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## **A review of recent health effects research**

# HEALTH EFFECTS OF PARTICULATE AIR POLLUTION

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The principal drivers for emissions measurement research relate to the contribution of combustion-derived particle emissions to ambient air quality, and the concern over particle-related health effects. This presentation discusses recent health-based findings with implications for emissions measurement.

The key research activities relate to the epidemiology of short- and long-term particle exposure, and to the potential toxicological mechanisms, which may help identify key components of the ambient particle mix, where targeted reductions might bring potential population health benefits. It is noted that there are still few definitive toxicological studies identifying key species; particle number, surface area, mass and composition all remain implicated as markers.

## **Epidemiology**

Population studies fall into two principal categories; short-term time series studies and long-term cohort based longitudinal studies. In each case, causal and/or associations are related to mass based particle metrics (PM<sub>10</sub>, PM<sub>2.5</sub>) as these parameters are most commonly monitored in ambient monitoring networks. There are however, some limited data on health effects related to alternate markers such as particle number or surface area equivalents.

The short-term time series studies look at the association of health outcomes on any day versus environmental and / or air quality metrics. In these studies, whole population statistics are evaluated. A recent re-analysis of time series studies by the Health Effects Institute ([www.healtheffects.org](http://www.healtheffects.org)) has supported the previous conclusions, and the belief that key confounders (most notably NO<sub>2</sub> and temperature) have been adequately controlled. In addition, the strength of the effect of particle exposure on cardiovascular mortality in pollution episodes has been remarkably consistent across many developed and developing cities. These data support a causal association between particles and health.

Longer-term cohort based longitudinal studies follow health outcomes in known populations within various cities, but with limited exposure data (usually annual-averaged PM data). Again, the Health Effects Institute has reported new data from 6 Cities study and the American Cancer Society cohort studies (at 151 centres) and confirmed the finding that life expectancy is reduced in cities with higher air pollution. These data have been re-analysed by the UK Committee on the Medical Effects of Air Pollutants (COMEAP: <http://www.doh.gov.uk/comeap/index.htm>) who have calculated potential population life expectancy benefits from reductions in particulate air pollution. They conclude that it is more likely than not that there is a causal relationship between particulate air pollution and long-term health effects, and that the magnitude of the long-term health effects is at least equivalent to that of short-term effects, but may be more significant.

Thus, these data reinforce the existing health drivers against which improvements in air quality and reductions in emissions are being sought, with associated population-based economic benefits. Future insights are likely to be gained from improved source apportionment of ambient particles, for which methods exist, being adopted in epidemiology studies.

# Health Effects of Air Pollution

5. Nanoparticle Conference, Zürich  
2001

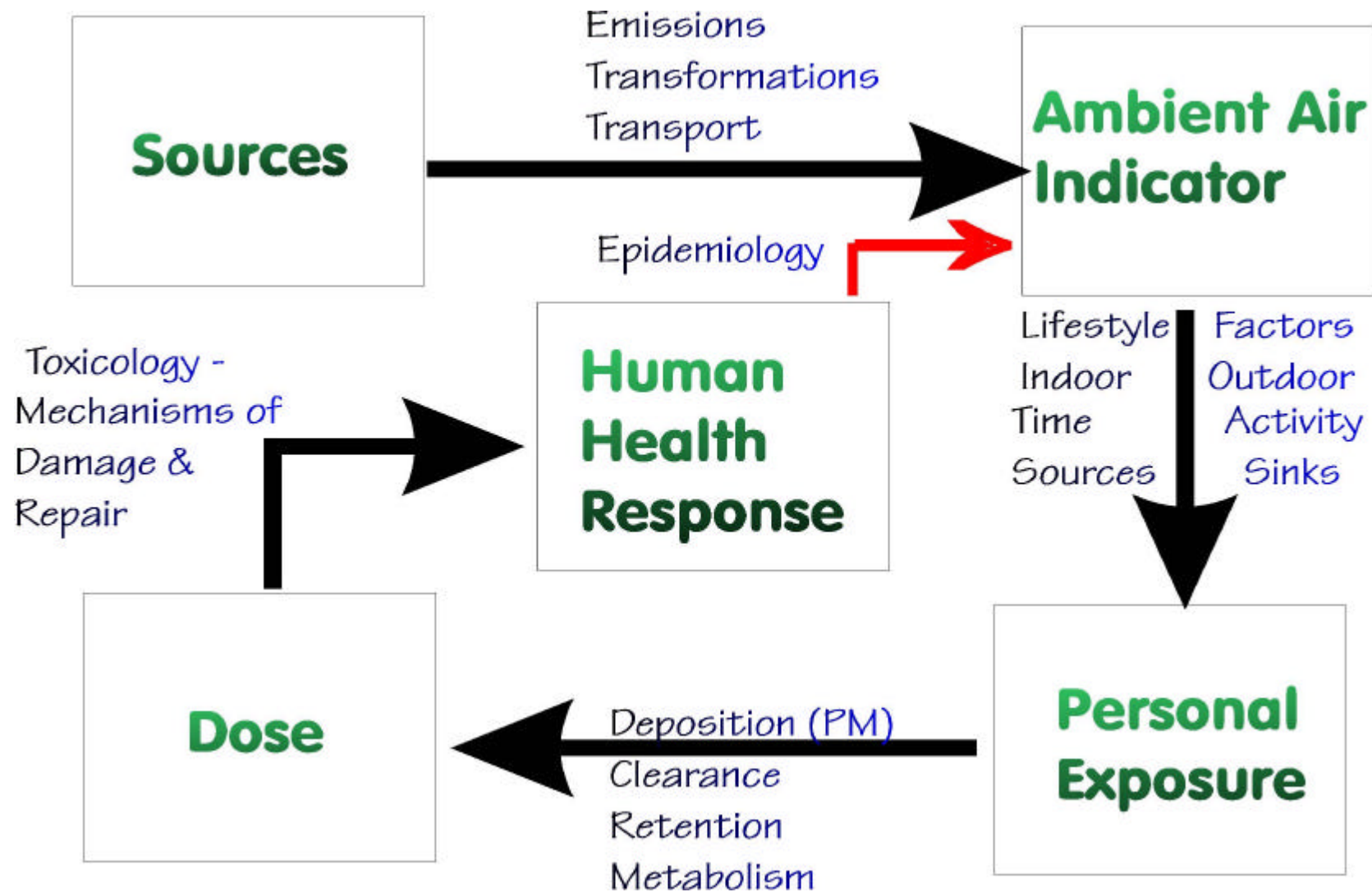
John McAughey



# Structure of this talk

- Relationships of exposure and health
- Epidemiology
  - Time-series
  - Long-term longitudinal studies
- Toxicology
  - Ultrafines
  - Metrics
- Implications for vehicle emissions

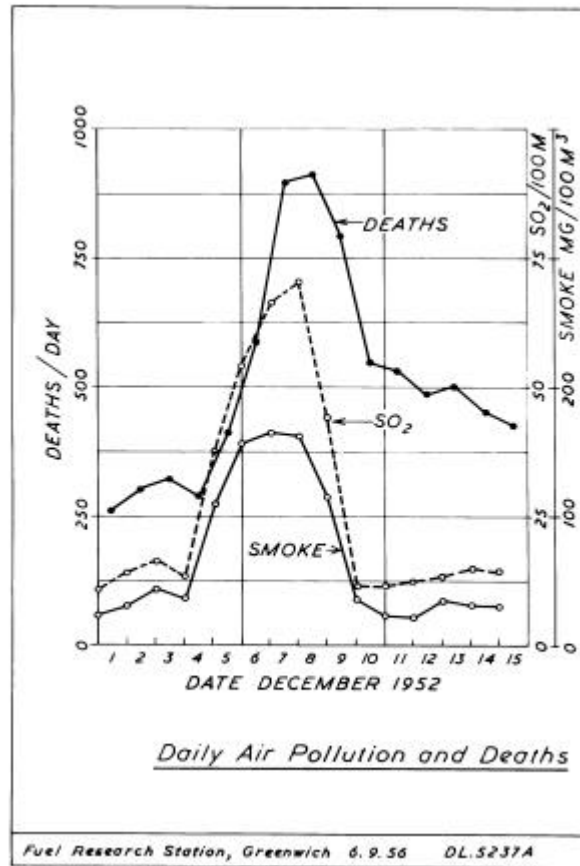
# Framework of Human Health Impacts



# Epidemiological Evidence for Health Effects

<i>Method</i>	<i>Strengths</i>	<i>Limitations</i>
<b>Epidemiology</b>	Populations in normal environments	Association v Causation Exposure estimates Potential confounders
<b>Controlled human exposures</b>	Relevant species	Small numbers Limited exposures Healthy subjects
<b>Toxicology Studies</b> (Animals / cells)	Addresses mechanisms Rapid results Controlled conditions	Relevance to humans 'Artificial'

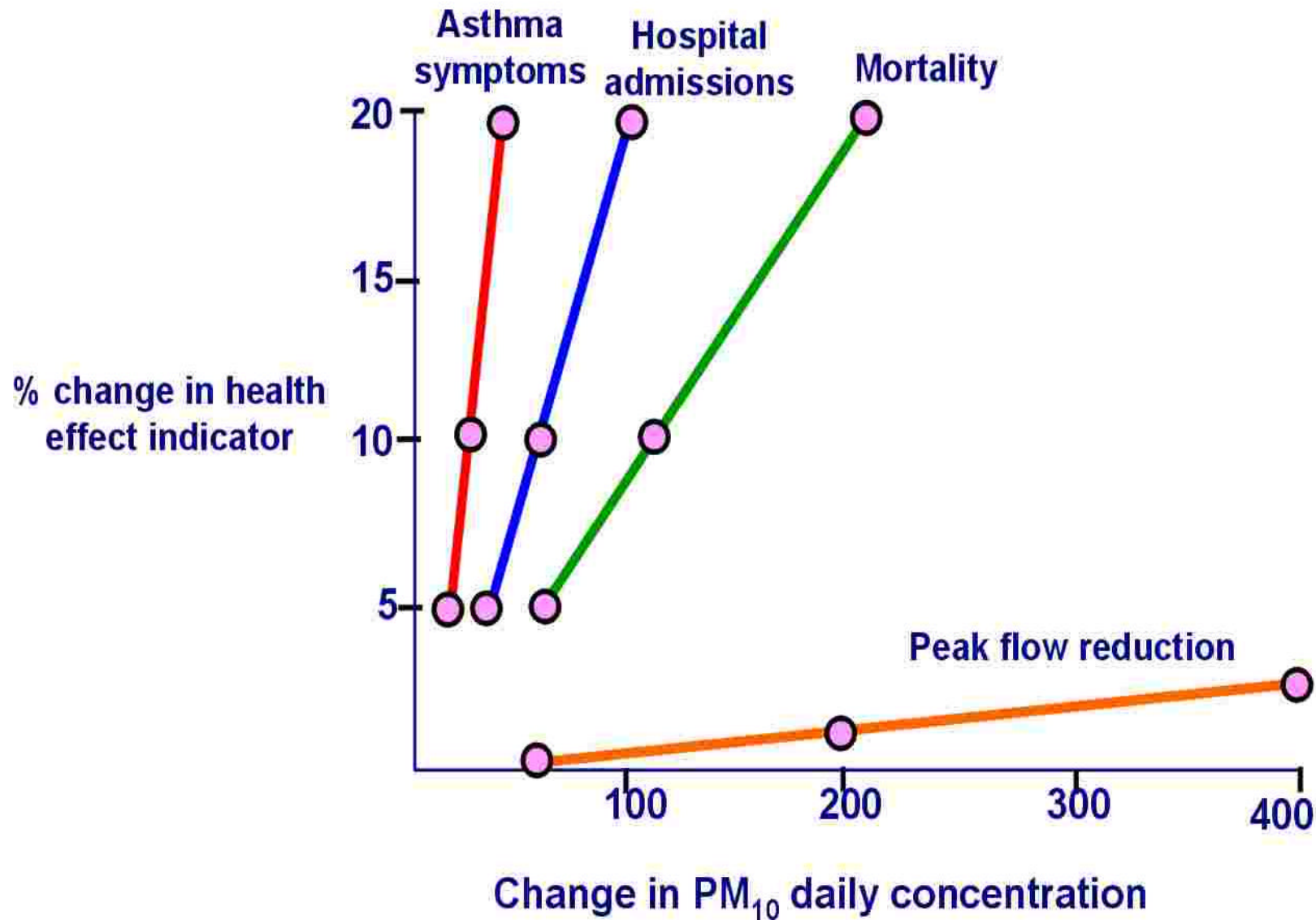
# Quantifying Mortality



PM<sub>10</sub> - cardiac and respiratory mortality  
Santiago, Chile : 1.1% for +10 µg/m<sup>3</sup>  
Birmingham, UK : 1.1% for +10 µg/m<sup>3</sup>  
International values all in similar range

- high pollution days correlate with increased mortality / morbidity
- 1990s xs mortality lower than 1950s - statistical association
- HEI & APHEA re-analysis such that PM<sub>10</sub> exposure effects robust
- Believed that effect modifiers (confounders) controlled (T, NO<sub>2</sub>)
- COMEAP accepts as causal

# Short-term Exposure Responses





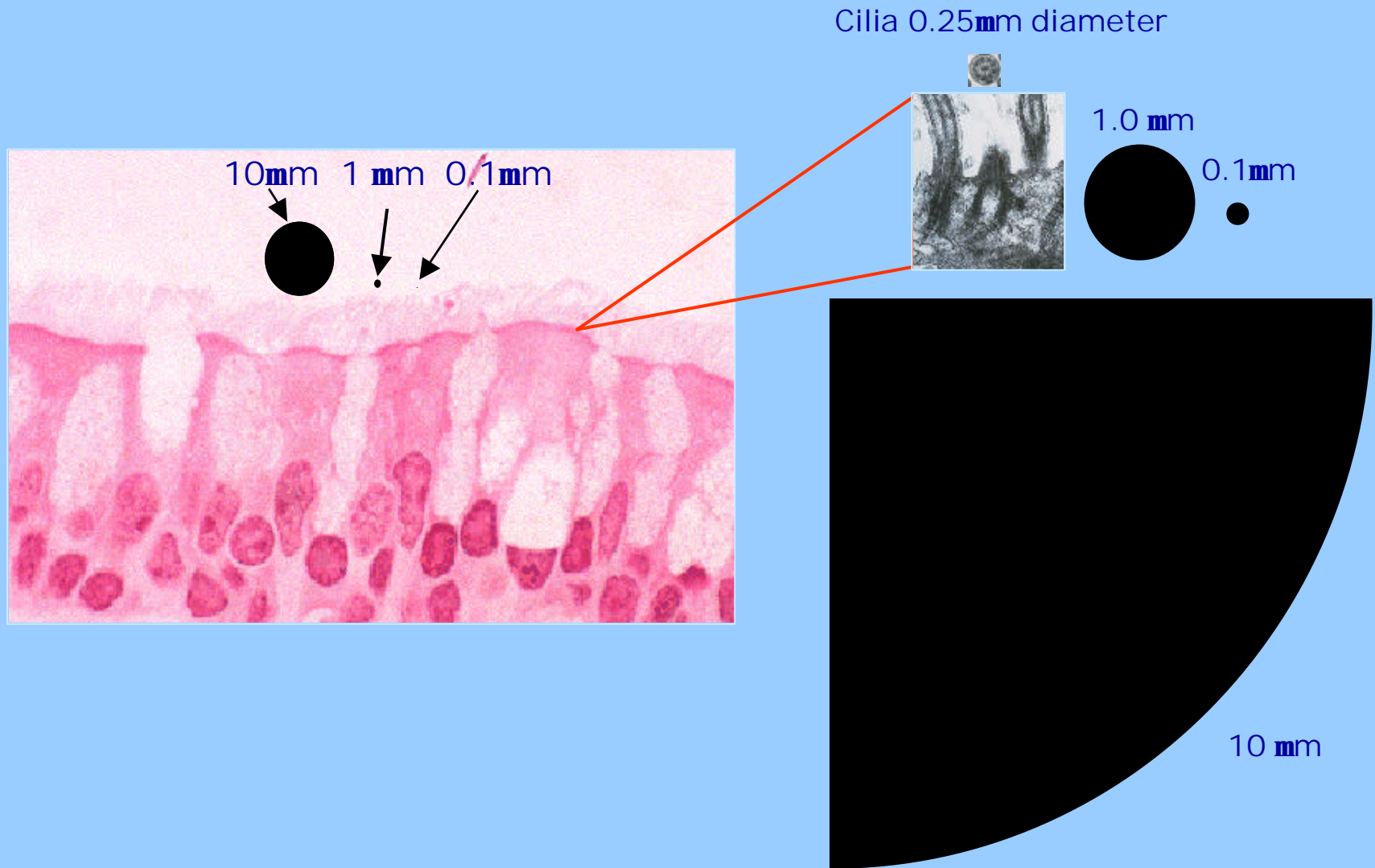
# Longitudinal cohort studies

- Known population cohorts followed over time in known locations
- Health outcomes versus annual average pollution levels
- e.g. US 6 Cities study and American Cancer Society cohort study (150 cities)
- estimate of change of life expectancy and number of life years saved for improved AQ by interpolation
- important for susceptible groups
- UK COMEAP
- <http://www.doh.gov.uk/comeap/index.htm>
- ‘considered more likely than not that a causal association exists between long term exposure to particles and mortality’

# Toxicology

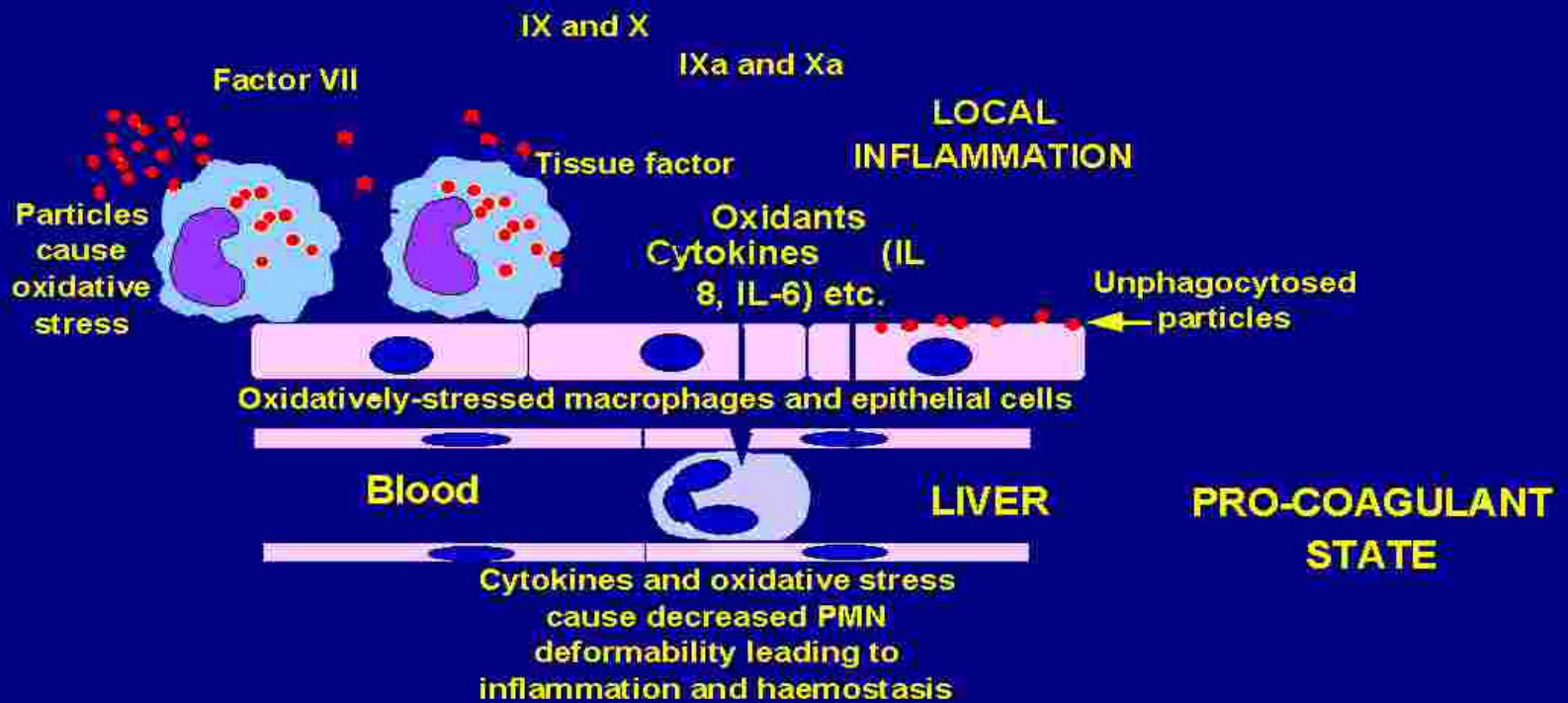
- lagging behind epidemiology
  - more focused with respect to mechanism of effect
- ultrafine particle effect noted from 'inert' materials ( $\text{TiO}_2$ , PTFE)
- ultrafine mode dominated by particle number
- dose response relationship with surface area (but linked to composition)
- surface reactivity (metals) mechanisms postulated
- CVD effects postulated - blood clotting, reflex control of heart rate variability
- Long-term cancer risks
- Limited epidemiology data from occupational exposure
- Toxicology data confounded by 'lung overloading'
- However, known carcinogens associated with particle phase

# Epithelium and particle size

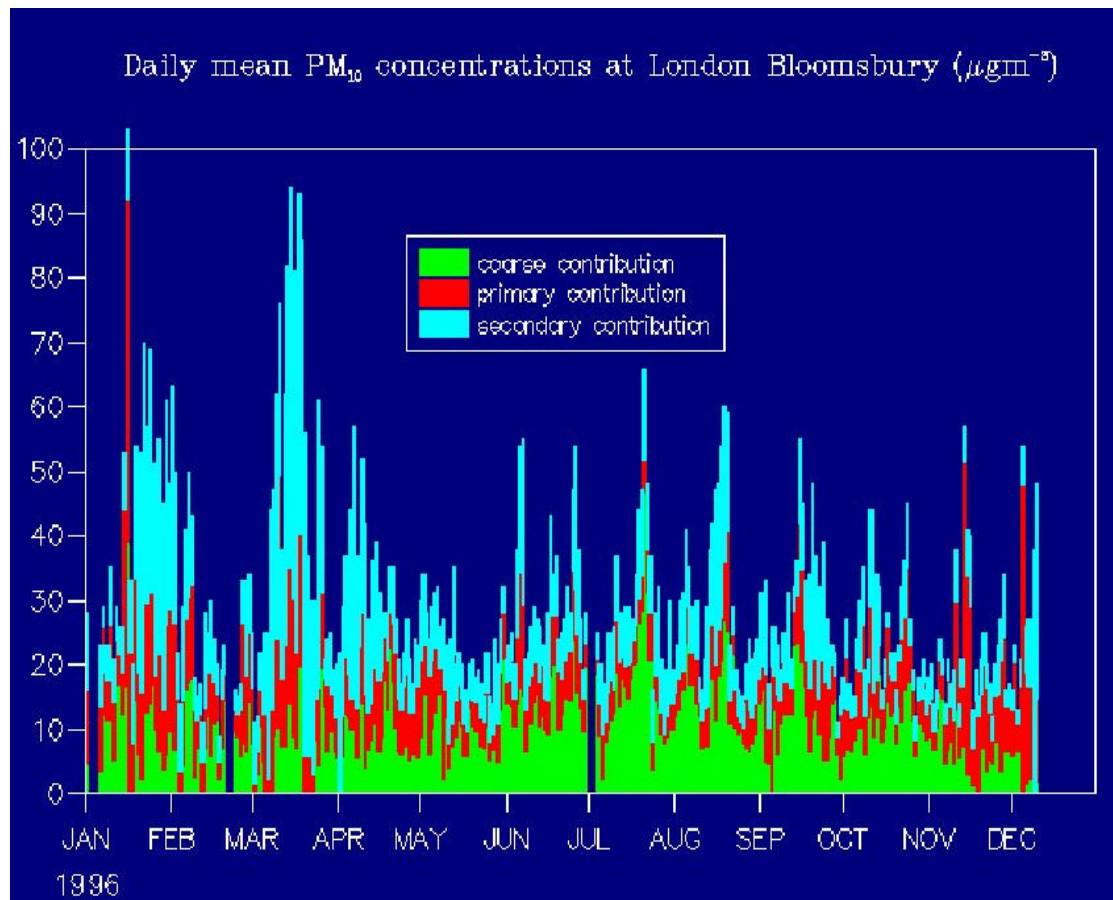


# Markers of effect

## Possible mechanisms for the production of local inflammation and a systemic pro-coagulant state after PM10/2.5 exposure



# Receptor Modelling Approach for Source Apportionment



- *Coarse mode*  
constant (varies with wind speed in later model)
- *Secondary mode*  
from sulphate + nitrate + ammonium
- *Primary mode* from Black Smoke

## Conclusions- health effects of air pollution

- Particles increasingly accepted as having causal relationship with short- and long-term CVD mortality, and to respiratory morbidity
- Significant economic consequences have been estimated
- Ultrafine particles are significant contributors
- Combustion products are principal man-made source of ultrafines
- Further mechanistic research required
- Epidemiological studies need to address more complex metrics



*“If you ask me, the fire has the most potential,  
but it’s the smoke that has people talking”*