Health Canada Santé Canada

Healthy Environments and Consumer Safety

Santé environnementale et sécurité des consommateurs

Health Effects of Air Pollution

CASA Nitrogen Symposium

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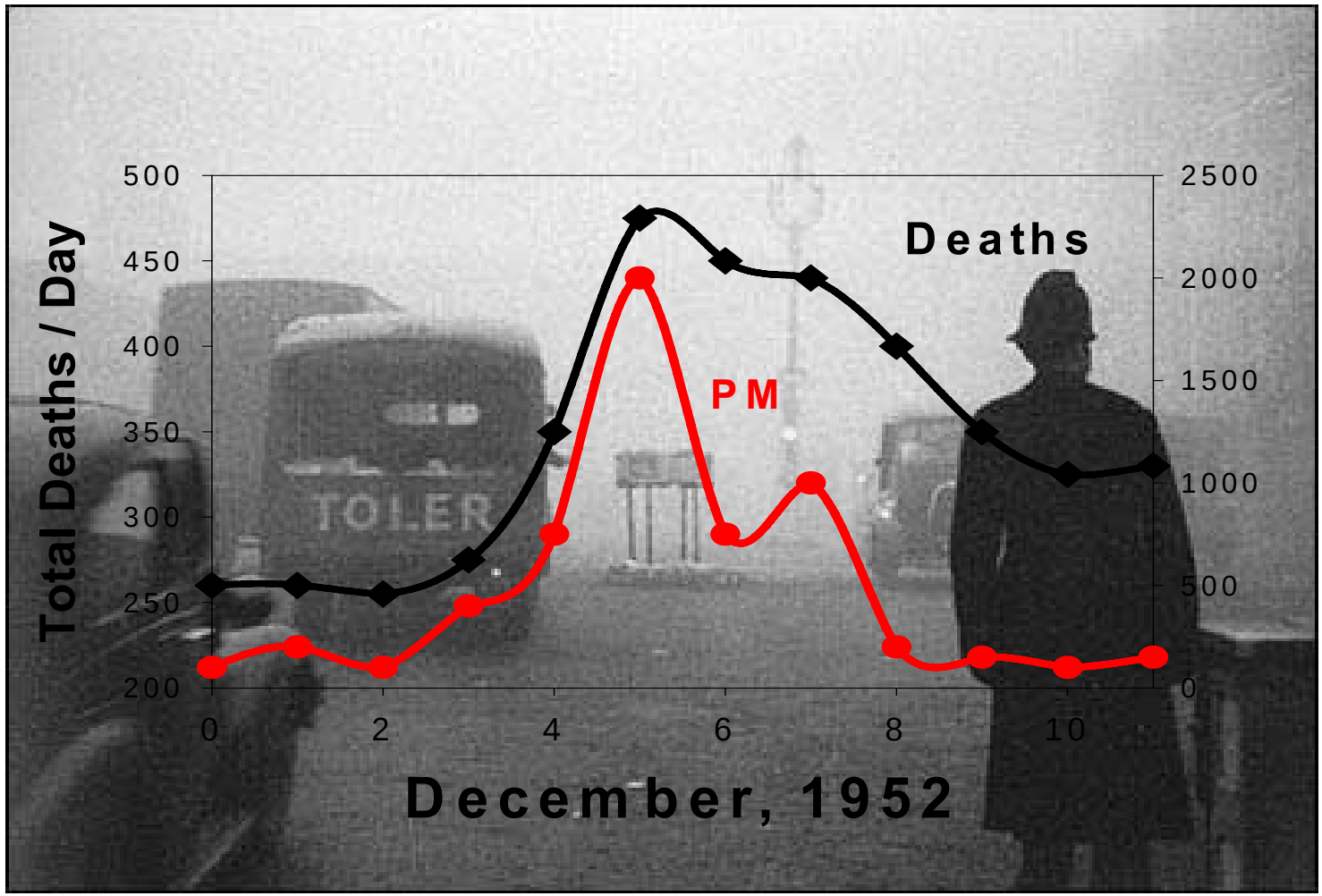
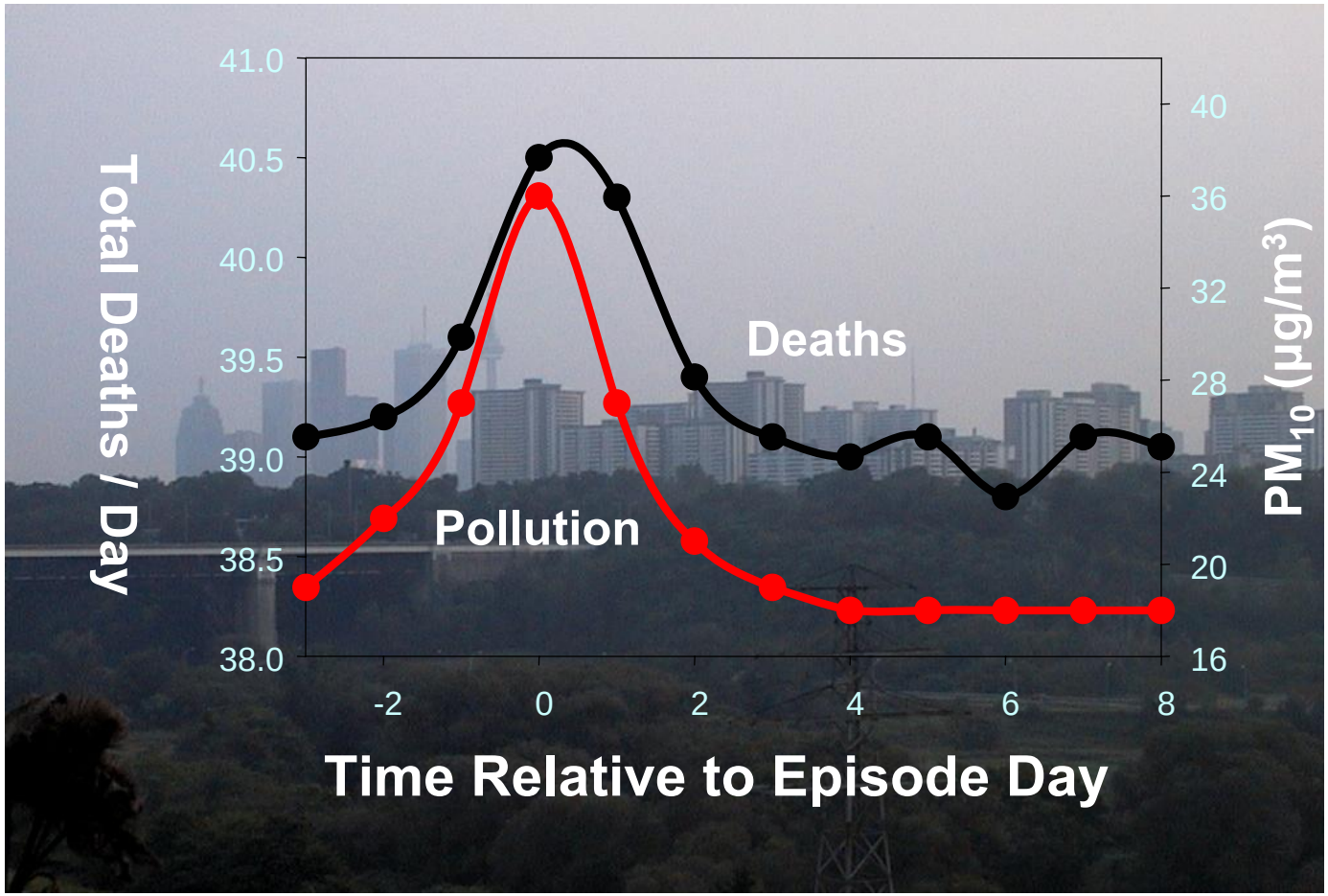
Why has air quality become a priority?

- Effects occurring at ambient levels
- Involuntary nature of exposure
- No apparent population threshold for effects
- Sensitive groups affected
- Suspect air pollution creates disease
- Magnitude of the health effect
    - Ranks high in WHO burden of disease report

Air Pollution And Mortality: The London Fog



Air Pollution And Mortality: Toronto



We no longer have to deal with events like the London Fog; but simple smoggy days have consequences for human health.

Toronto: a typical summer smoggy day

PM/Ozone around the world

- Canada
  - $PM_{2.5}$: 30 µg/m^3 (24 hr ave.)
    - Evidence of mortality, COPD, asthma, many other
  - Ozone: 65 ppb (8 hr ave.)
    - Evidence of mortality, hospital visits and respiratory effects
- U.S.
  - $PM_{2.5}$ (24 hr ave.) : 65 µg/m^3 *reduced to* 35 µg/m^3
  - $PM_{2.5}$ (annual) : 15 µg/m^3 *remains the same*
    - Evidence of short-term acute effects (mortality etc.) and especially chronic mortality effects.
  - Ozone 80 ppb (8 hr ave.)
    - Hospital admissions, children, permanent lung injury
    - Under revision (mortality studies)
    - CASAC review strongly recommends lowering this to 60-70 ppb

PM/Ozone around the world

- Australia
  - $PM_{2.5}$ (24 hr ave.): 25 µg/m^3
  - $PM_{2.5}$ (annual): 8 µg/m^3
    - Australian short-term acute mortality/h.a.; U.S. cohort studies for chronic effects
    - Lack of apparent population thresholds
  - Ozone: 100 ppb (1 hr ave.)
  - Ozone: 80 ppb (4 hr ave.) (*Sydney)
    - Evidence of respiratory distress, athletic performance, asthma

- New Zealand (interim)
  - PM_{10} (24 hr ave.): 50 µg/m^3
    - Mortality, respiratory disease, asthma, lack of apparent population thresholds.
  - Ozone (8 hr ave.): 50 ppb
    - Persons with cardiovascular disease, elderly, asthmatics, exercisers

PM/Ozone around the world

- Thailand
  - PM_{10}(24 hr ave.): 120 µg/m^3
  - $PM_{2.5}$ (annual): 50 µg/m^3
    - Bangkok daily mortality epidemiology;
    - evidence of chronic effects + U.S. ACS
    - Lack of apparent population thresholds
  - Ozone: 100 ppb (1 hr ave.)
    - Bangkok daily respiratory admissions study and worldwide literature
- India
  - PM_{10} (24 hr ave.): 150 µg/m^3
  - $PM_{2.5}$ (annual): 100 µg/m^3
    - Based on WHO: epidemiology of short-term and chronic mortality & time-series epidemiology in Indian cities

PM/Ozone around the world

- World Health Organization
  - $PM_{2.5}$ (24 hr ave.): 25 µg/m^3
  - $PM_{2.5}$ (annual) : 10 µg/m^3
    - Main focus is on U.S. ACS chronic mortality study and its reanalyses
    - Lack of apparent population threshold
    - Interim annual targets set at 35/25/15 (15%, 9% and 3% excess mortality)
    - Interim daily targets set at 75/50/37.5 (5%, 2.5% and 1.25% excess mortality)
    - Main focus is on chronic effects
    - Recognizes effects will occur at Guideline levels
  - Ozone: (8 hr ave.): 50 ppb (100 µg/m^3)
    - Respiratory effects, hospital admissions, mortality (1-2%)

Chronic Exposure To PM & Mortality

- ACS 151 cities, 1.2 million people, followed 20 yr.
  - risk = 17% increase in deaths per 24.5 μg/m^3 in yearly fine PM
  - lung cancer > heart-lung > all-cause
  - life-shortening in the range of 8-18 months
  - Adjusting for socioecon., occup., med care, weather, etc.:
    - risk of fine PM remains; educational status a modifier
  - L.A. intra-urban analysis
    - risk increases by ~ 3-fold
- Harvard 6-city mortality study re-analysis
  - Original ~15 year study extended by 8 years
  - Lung cancer = cardio vascular > all-cause
  - Mortality rates have dropped with lower air pollution

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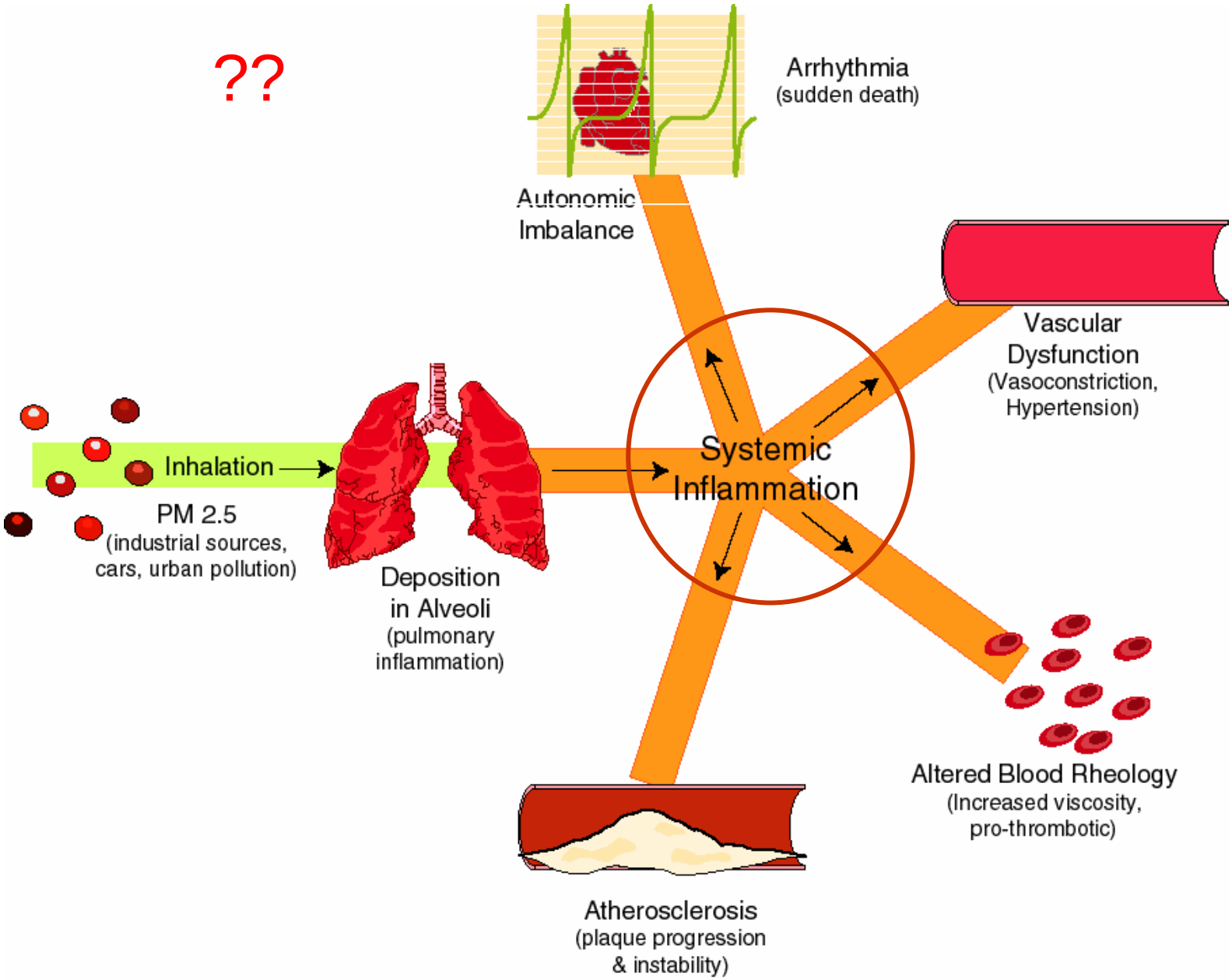
Arrhythmia
(sudden death)
Autonomic
Imbalance
Vascular
Dysfunction
(Vasoconstriction,
Hypertension)
Systemic
Inflammation
Inhalation
PM 2.5
(industrial sources,
cars, urban pollution)
Deposition
in Alveoli
(pulmonary
inflammation)
Altered Blood Rheology
(Increased viscosity,
pro-thrombotic)
Atherosclerosis
(plaque progression
& instability)

Atherosclerosis: Epidemiology

Ambient Air Pollution and Atherosclerosis in Los Angeles

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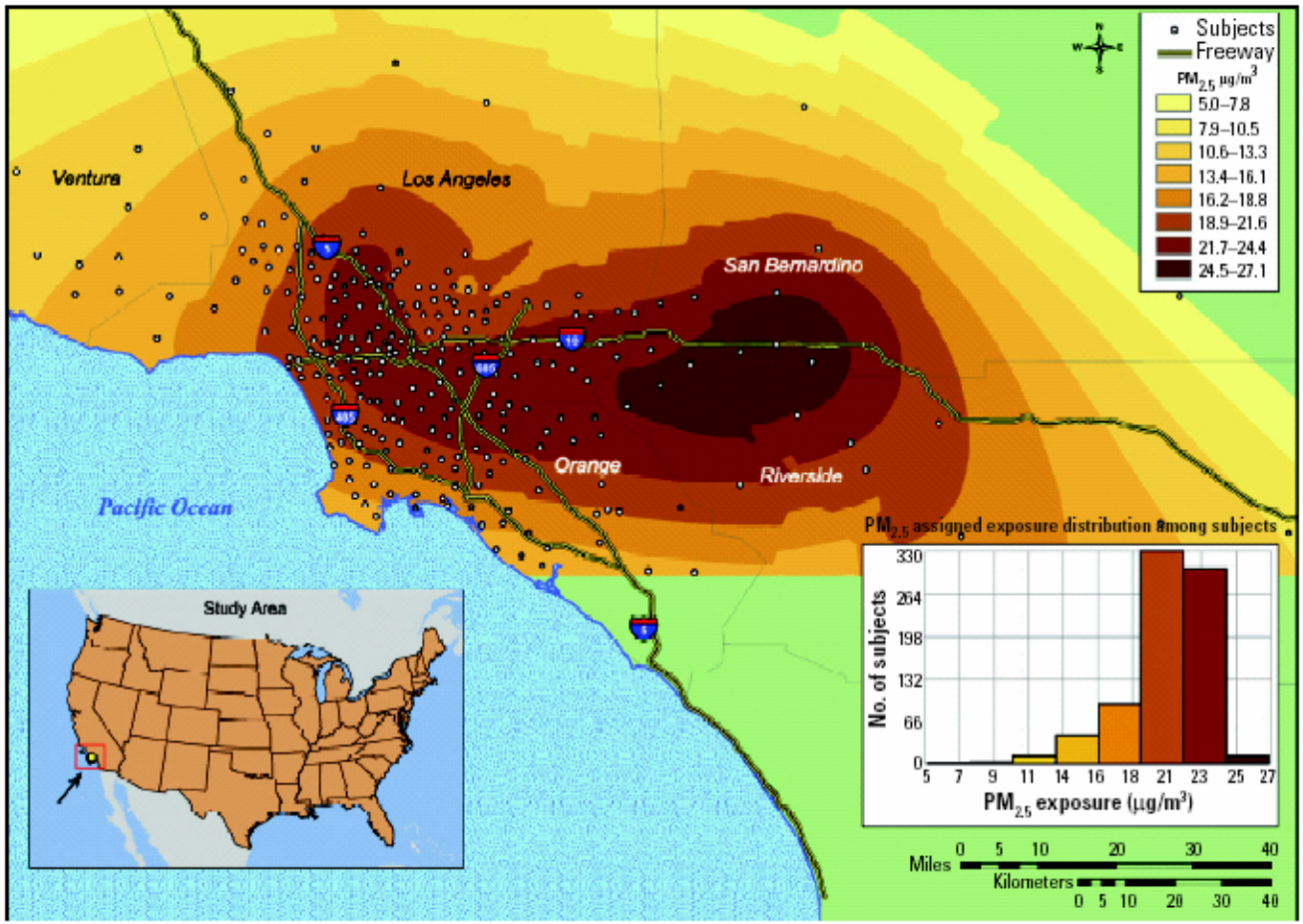


Figure 1. ZIP code locations of the study population geocoded on the $PM_{2.5}$ surface, modeled with 2000 $PM_{2.5}$ data, and distribution of individually assigned concentrations.

- Used data from two clinical trials on atherosclerosis prevention
- Mapped study subjects to $PM_{2.5}$ exposure
- Exposure was associated with atherosclerosis in carotid artery

  (4% per 10 ug/m^3)

Source: Künzli et al., 2005

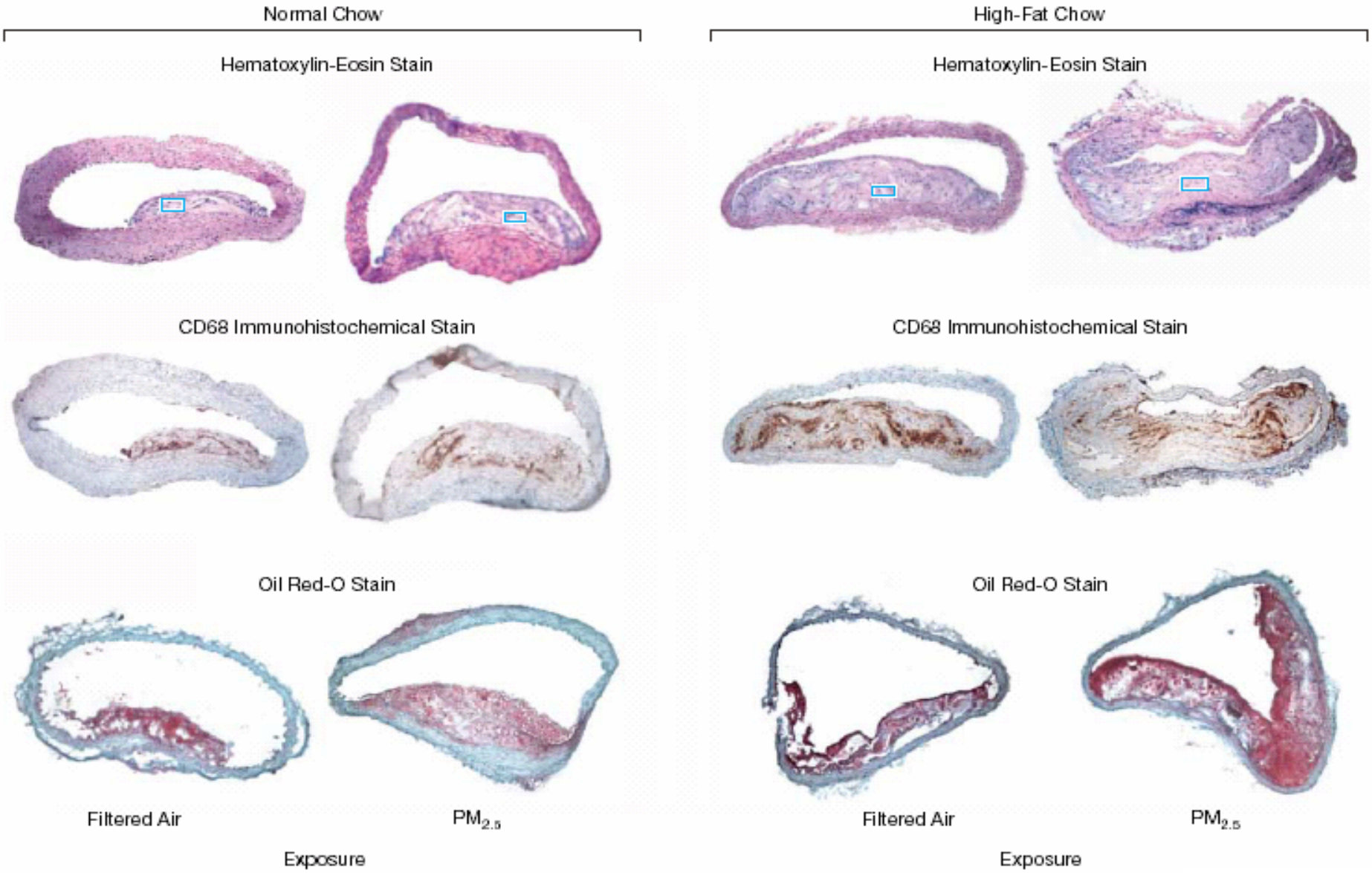
Mice developed clinically significant disease states after exposure to PM

- NYU Study
  - CAPs and compromised mice: 6 month exposures
  - Cardiac rhythms: daily and chronic
  - Atherosclerosis
  - Coronary artery disease
  - Brain lesions (substantia nigra)
  - Gene expression (circadian rhythm: suggestive)
  - Sources?: fuel oil>coal>traffic>soil
  - *Overall: clinically significant disease states induced by PM*

Atherosclerosis: Toxicology

- Mice developed clinically significant disease states after exposure to ambient PM
- PM at current levels has the potential to increase arterial plaque deposits (chronic)
- PM at current levels increases arterial inflammation (acute)
- PM appears to interact with a fatty diet to produce an enhanced effect
- PM induces the production of a wide variety of reactive oxygen species
- PM appears to play a role in the inactivation of proteins needed to maintain vascular health

Figure 2. Representative Photomicrographs of Hematoxylin-Eosin Staining and CD68 Immunohistochemical Staining of Abdominal Aortic Sections, and Oil Red-O Staining of Aortic Arch Sections



$PM_{2.5}$ indicates concentrated ambient particles of less than 2.5 μm. CD68 immunohistochemical staining from abdominal aortic sections wer Standard indirect biotin-avidin immunohistochemical analysis was performed. Brown chromogen indicates positive staining and hematoxylin staining. Original magnification ×100. Squares represent where the detection of 3-nitrotyrosine and inducible nitric oxide synthase were observed (see Figure 3).

Source: Sun et al., 2005

Chronic effects of ozone

Children's Health Study in California (1992-2002)

- Yearly within-community variability in ozone associated with bronchitic symptoms in asthmatic children
- Exercising children in high pollution communities and instigation of asthma
- Ozone associated with increased school absenteeism
- Ozone *does not* appear directly associated with chronic deficits in the growth of lung function in adolescents aged 10-18 years
  - But other pollutants were (permanent)
  - Moving away from high pollution: function recovered (and *vice versa)*

Ongoing Lines of Enquiry

- Genetics and ozone effects
  - Mice to humans
- Ozone mortality
  - EPA meta-analyses: EPA review
- Reproductive and inherited effects
  - *In utero* effects, Hamilton cage study
- Stroke (ischemic)
  - London, Edmonton, Sydney, Europe, China
- Diabetes
  - Montreal, Toronto, U.S. cities
- Etc.

So that brings us to NO_2….

- Significant signals in acute mortality studies
- Generally regarded as a signal for traffic
  - Best predictor of exposure to roadways
  - Near equivalent to traffic counts
- Along with ozone and PM, driver in Canadian studies
  - AQI, AHI

… NO_2….

- California's proposed new Standards
  - 180 ppb (1hr); 30 ppb (annual)
  - Based on respiratory effects (asthma, infections), concern for vulnerable pops., lung growth and damage, precautionary with epidemiology
- APHEA mortality study
  - 30 European cities
  - Significant association with mortality and cause-specific mortality
  - Results "consistent with an independent effect, but role as a surrogate cannot be ruled out"

In Sum

- PM story gets more complex and interesting
  - Sources, components
- Ozone story appears largely written
  - Emphasis has moved to PM
- NO_2 is sneaking in
  - Generally consistent appearance in epidemiology
  - Is it direct effects or surrogacy?
  - Risk management simple; but effective?