Health Risks and Air Quality:
Fine Particulate Matter and Ozone

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Atmospheric Change and Health: Research Agenda

Ecosystem

- Climate change
- Strato ozone depletion
- Acid deposition
- POPs transport

Airshed

- Ambient air quality
- Air toxics
- Allergens

Built

- Indoor air quality
- Occupational exposure

Atmospheric Change

Industrialization
Technological choices
Economic choices →
Population

Greenhouse gases	→	Climate change
O_3 depleting chemicals released	→	↑ incident UV radiation
Acidifying pollutants	→	Acid deposition
Persistent organic pollutants	→	Long-range transport of POPS
Air toxics	→	Air toxics
Ambient air pollutants	→	Ambient air pollution
Air quality in the built environment	→	Indoor air quality issues
Airborne emissions in the workplace	→	Occupational respiratory hazards

Human Health Implications

Mediated by toxic effects

- ambient air pollution
- air toxics
- IAQ
  - sick building
  - moulds, bioaerosols
- occupational exposures
- noxious odour effects

Mediated by ecosystem effects

- climate change
- strato ozone depletion
- effects on food production

Socially mediated effects

- risk perception
- odour/nuisance

Progress has Social Drivers

Lead in gasoline	1986 → 1995	Children's health
Acid deposition	1990's	Northeastern lakes
Fine particulate air pollution, ozone	1999	Relative risk → Attributable risk
Climate change	2006	Al Gore
Air toxics (e.g. WISSA)	2007	Community "outrage" and conflict
NAAQS for ozone	2008	Asthma concern

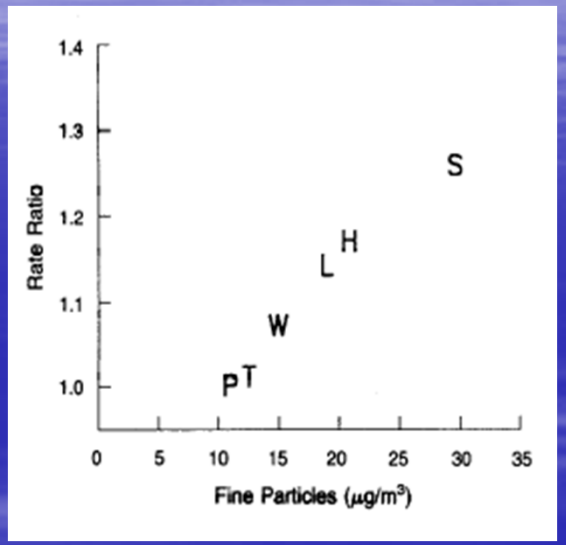
Air Quality and Health

- There is no “air pollution disease”
  - Health effects take place against a background of same outcomes
  - Statistical excess is the marker
- Air pollutants covary
  - Track each other so closely that separation is difficult
  - Averaging and regression: covariance
- Synoptic weather pattern effects (heat, humidity)

John R. Goldsmith, founder of environmental epidemiology

An Unsuspected Effect

- CHESS studies and earlier efforts to document health effects of particulate air pollution.
- Some false alarms (e.g. LA lung cancer study)
- General consensus until 1990 that effects were minor.
- Total suspended particulates (TSP) most common measure.
  - Particle distribution observed (Junke)
  - PM10 introduced: effect observed!
  - PM2.5 followed: more effect!
  - PM1.0 followed: ultrafines!

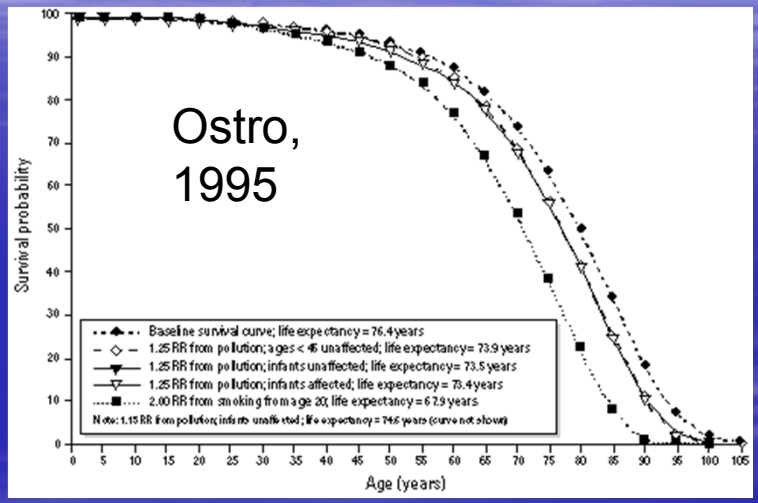


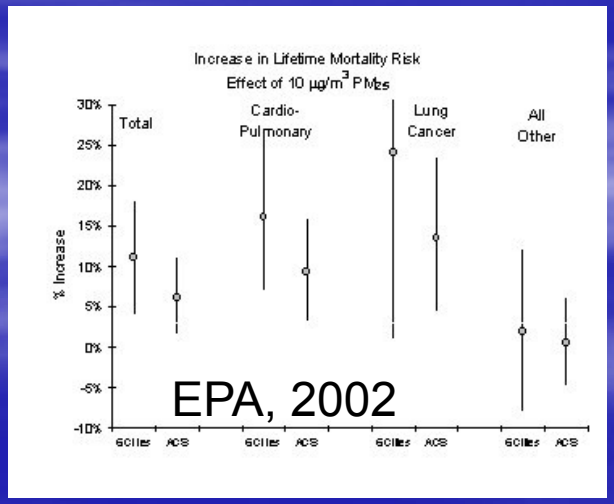
Three Paradigm Shifts:

1. Less mass can be *more* toxic.
2. Size as a marker for species
3. How to measure mortality

Compelling Calculations

- Risk estimates
  - Conventional relative risk (RR) was not persuasive
  - Survival was more dramatic
  - Attributable risk fraction (%)
  - Attributable risk (pure number)
- Natural Resources Defense Council took it to its logical conclusion: 64,000/year





“I can see clearly now….”

The fact that we can see these effects now because:

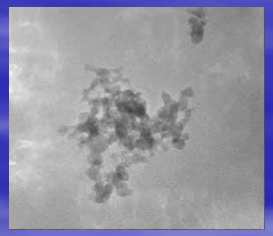


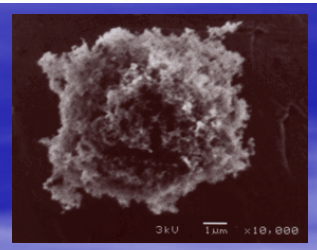
- Improved methods in epidemiology
  - Epidemiology as “Big Science”
  - Large data sets, time series
  - Attributable risk v. relative risk
  - We now know what to look for and we did not 60 years ago.
- Improved technology for exposure assessment
- Improving ambient air pollution levels in response to the earlier response to NAAQS has stripped away other health effects
- Work has undergone unmatched validation, confirmatory replication, stringent review, and critical analysis.

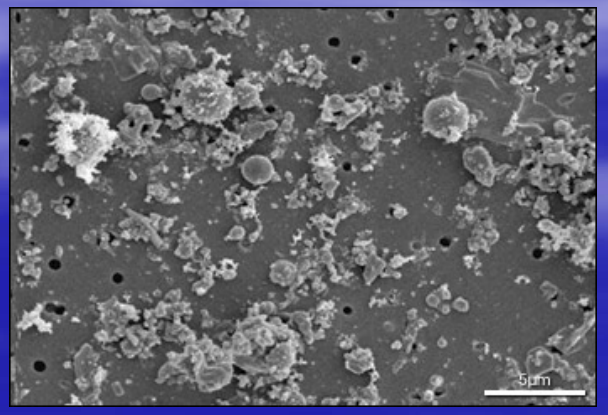
The second generation

The Discovery of Fine Particulate Matter

- Particulate matter ≤ 2.5 μm
- Small mass, huge surface area
- Produced by
  - Diesel, primarily
  - Sulfate, nitrate aggregation
- Composition: C, S, N, M
  - Variable seasonally
  - Metals content small but highly significant

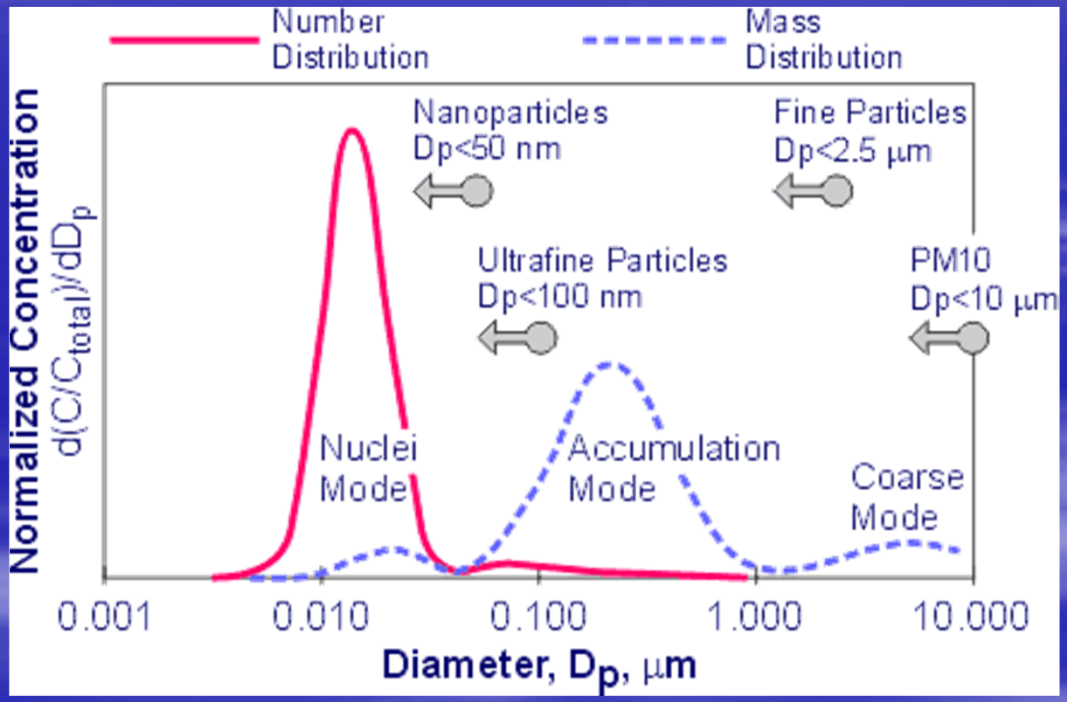






Three Populations of Particles

- Define cut size of particles
- Coarse mode = PM10, includes all particles <10 μg
  - Direct emissions
  - Crustal origin
- Fine mode = $PM_{2.5}$, includes ultrafines (nuclei mode)



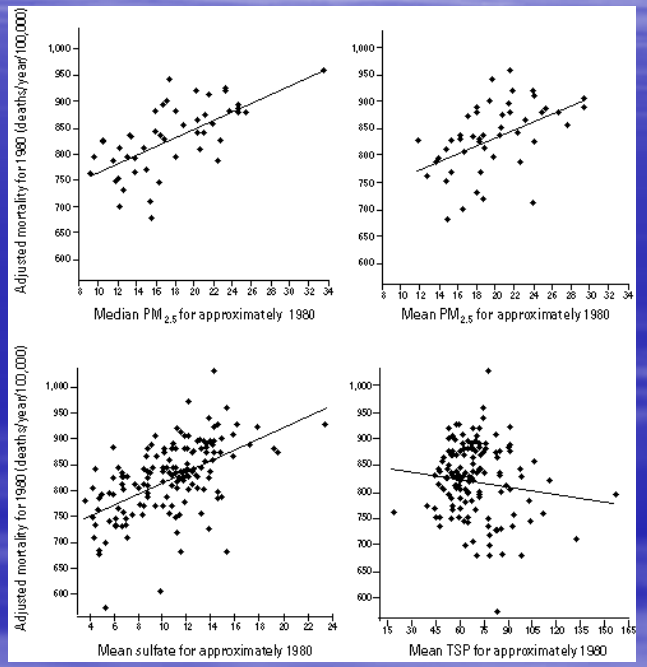
Health and Fine Particulate Matter

Epidemiological patterns

- Relative risk on order of 1.03 to 1.15
- Attributable risk profound: thousands of deaths in major cities

Characteristics

- Effect confined to fine particulate matter (<2.5 μm)
- Implies great potency for tiny mass
- No threshold
- Susceptible populations mostly elderly
- Primarily cardiovascular mortality
- Secondarily pulmonary
- Harvesting



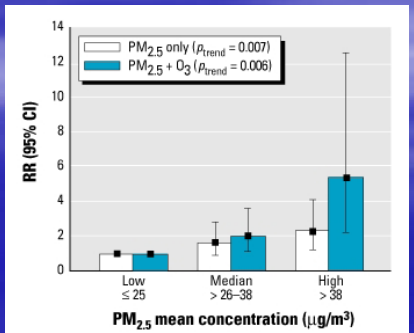
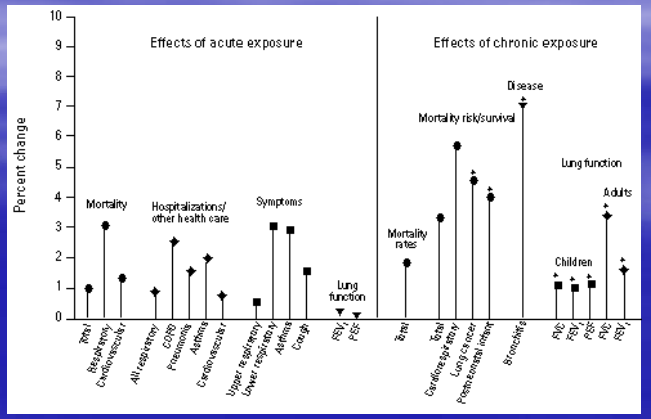
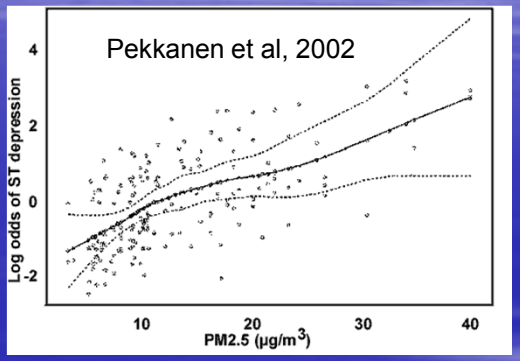


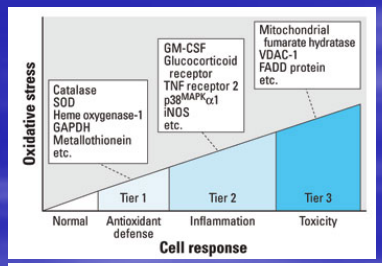
Figure 3. RR of fatal CHD and tertiles of $PM_{2.5}$ mean concentration in single- and two-pollutant models ($PM_{2.5}$ + O_3); all females.



Theories of Pathophysiology

- Conduction instability (Peters)
- Vagal interruption (Godleski)
- Decompensated lung function (Hoek)
- Interaction with other air pollutants (Moolgavkar)
- Macrophage overload (Mauderly)
- “Irritation signal” (Oberdorster)
- Acid effect - acid-forming particles
- Transition metals, Fenton-like catalysis (various)
- Oxidative stress - reactive O_2 species (Donaldson)
- Inflammation, blood viscosity, and coagulation balance (Seaton, Peters)





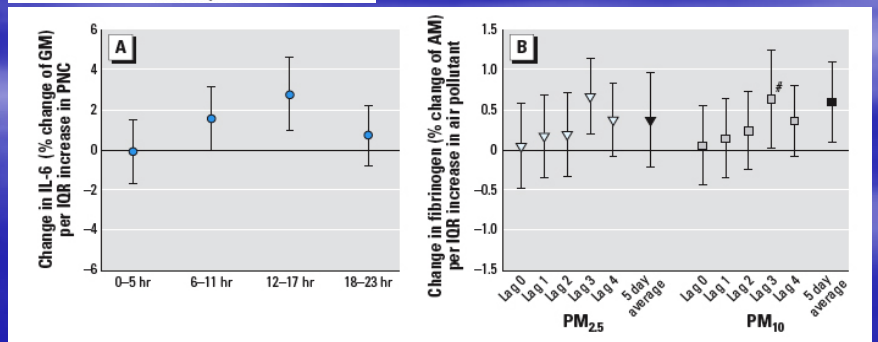
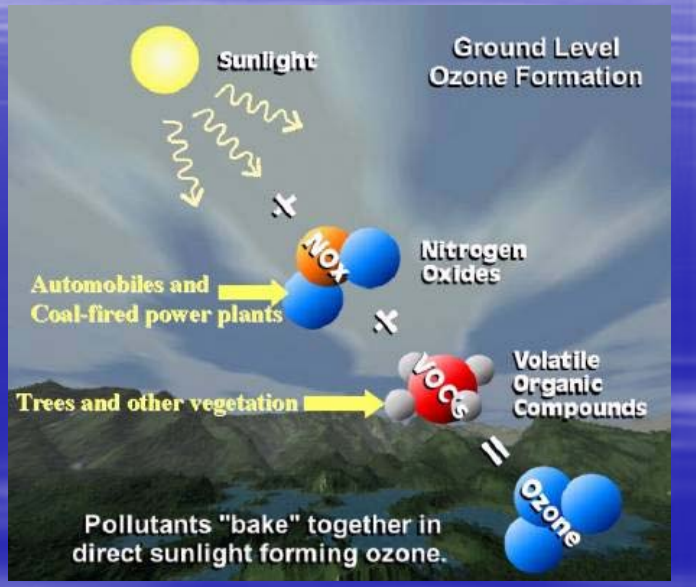


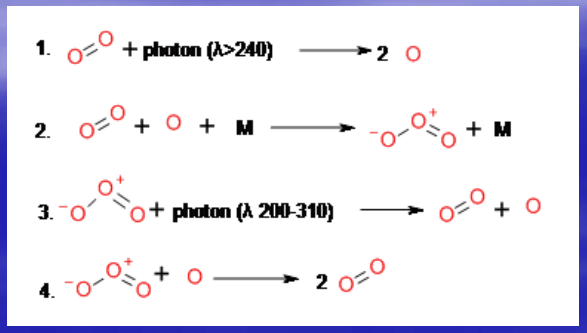
Figure 3. Pooled effects of PNC on IL-6 (*A*) and of $PM_{2.5}$ and PM_{10} (*B*) on fibrinogen, different lags. Abbreviations: AM, arithmetic mean; GM, geometric mean. Error bars indicate 95% CIs.
#Heterogeneity between the cities present.

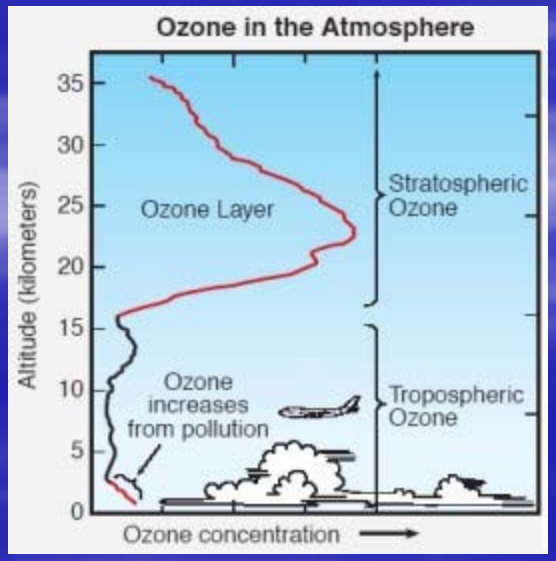
Rückert et al., 2007

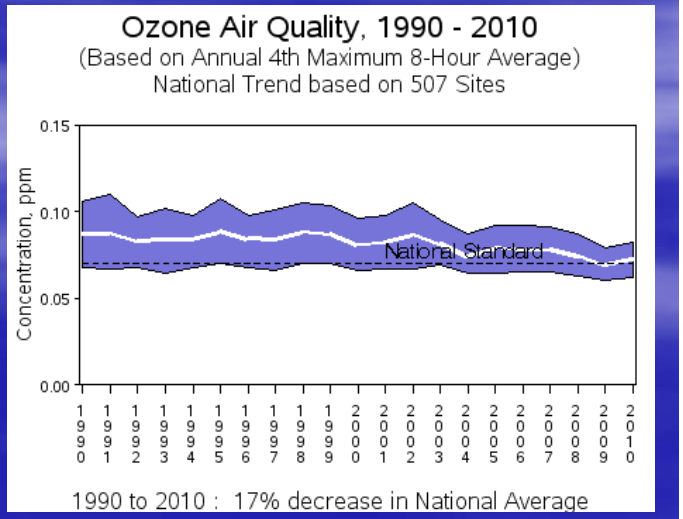
Ozone

- Secondary pollutant in photochemical air pollution
- Highly oxidizing gas, not water soluble
- Major fraction of Tox
- Mobile sources
- Long-range transport



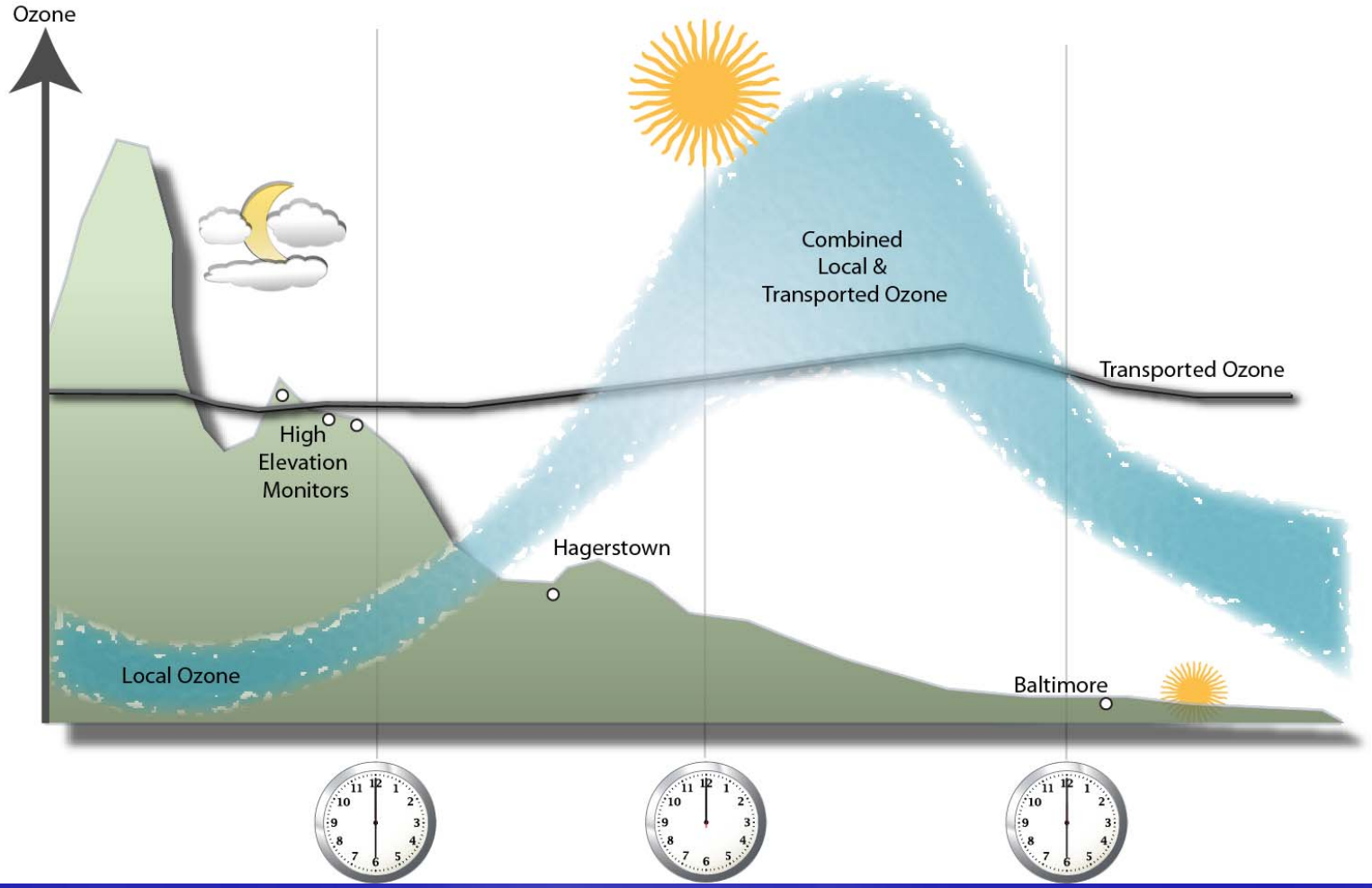






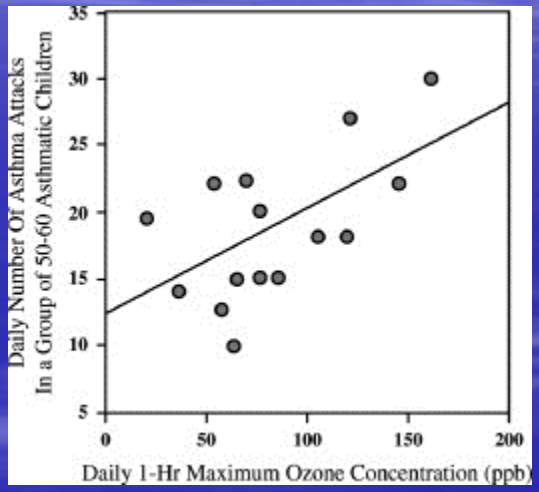
Transported Ozone

http://www.mde.state.md.us/programs/Marylander/Pages/AirQualityAwarenessWeek.aspx

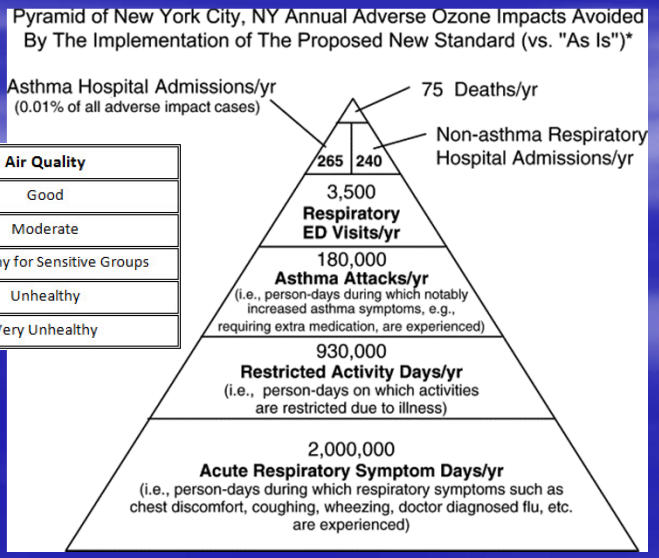


Ozone Health Effects

- *Primary* effect on peripheral ***airways***
- *Secondary* effect on cardiovascular system, morbidity
- Short-acting, lag-time
- Provocation of asthma +
- Causation of asthma ?
- Major issue in children's environmental health



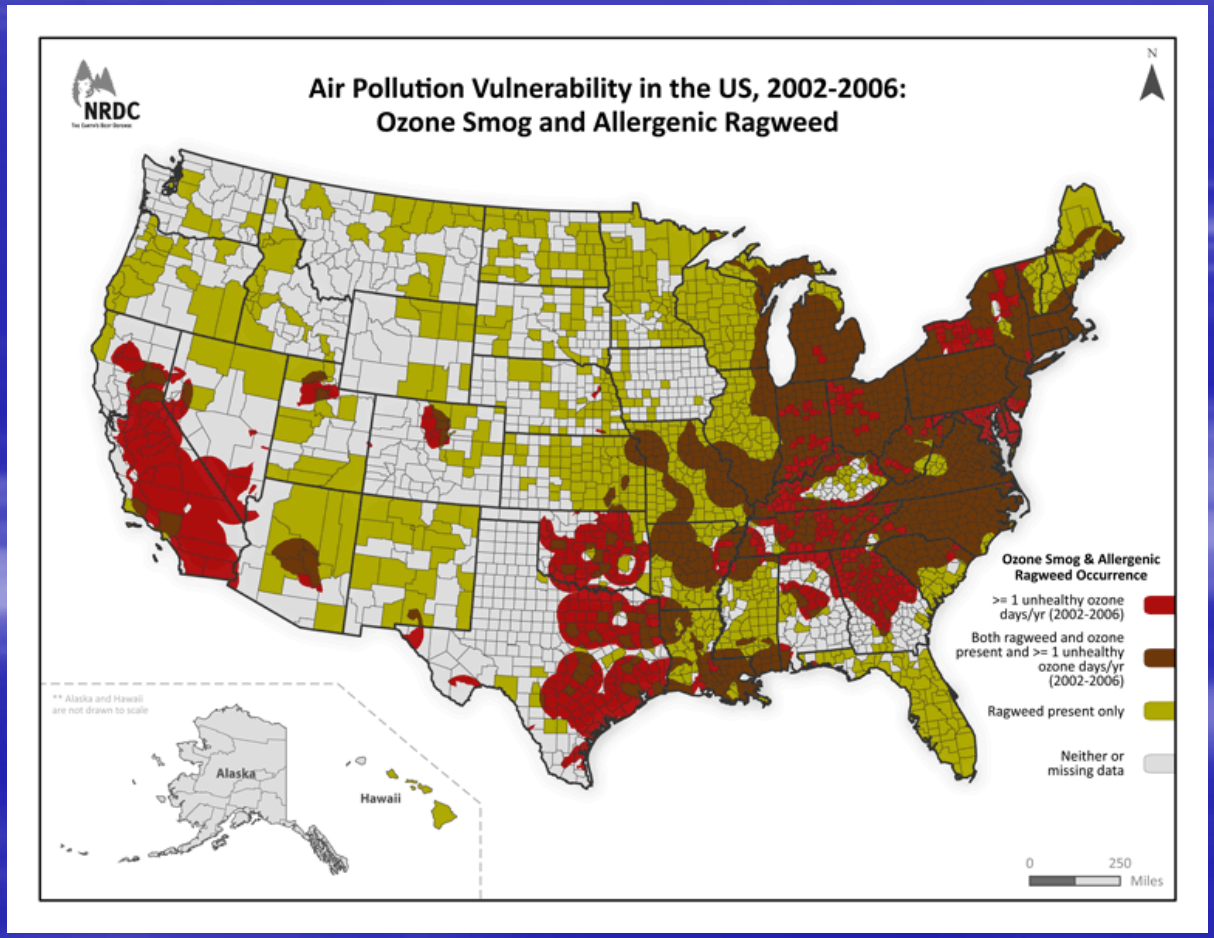
AQI Color Code	AQI Value	Air Quality
Green	0-50	Good
Yellow	51-100	Moderate
Orange	101-150	Unhealthy for Sensitive Groups
Red	151-200	Unhealthy
Purple	201-300	Very Unhealthy



Trasande et al. *J Allergy Clin Immunol* 2005;115:689

Ozone Aggravates Allergies

- Irritant effect on airways
- Antigen processing?
- “Double whammy” for Metro Washington



Comparing the Issues

	Fine Particulate Matter	Ozone
Origin	1. Primary from combustion, esp. diesel 2. Aggregation from sulfate, nitrate	1. Secondary from photochemical reactions in presence of VOCs and NO_x 2. Long-distance transport
Minor sources	Crustal, local	Mountains, “tongues” from upper atmosphere
Population effects	Morality by cause	Morbidity: emergency room visits, asthma attacks
Predominant effects	Cardiovascular, pulmonary, systemic	Pulmonary (esp. asthma), cardiovascular
Most susceptible groups	Elderly, preexisting cardiovascular or lung dz	Children, preexisting lung dz, esp. asthma

Rationale for Reducing Standards for $PM_{2.5}$ and Ozone

Reducing the standard for fine particulate air pollution and ozone will:

- allow more Americans to live,
- improve the health of the American people overall and particularly those who live in cities,
- allow Americans with asthma, heart disease, diabetes, and a high risk for stroke to manage their health risks more easily,
- push improvements in pollution, but especially source control, that will reduce many forms of pollutions together,
- improve health and quality of life and productivity,
- lead to more efficient and therefore advanced and therefore competitive technologies.