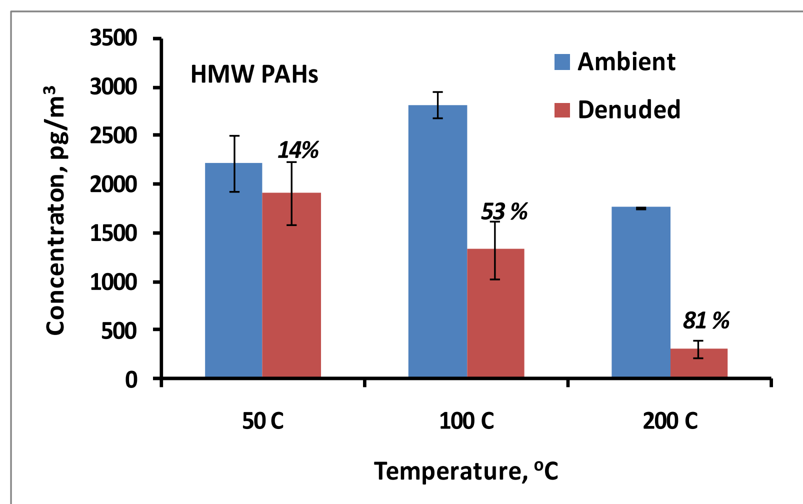
$$\frac{m/z\ 44\ (CO_2^+)}{m/z\ 55\ (C_4H_7^+)} \cong 0.4$$

larger particles
oxygenated

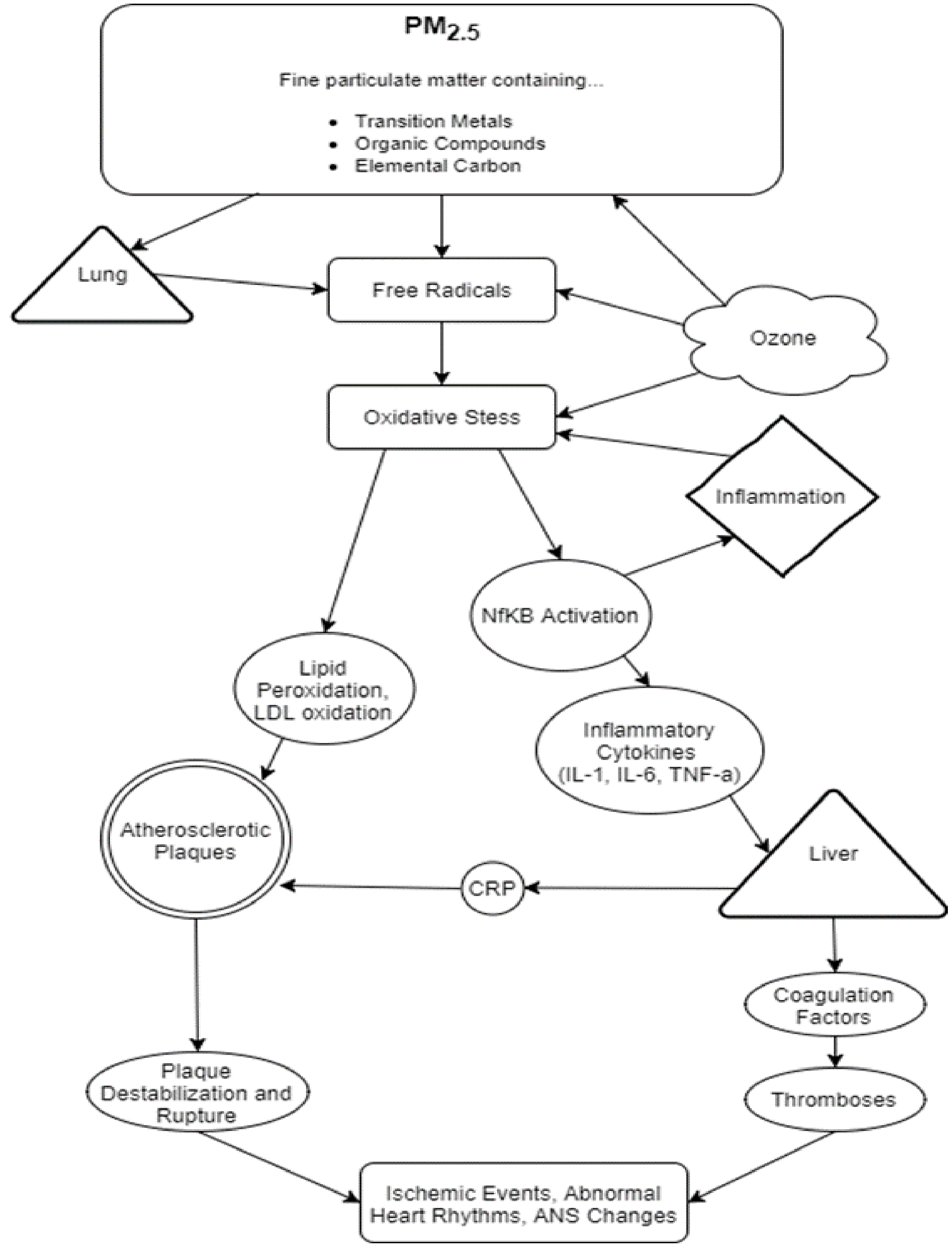
$$\frac{m/z\ 44\ (CO_2^+)}{m/z\ 55\ (C_4H_7^+)} \cong 4$$

When you denude the UFP

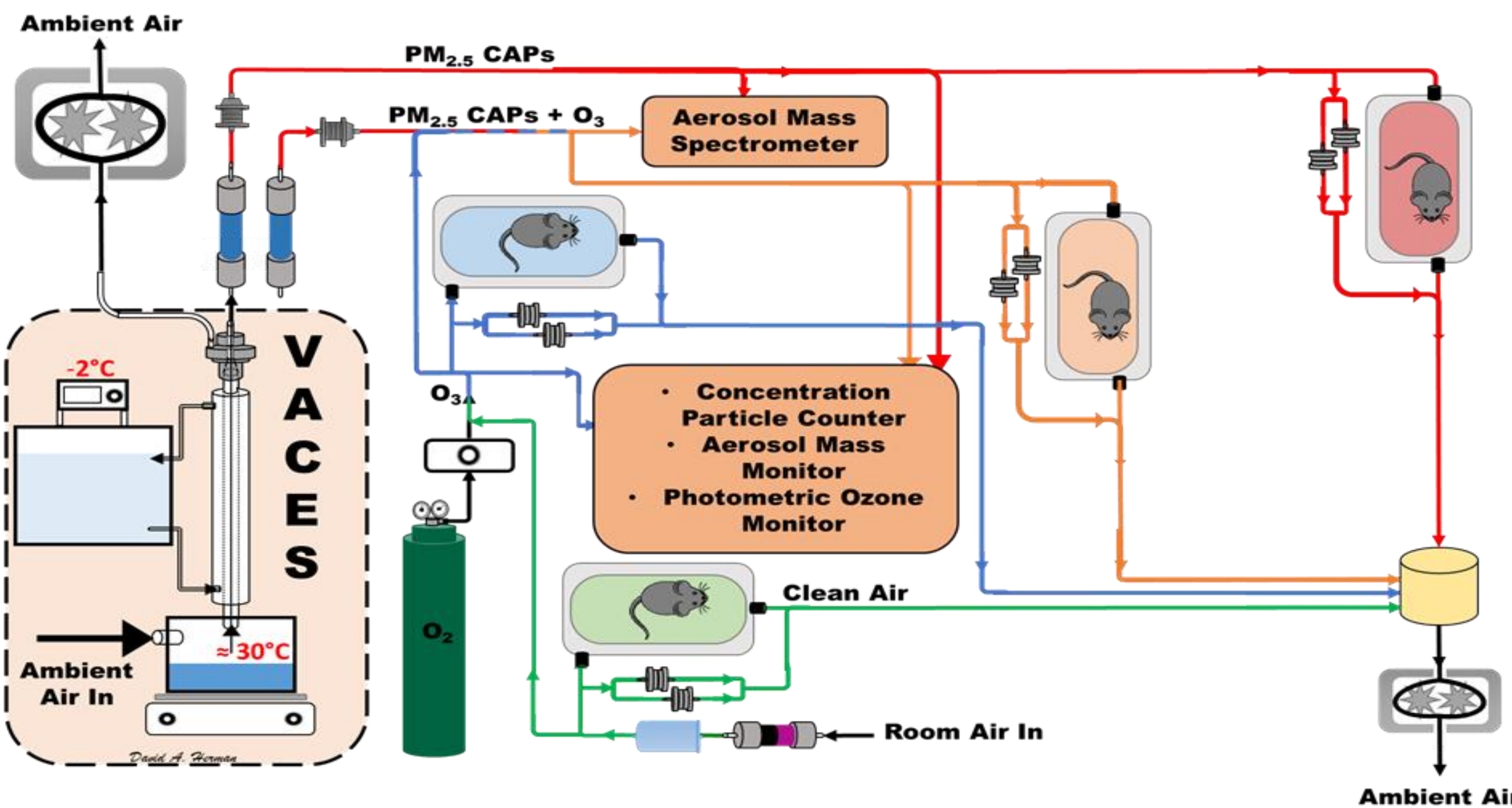




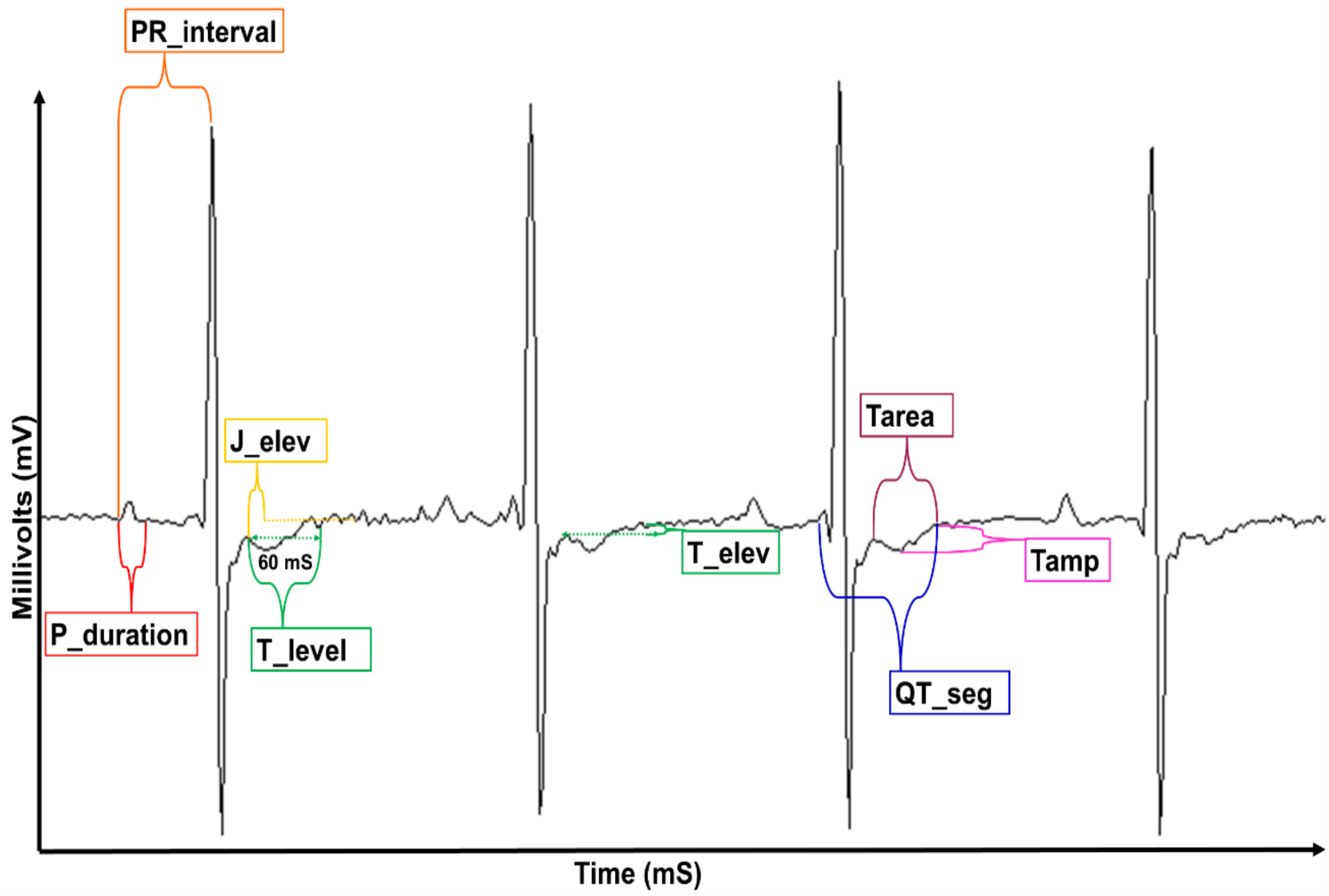
A Mechanistic Framework for PM2.5 and Ozone Effects Leading to Cardiovascular Disease



We designed a study to **1)** elucidate the mechanisms through which concurrent PM2.5 and ozone exposures might induce toxicity and cardiovascular effects; and **2)** determine whether there are interactions between these two air pollutants such that the effects of the concurrent exposure might differ from those of exposures to PM or O_3 when administered alone.

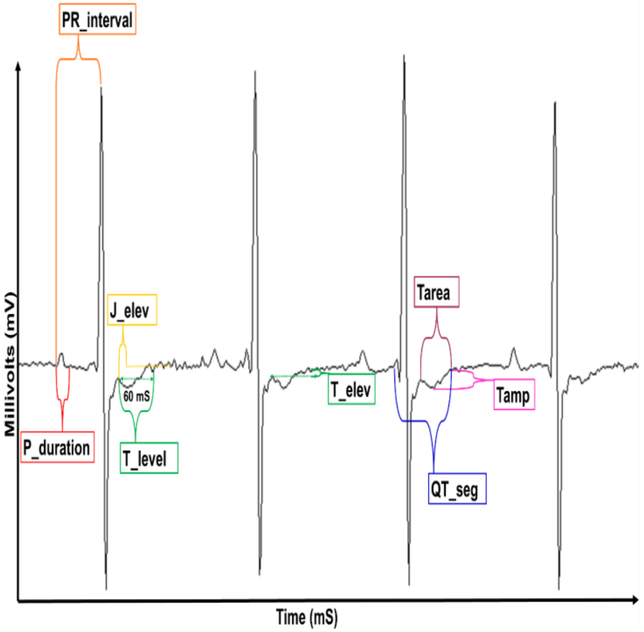


We are going to focus on how cardiac physiology is changed after exposures

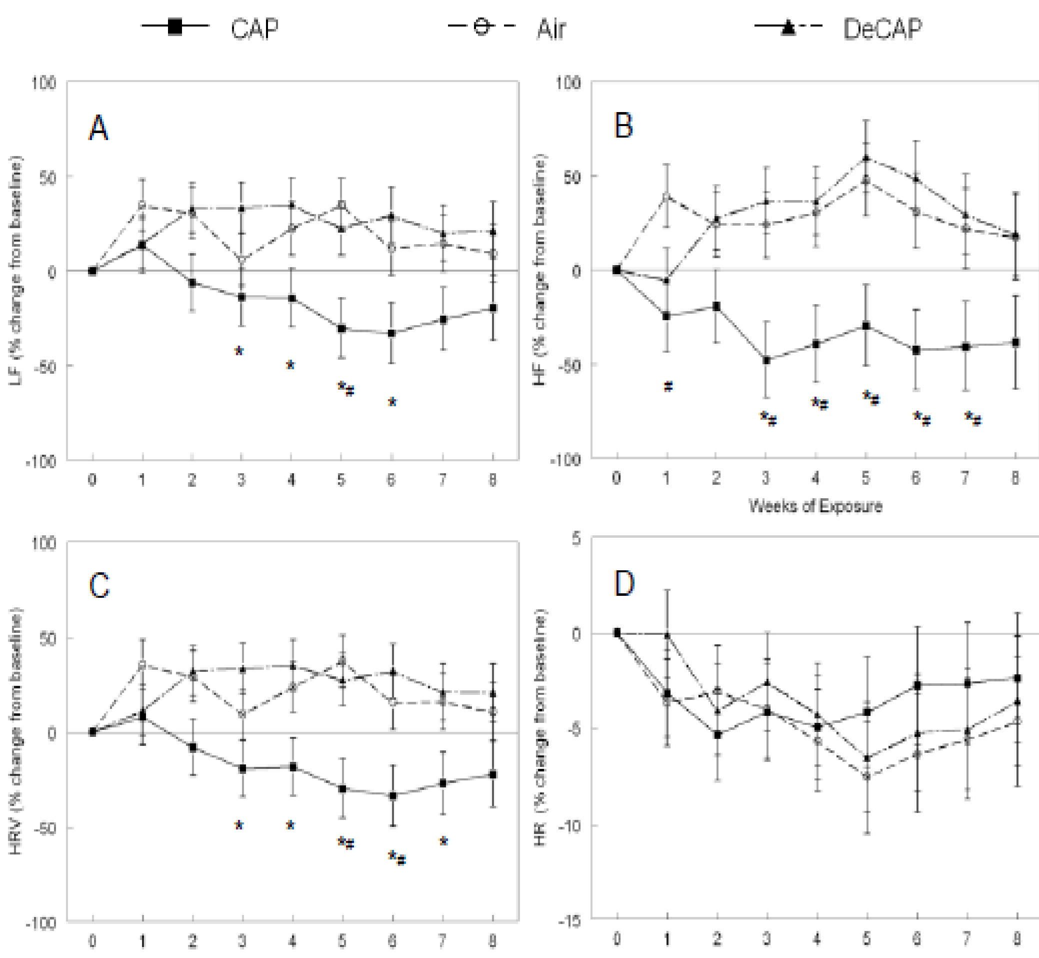


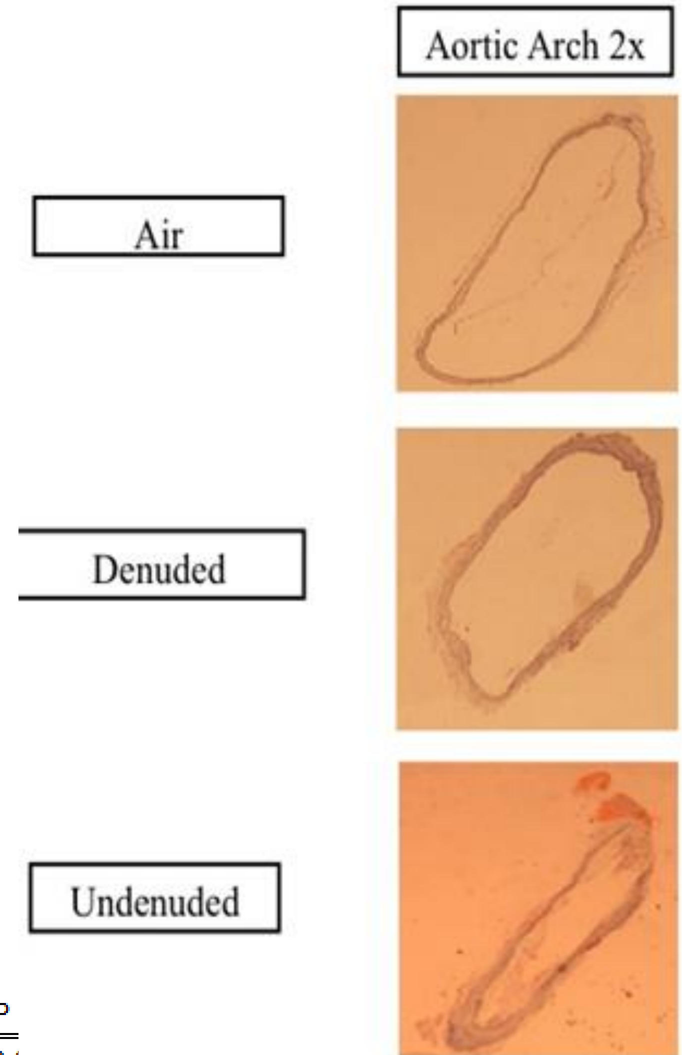
We are going to focus on how cardiac physiology is changed after PM and PM + O_3 exposures

- Changes in heart rate variability (HRV)
  - Decreased HRV is an adverse effect
- Changes in electrocardiographic waveforms

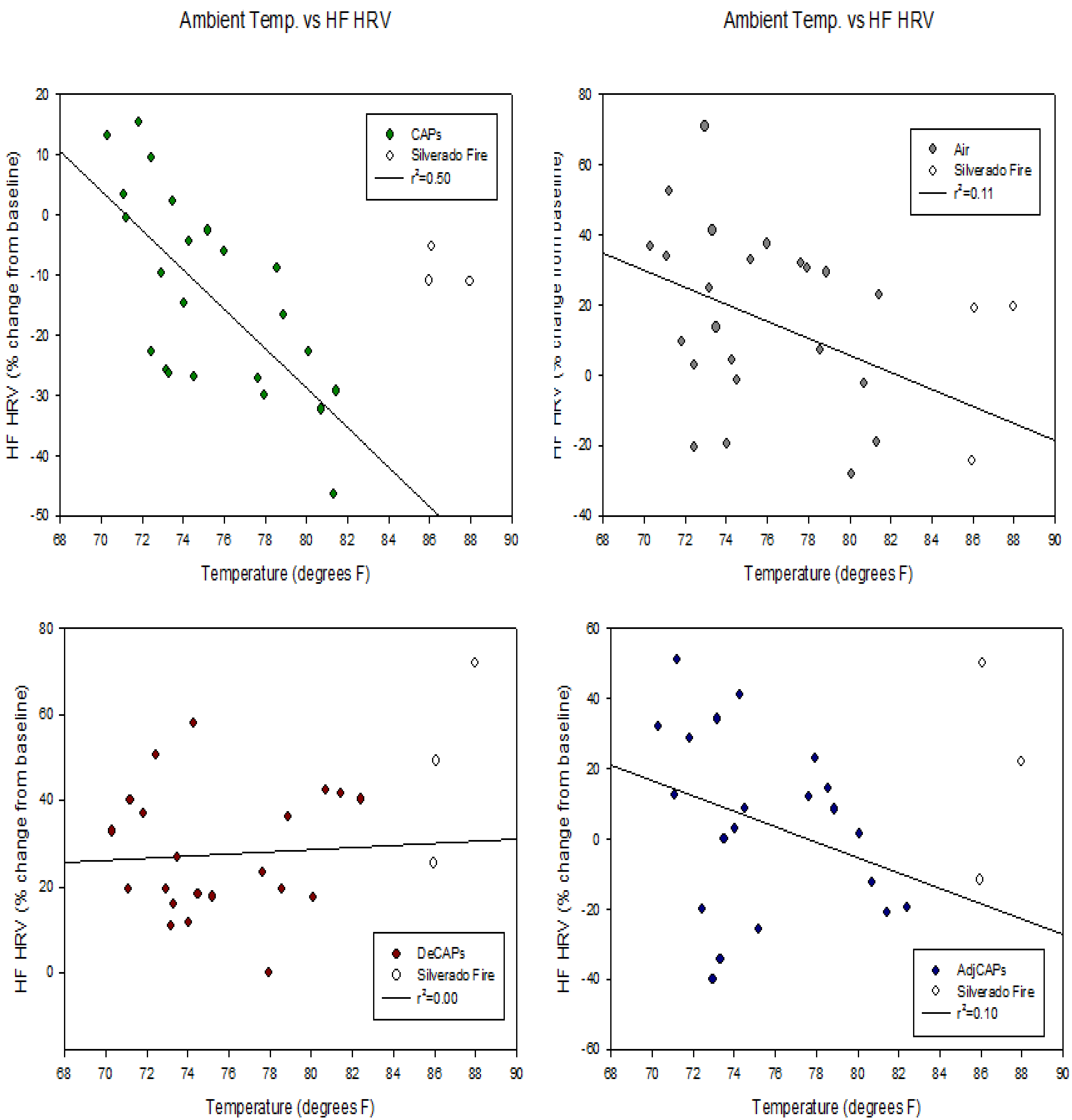


Removing the Organic Constituents From Ambient UFP Blocks CV Effects



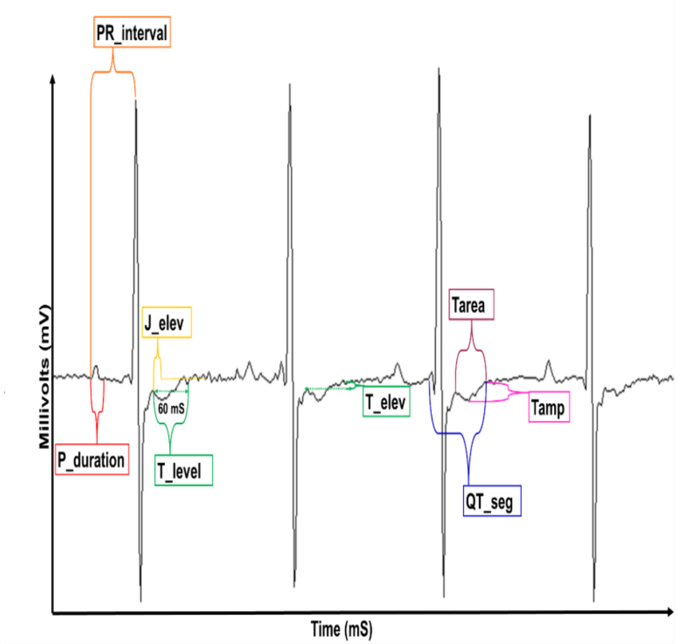


	Air	CAP	deCAP
Plaque Size (% area of plaque in of total lumen CS)	14.5 ± 6.3	29.9 ± 10.0*	2.3 ± 0.
Lipid Accumulation (% area of lipid in total tissue CS)	5.1 ± 3.3	8.9 ± 2.4,[#]	2.2 ± 0.3
Lipid Peroxidation (nM MDA/mg protein)	134 ± 29	218 ± 32*,**	141 ± 17



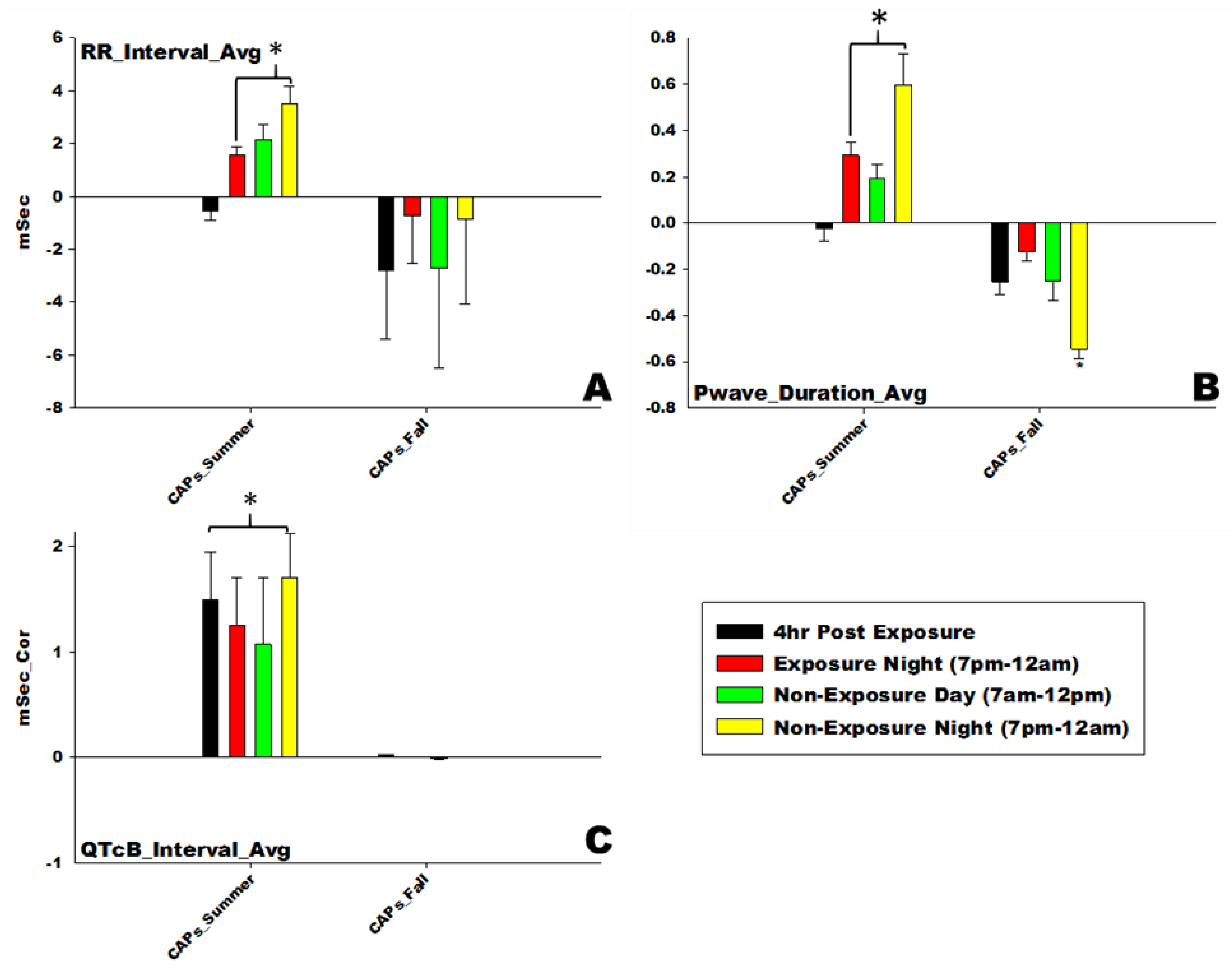
Daily changes in ambient temperature correlated with post-exposure changes in HF HRV in CAPs exposed mice. This correlation was not observed in any other exposure group. Three days (September 15th-17th, shown as white dots) were excluded from the analysis due to the occurrence of Santa Ana winds and potential exposure to wildfire smoke that may be responsible for HRV changes that did not follow the rest of the correlation. The association between temperature and HF HRV is likely

Two additional hypotheses can be examined

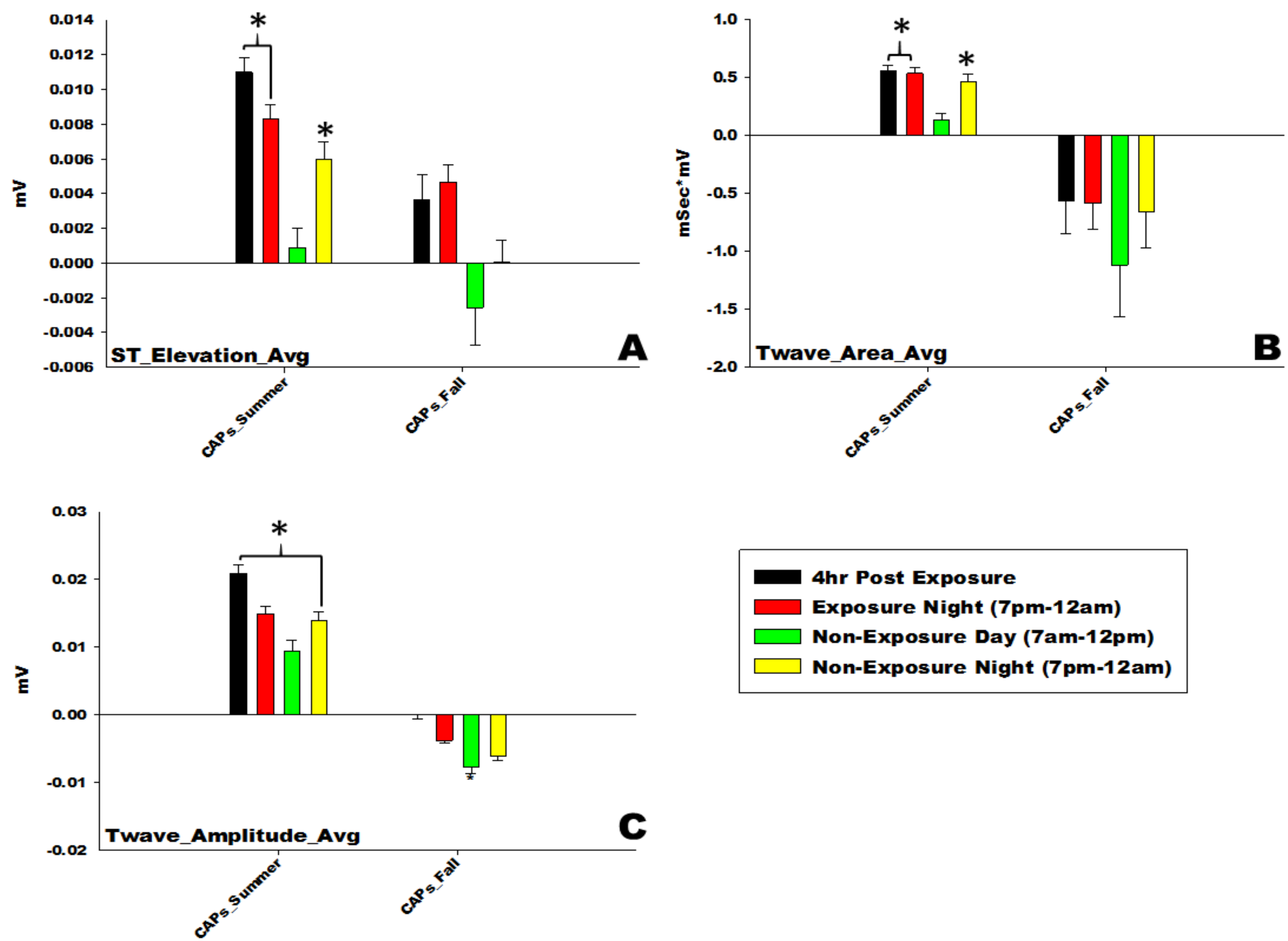


1. Are adverse effects different after summer exposures compared to fall/winter exposures?
   - Changes in electrocardiographic waveforms
2. Does O3 oxidation explain some of these differences?
   - Changes in HRV

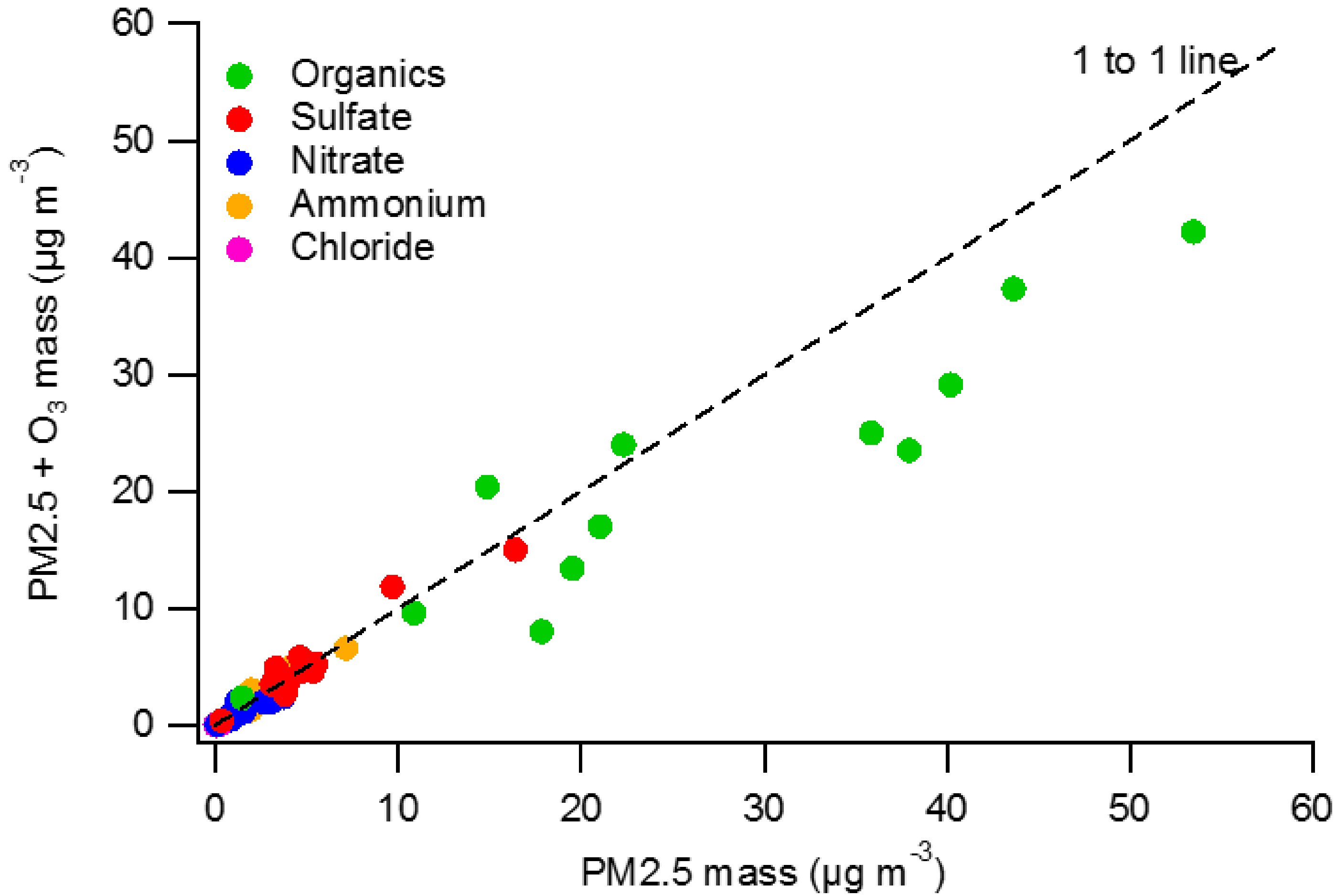
ECG Changes Related to Heart Rate



ECG Changes Related to Ischemia

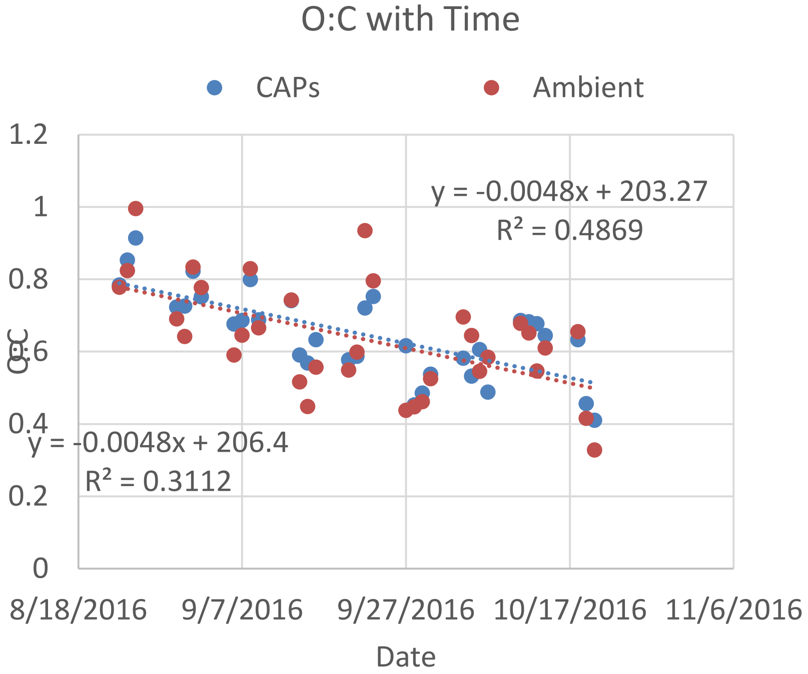


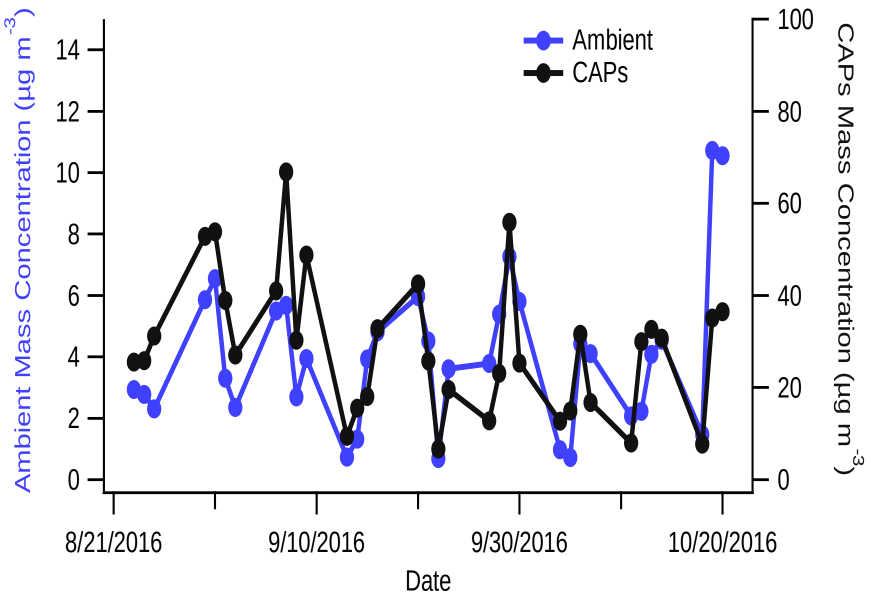
So, does O_3 Oxidation Affect Particle Chemistry and Heart Function?



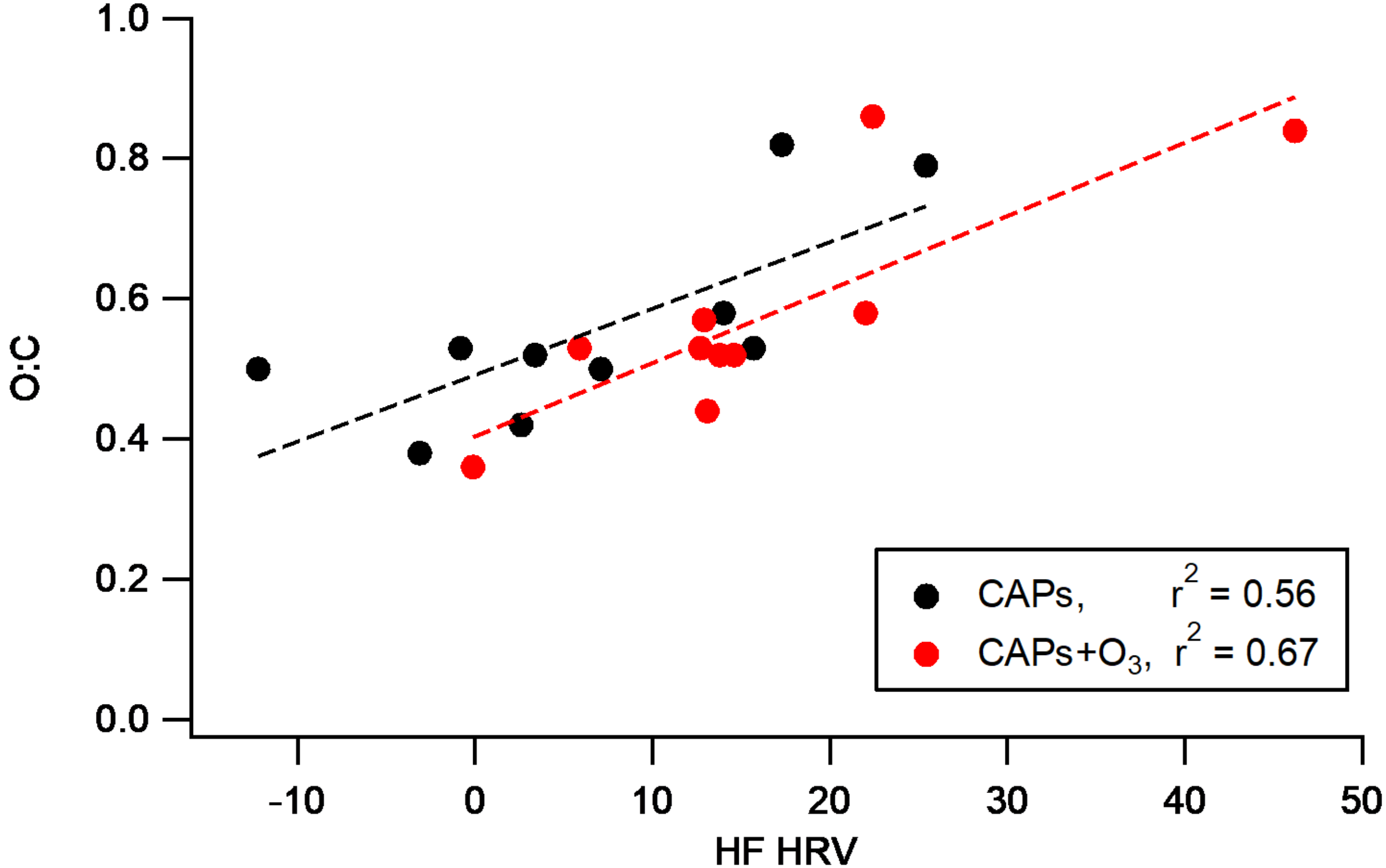
There is the question of whether our concentration methods alter the composition of the exposure aerosols.

- Mass concentrations and O:C are both highly correlated between ambient particles and CAPs.

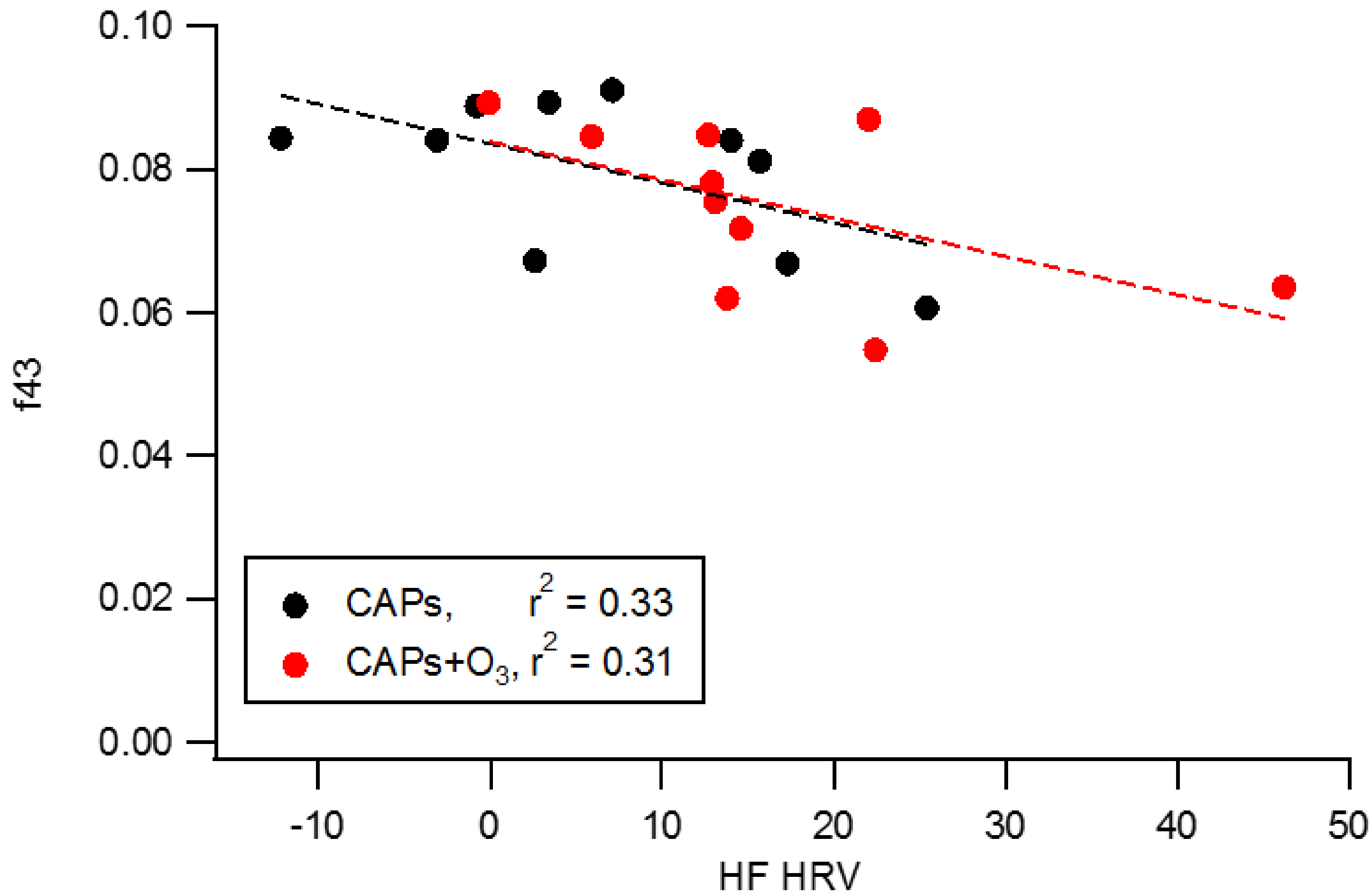




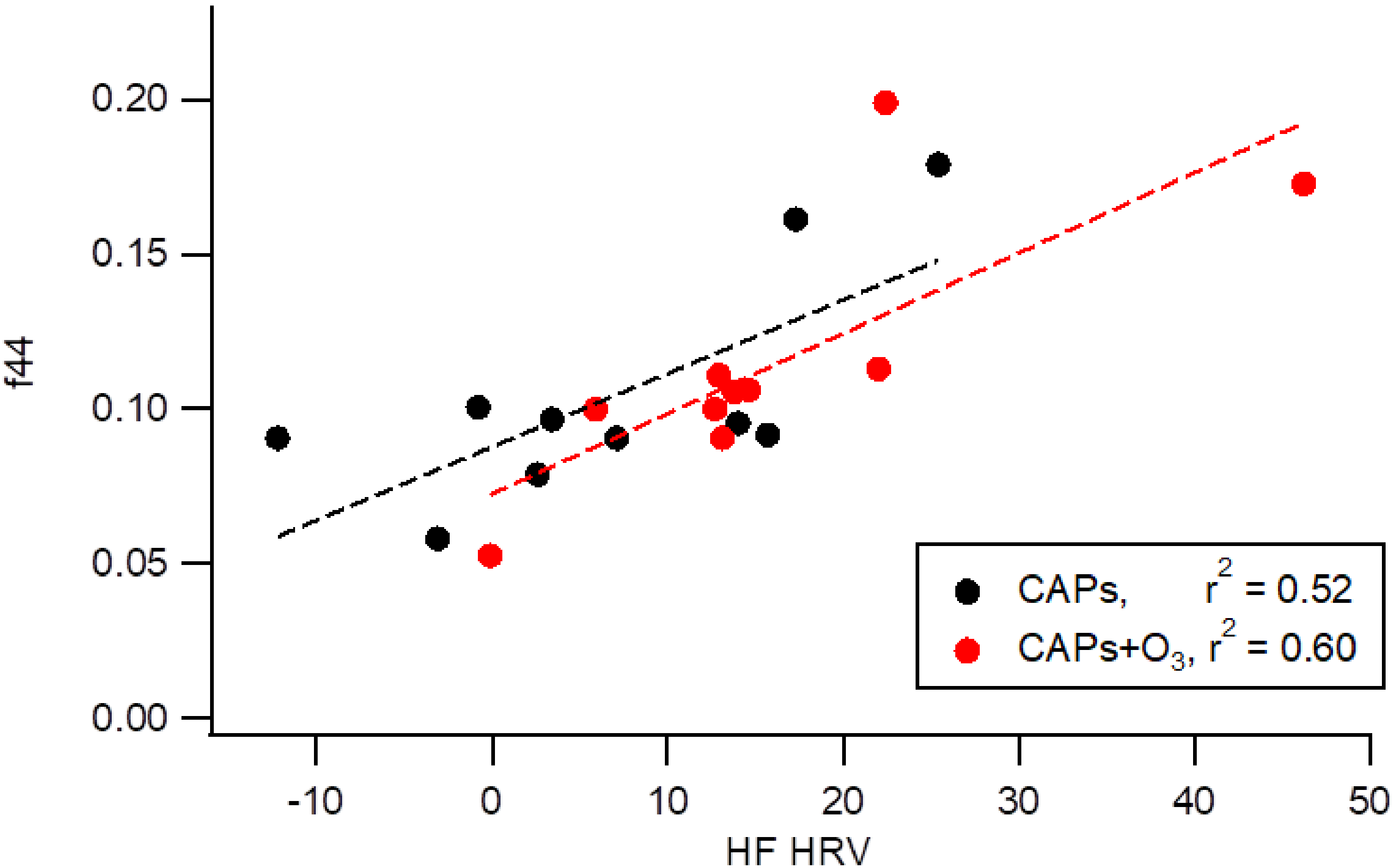
O:C ratio correlates with changes in HRV – O_3 Oxidation may affect biological activity



Increased concentrations of partially oxidized HCs (aldehydes) is associated with more adverse HRV

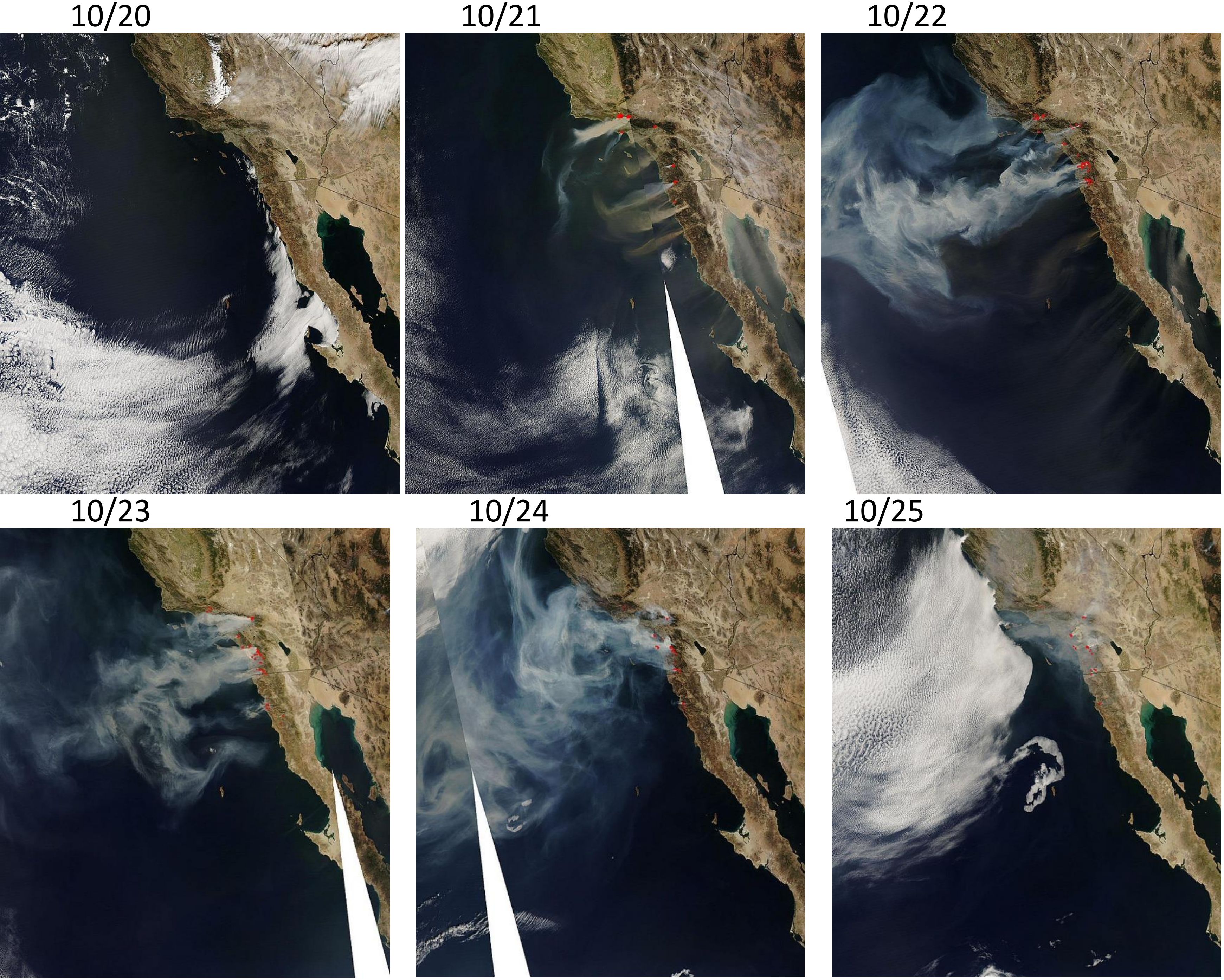


Increased concentrations of more oxidized HCs (organic acids) are associated with less adverse HRV



Our Mobile Systems Can Be Deployed to the Field to Help Understand Effects of Real World Exposures

- Case in point – Wildfires in Southern California
  - Rats exposed before, during and after the fires.
    - SHR
    - WKY
  - Cardiac Physiology Monitored During Exposures.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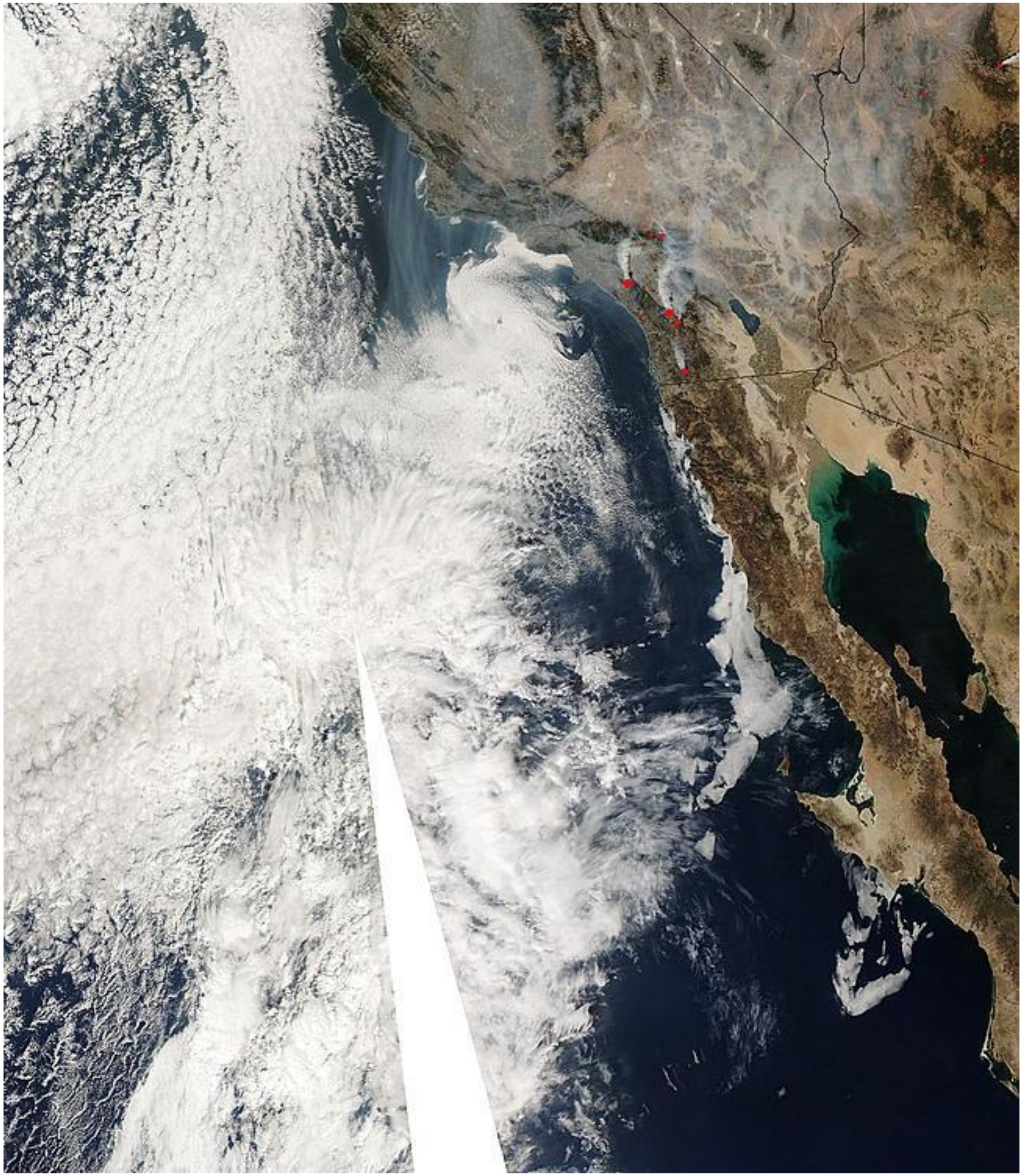


10/26

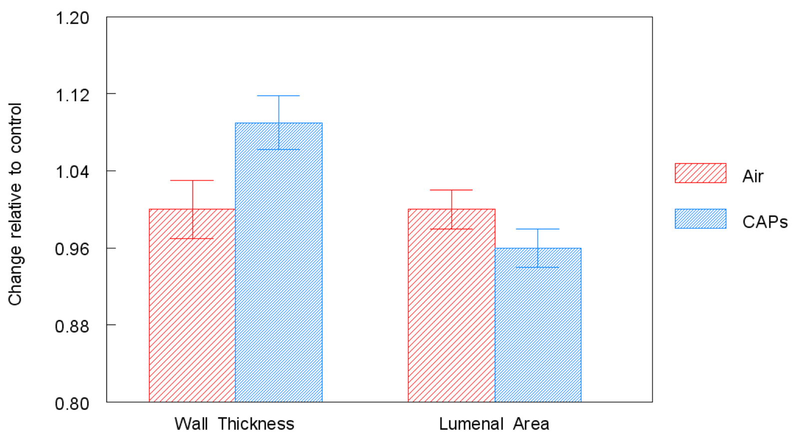
Chronic PM Exposure Leads to Different Effects in Susceptible Populations

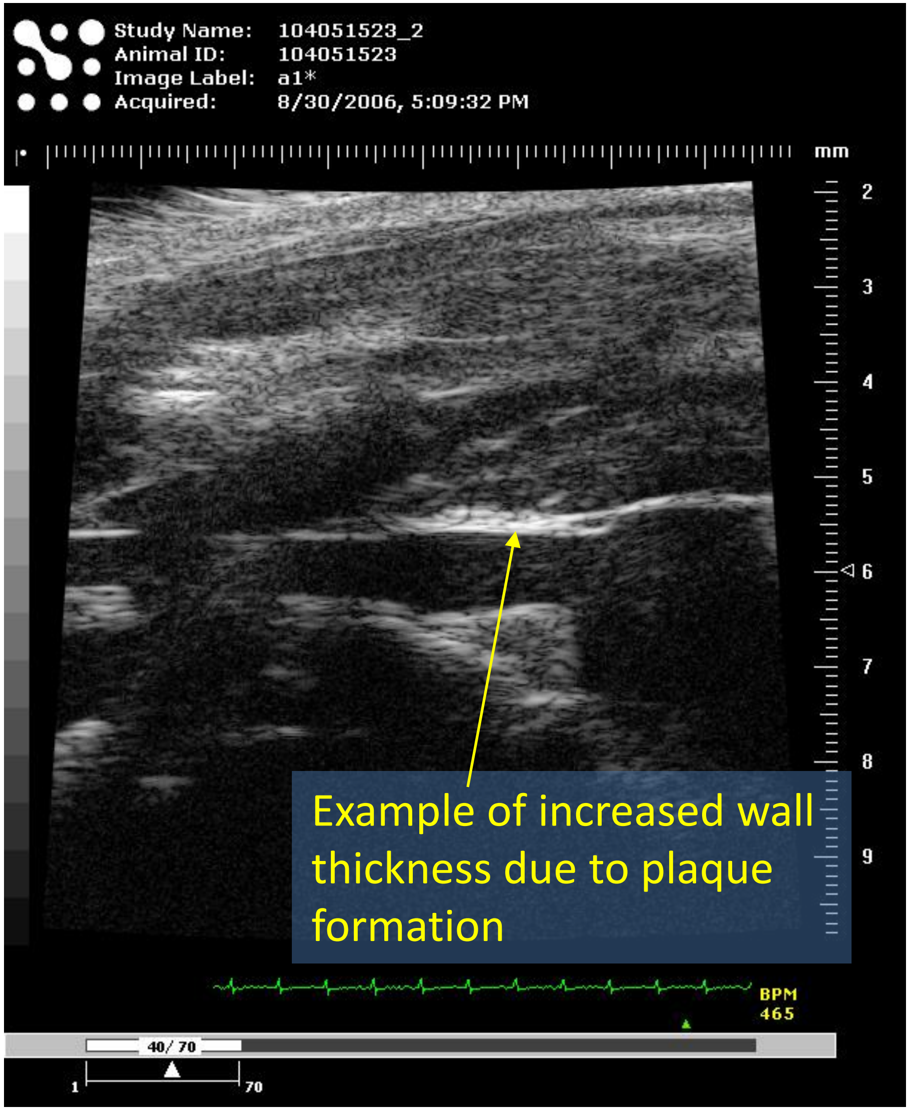
- Blood pressure increased to a greater extent in SHR exposed to CAPs than in SHR exposed to Air.
- WKY showed a more “exaggerated” diurnal pattern when exposed to CAPs than when exposed to Air suggesting that effects may be mediated through autonomic system imbalances.

After 8 weeks of Exposure to F PM in Riverside

- Ultrasonic imaging showed increased plaque development in PM-exposed mice.

Increased Plaque in mice after 2 months of CAPs Exposure

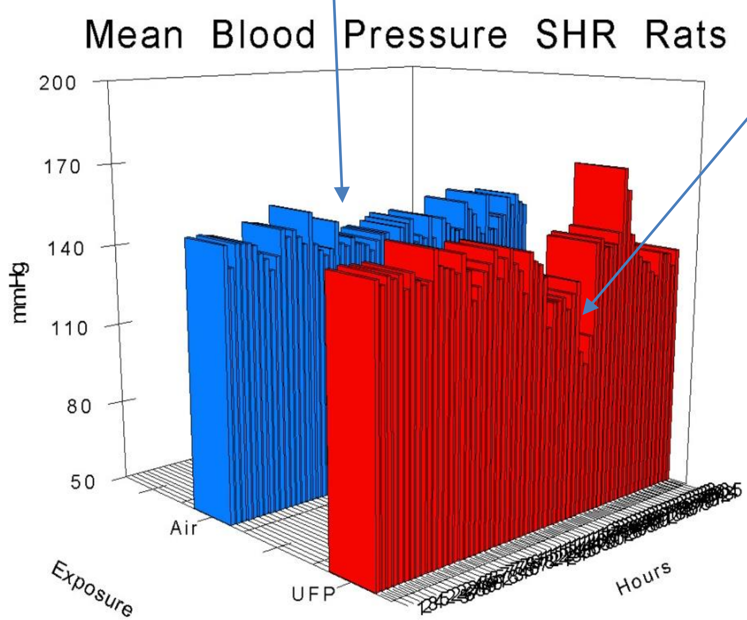


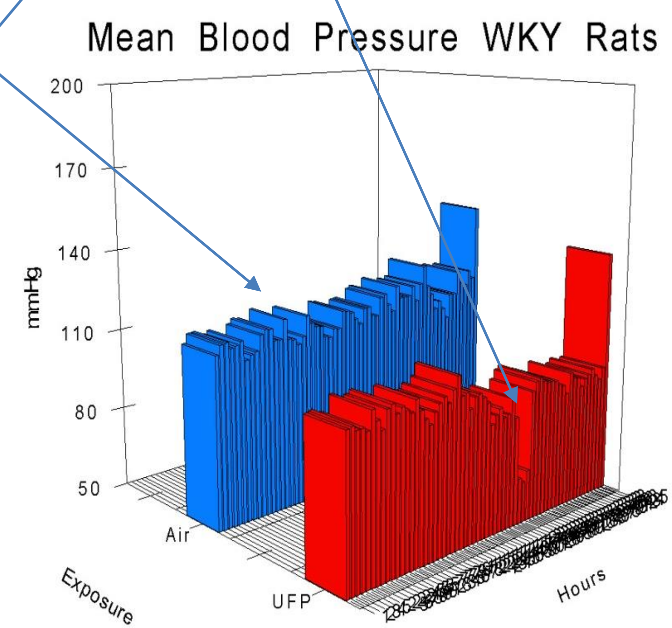


Blood Pressure

Blood Pressure Stable in Air-Exposed Rats

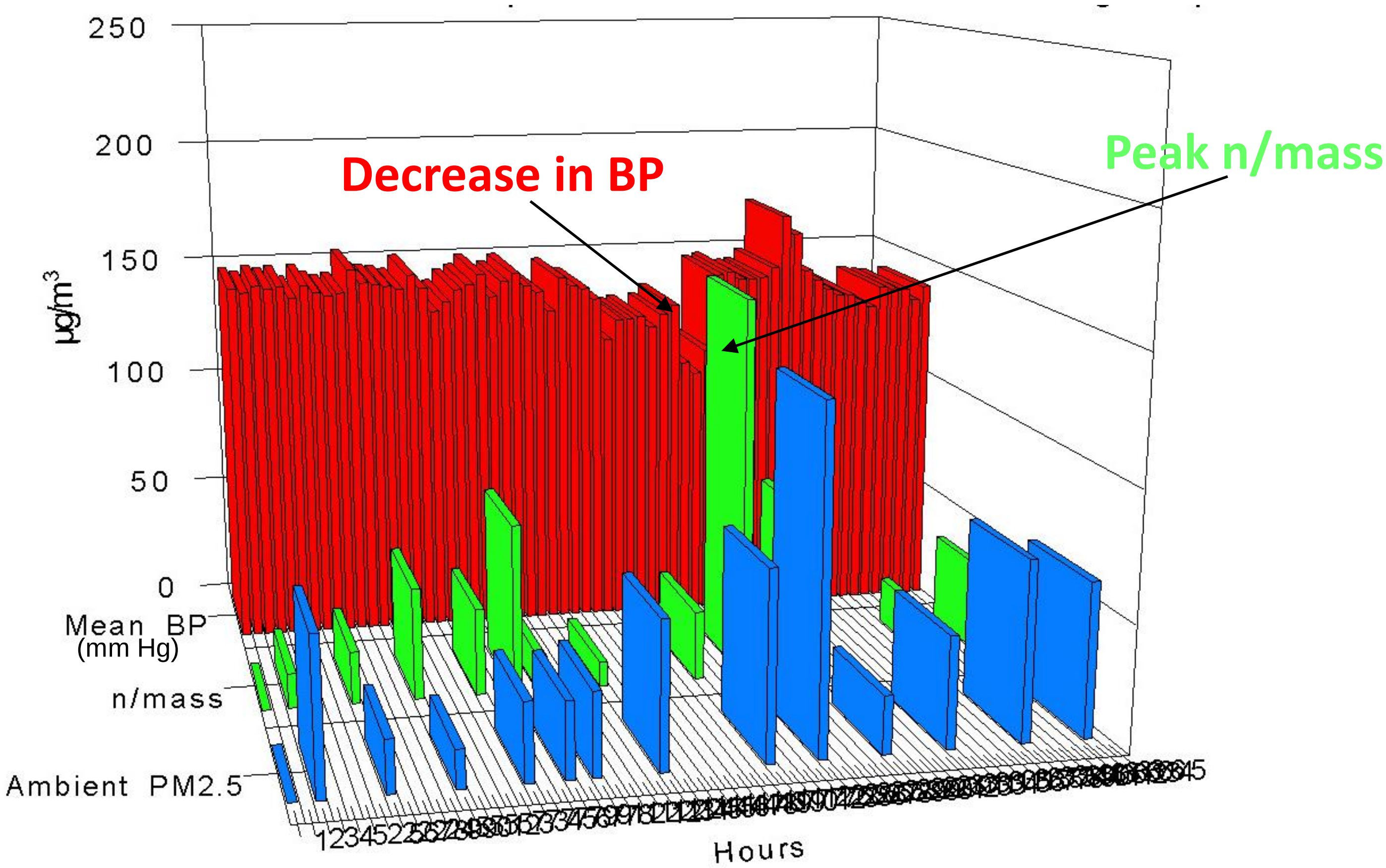
Significant Drop in Blood Pressure in UFP-exposed Rats During Fire





Results… (cont.)

Ambient $PM_{2.5}$ concentrations during exposures

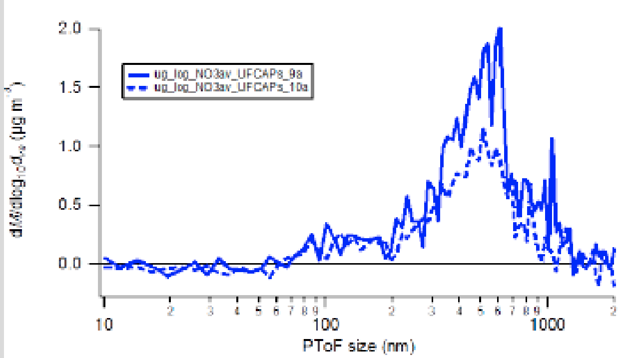


BP decreases with increase in number/mass ratio, suggesting a larger impact of UFP during fires

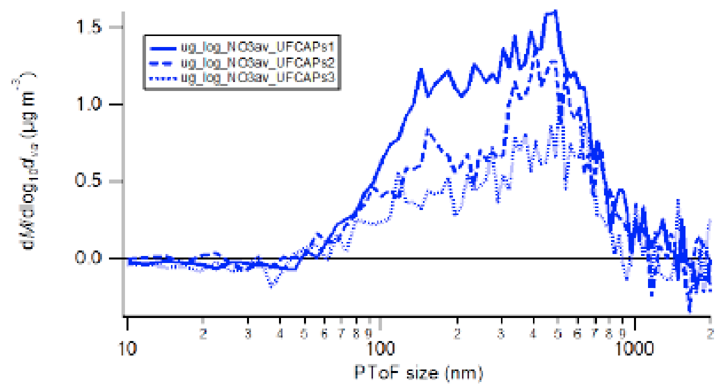
Additional Silverado Forest Fire Influence

Nitrate

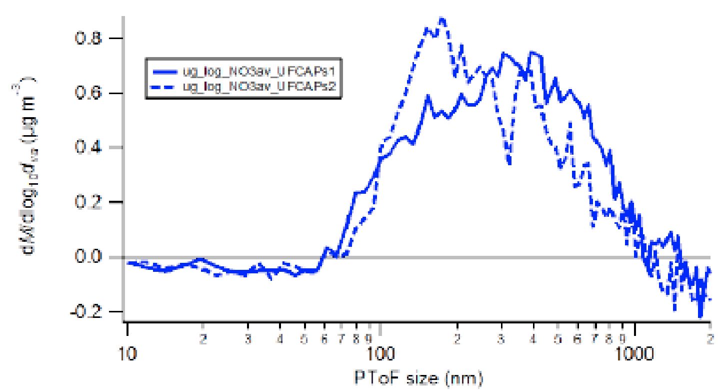
08/26/14 – before fire



09/15/14 – during fire



09/16/14 – during fire

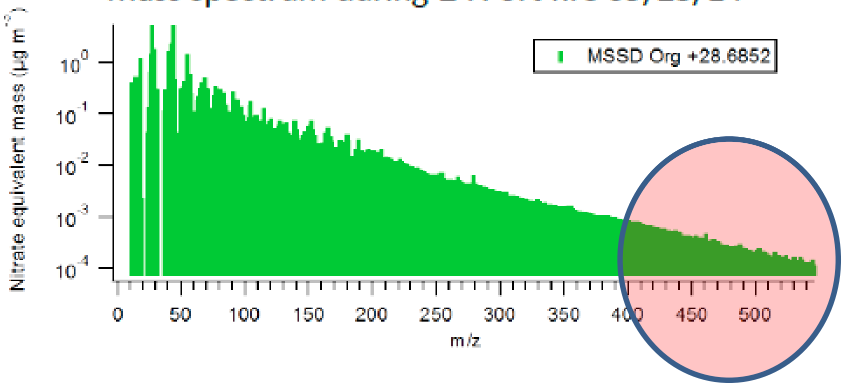


- More nitrate in smaller diameters during Silverado forest fire.
- Particulate nitrate increases during biomass burning periods, e.g. Behera and Balasubramanian (2014), Bi et al. (2011), and Qin and Prather (2006).

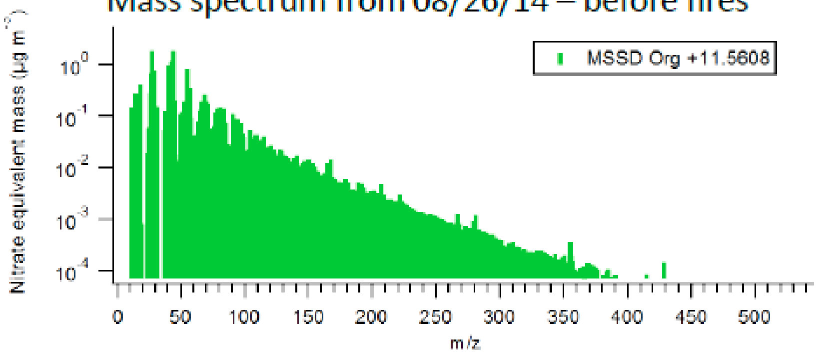
Port of L.A. Lumber Fire

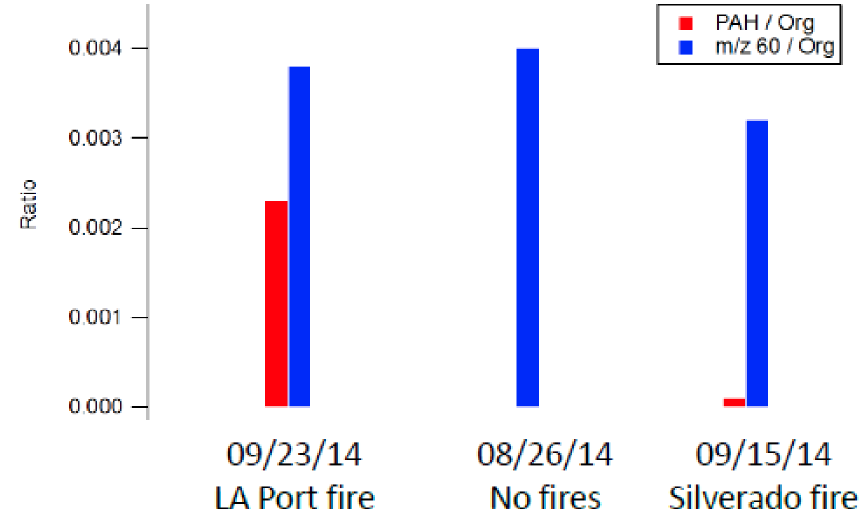
PAHs Possibly Present

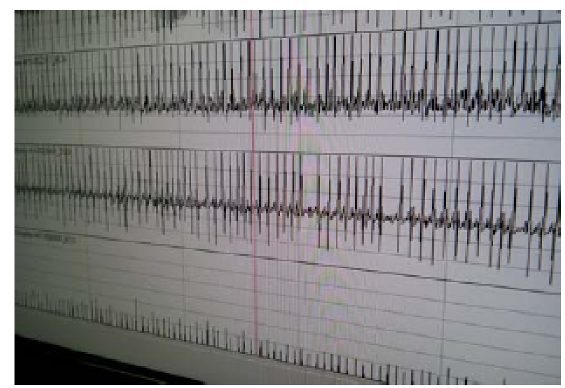
Mass spectrum during LA Port fire 09/23/14



Mass spectrum from 08/26/14 – before fires







- PAH are likely present in particles during LA port fire as suggested by intensity at m/z > 200 (Dzepina et al., 2007).
- For other sampling dates the Ratio PAH/Org is very low.
- Very little evidence of levoglucosan in particles; photochemical aging?

Conclusions

- Exposures to summer PM elicited significant changes in both heart rate-related and ischemia-related ECG parameters;
- The predominance of unoxidized or partially oxidized (carbonyls) HC in PM was associated with worsened cardiac function.
- Further oxidation (i.e. to organic acids) reduced the adverse biological effects.
- Organic constituents on the surface of particles may be responsible for making it more possible for particles to penetrate normal cell defenses and deliver toxic components to cell interiors.

Acknowledgements

- Research using the AMS and SMPS was through AirUCI and funded by the National Science Foundation.
- L.W. thanks Mike Ezell for SMPS measurements, Prof. Nizkorodov for loan of the CPC, and Dr. Veronique Perraud and Emily Bruns for helping move the AMS.

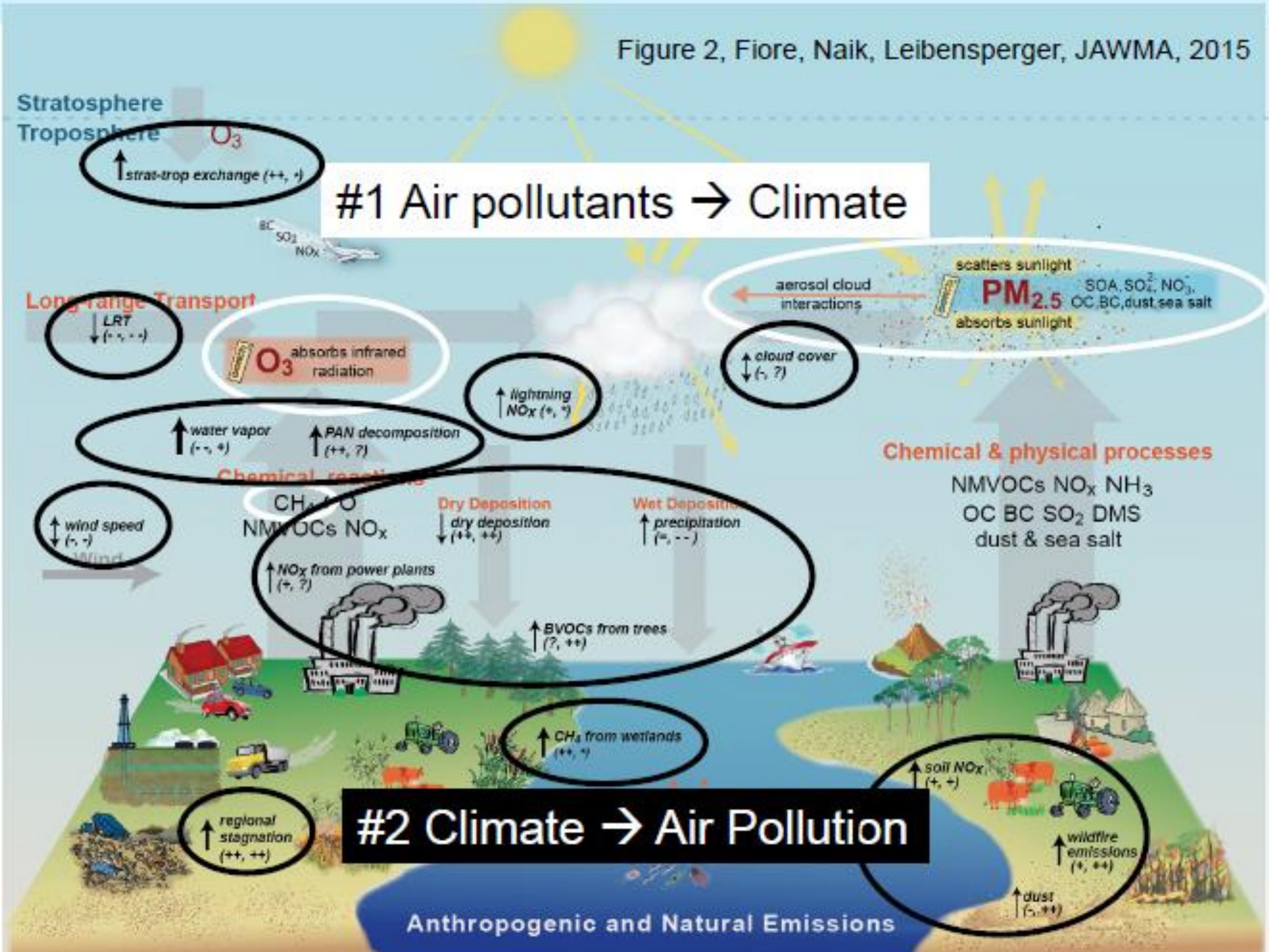
Moving the AMS is a group effort!

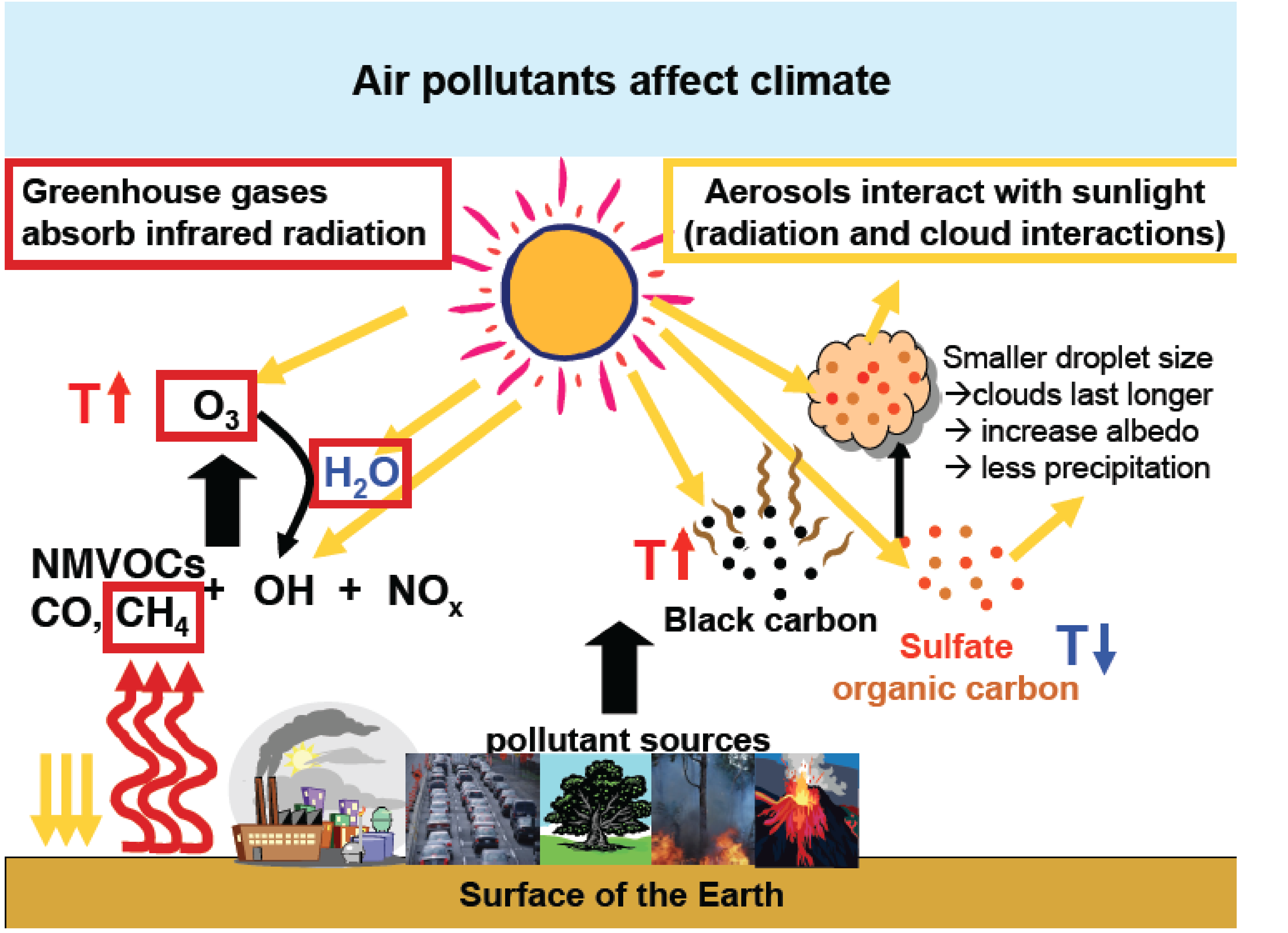
Health studies at are currently sponsored by the California Air Resources Board, the South Coast Air Quality Management District and the NIEHS

Collaborators

- Cardiovascular Project (ARB)
  - UCI/NYU collaborative project
    - Chen, Li, Xian (Ultrasound)
    - Meacher, Gookin, Salazar, Willett (ECG, Physiology, Arterial Plaque Measurements)
- Atherosclerosis Mechanisms
  - SCPC Project 2 (Nel, Araujo, Kleinman,Harkema, Sioutas)
- Direct Effects of PM on Heart
  - EPA STAR Grant
    - GSH-Kloner and Simkovitch
    - UCI- Kleinman, Willett, Gookin, Salazar,Meacher

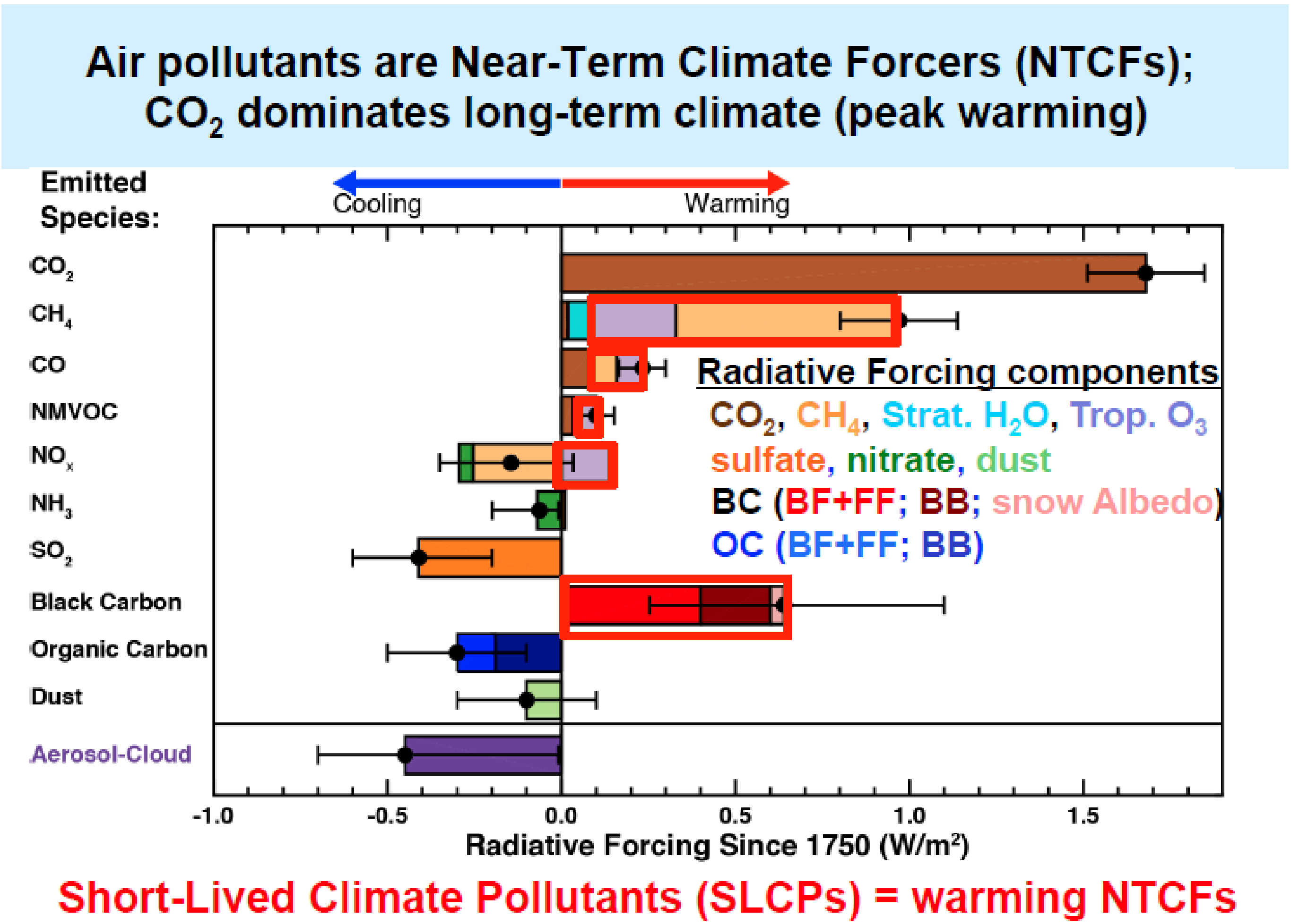
Summary schematic of air quality-climate connections





Air pollutants affect climate
Greenhouse gases absorb infrared radiation
Aerosols interact with sunlight (radiation and cloud interactions)
T↑
O_3
H_2O
Smaller droplet size
→clouds last longer
→ increase albedo
→ less precipitation
NMVOCs
CO, CH_4 + OH + NO_x
T↑
Black carbon
Sulfate
organic carbon
T↓
pollutant sources
Surface of the Earth

Globally averaged radiative forcing (RF) of climate from preindustrial (1750) to present (2011) of air pollutants and their precursor emissions.



Adapted by E. Leibensperger (SUNY Plattsburgh) from IPCC, 2013 for Fiore, Naik, Leibensperger, in press

O_3 correlates with surface temperature on daily to inter-annual time scales in polluted regions [e.g., Bloomer et al., 2009; Camalier et al., 2007; Cardelino and Chameides, 1990; Clark and Karl, 1982; Korsog and Wolff, 1991]

Observations at U.S. EPA CASTNet site Penn State, PA 41N, 78W, 378m

July mean MDA8 O_3 and July mean daily maximum temperature

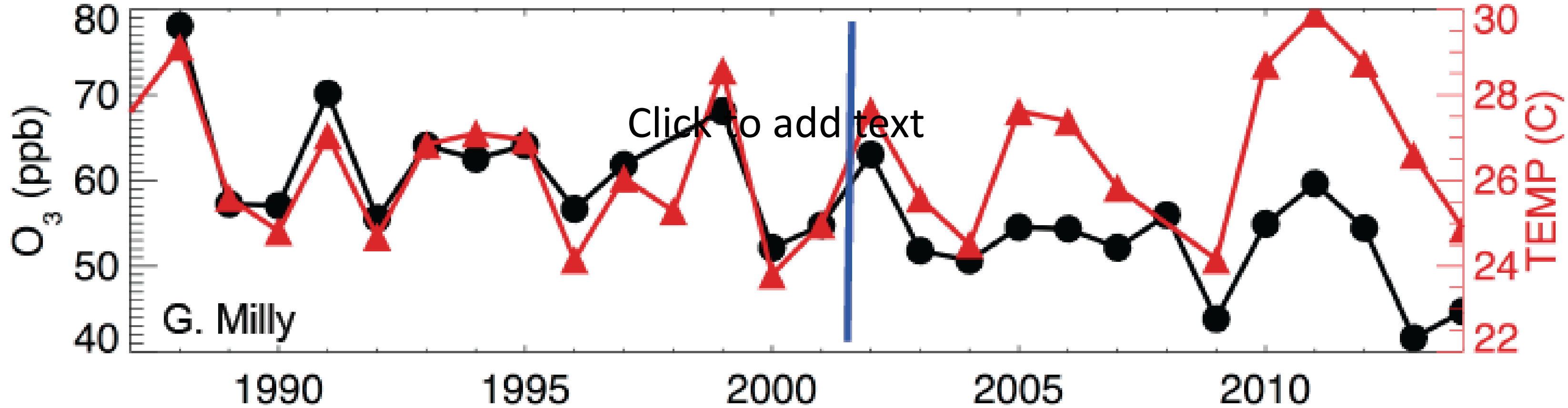


Figure 6a of Fiore, Naik, Leibensperger, JAWMA, 2015

NOx Control Imposed

→ Implies that changes in climate (via regional air pollution meteorology) will influence air quality

→ Downward trend in O_3 as EUS NO_x emission controls are implemented

How and why might extreme air pollution events change?

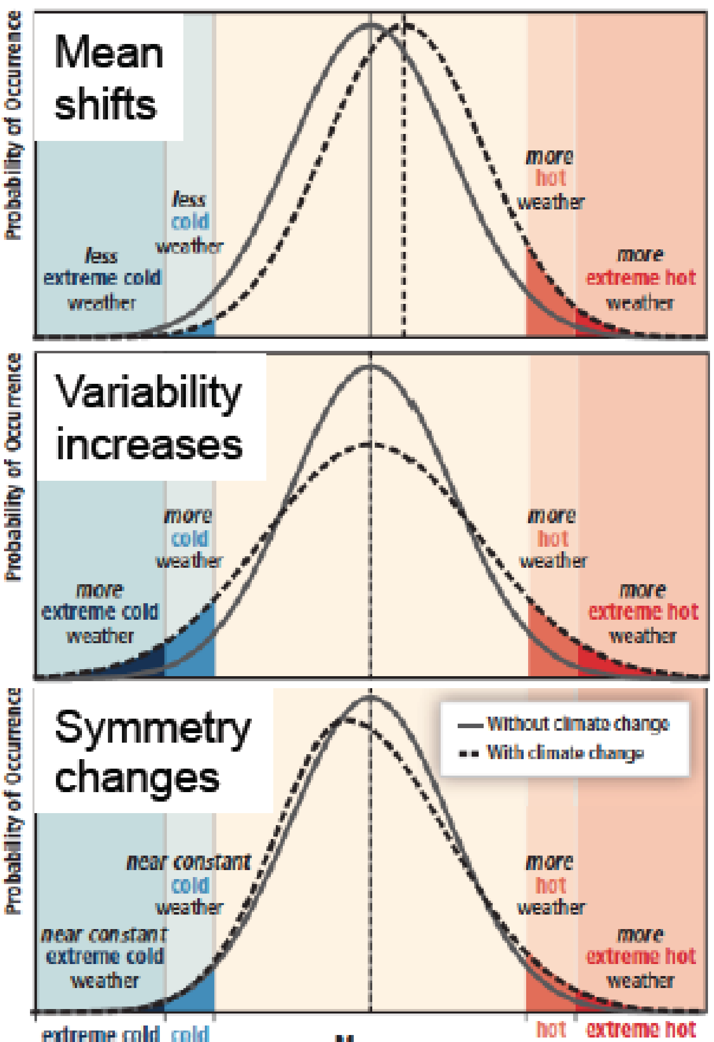
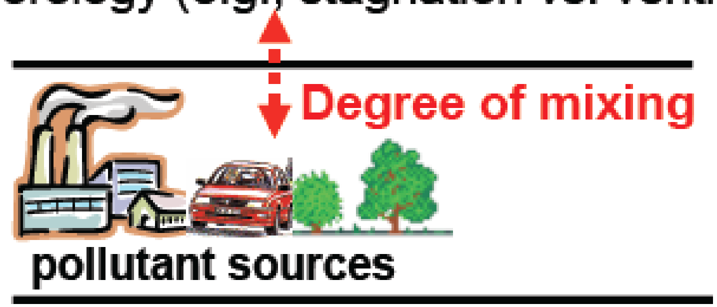


Figure SPM.3, IPCC SREX 2012
http://ipcc-wg2.gov/SREX/

→ **Need to understand how different processes influence the distribution**

- Meteorology (e.g., stagnation vs. ventilation)



- Feedbacks (Emis, Chem, Dep)

- Changing global emissions (baseline)
  → Shift in mean?
- Changing regional emissions (episodes)
  → Change in symmetry?

Questions and Discussion

Advisory Council Deliberation: Beyond Attainment – Prioritization for Local Health Impacts

Advisory Council Deliberation

AGENDA: 9

- The Air District has initiated a process to better understand PM and its effects on local communities, so as to better protect those communities.
- An important component of this effort is the ability to prioritize efforts and actions based on maximizing impact on human health
- Public access to District deliberations
- Council convening and advisory function

AGENDA: 10

Advisory Council Terms

Advisory Council Terms

- Per the Health and Safety Code Section 40263, each Council Member shall hold office for a term of two years and until the appointment and qualification of his/her successor.