WORKING FOR A HEALTHY FUTURE

Urban Air Pollution and Human Health

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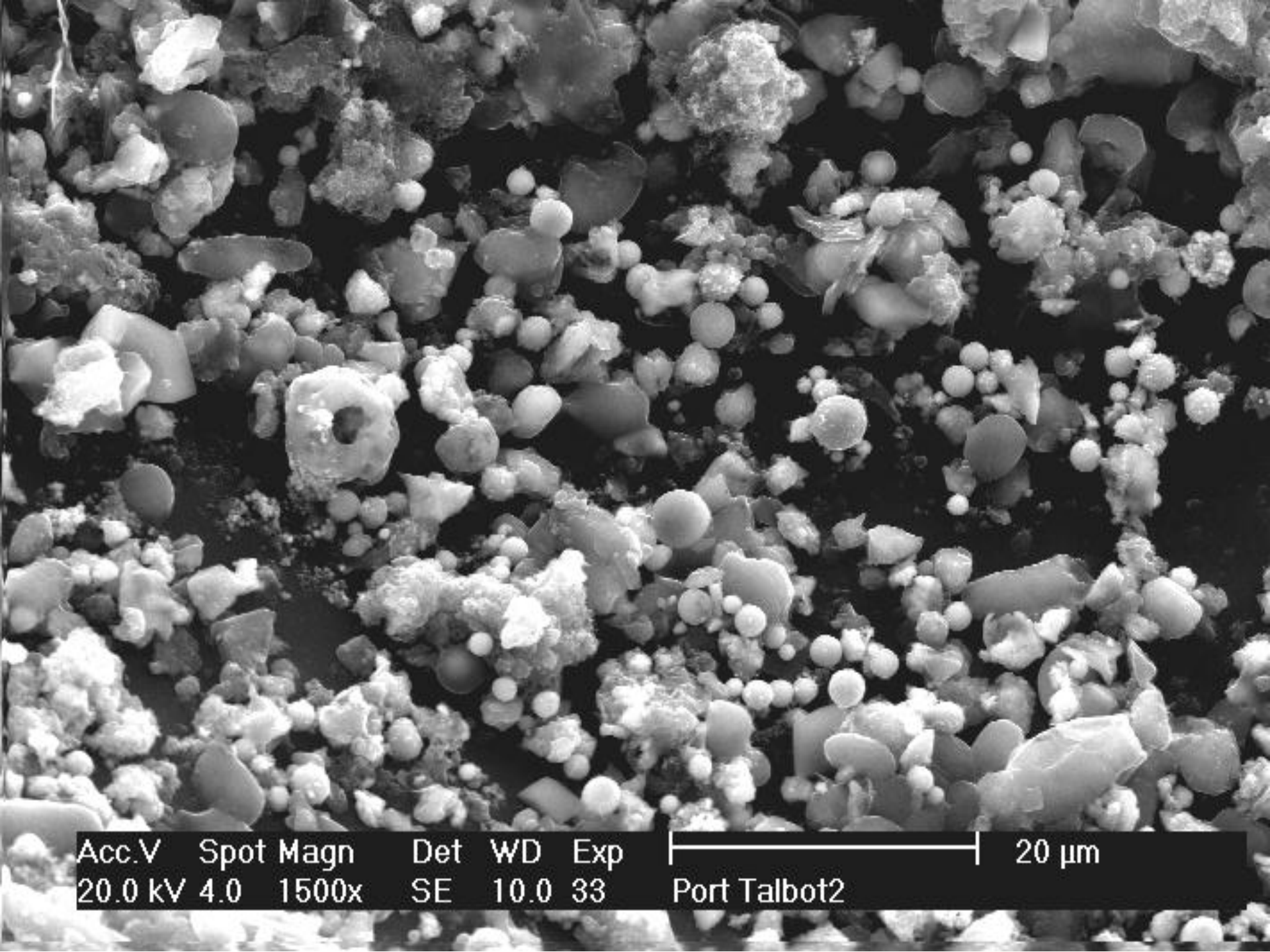
With slides from several colleagues, IOM and other

Key messages (1) – General and PM

1. Air pollution damages human health – it increases the risks of a very wide range of common conditions, mostly cardio-respiratory, from minor changes through to earlier death.
2. There are effects at all ages from during pregnancy (still debated) through to old age. The main public health effects are on older people.
3. Air pollution is a mixture of small particles (Particulate Matter, PM) and gases (NO2, ozone, SO2 especially) and it's not easy to determine what pollutant is causing what.
4. The evidence implicating PM is strongest: from studies of (i) short-term exposure ('daily variations'); (ii) cohort studies of longer-term exposure (annual average); (iii) interventions. There are plausible biological mechanisms.
5. PM is an issue that affects everybody: (i) Everybody is exposed (though to different amounts) both outdoors and indoors; (ii) Attempts to identify a 'safe level' ('threshold') for PM, for the population as a whole, have failed.
6. Also, PM is itself a mixture of various kinds of small particle from different sources and it's hard to tell what aspects are more dangerous; but there are clues…

Outdoor air pollution is a mixture…

- …of Particulate Matter (PM) and gases
  - Gases emitted from combustion – NO2 (mostly traffic), SO2 (mostly industry), [CO – mostly traffic]
  - Gases formed subsequently – ground-level ozone (O3), from NOx
- PM is itself a mixture – varies by size, composition, surface properties
  - Primary particles (i) combustion – with many surface components, e.g. transition metals; (ii) other primary – abrasion, natural dusts, re-suspended road dusts…
  - Secondary particles – nitrates and sulphates – formed over longer distances when gases (NO2, SO2) interact with e.g. ammonium
- Measure as
  - Mass in different size ranges: PM10, PM2.5, PM1, PM0.1; in $\mu g.m^{-3}$
  - Mass of different components – sulphates, black carbon ('soot'),..
  - Particle number – especially near roadside, high levels of ultrafines; e.g. often >20,000 per cc (!!), sometimes 100,000 per cc. (also, the surface area of the lung is huge…)
  - Trace metals, acidity…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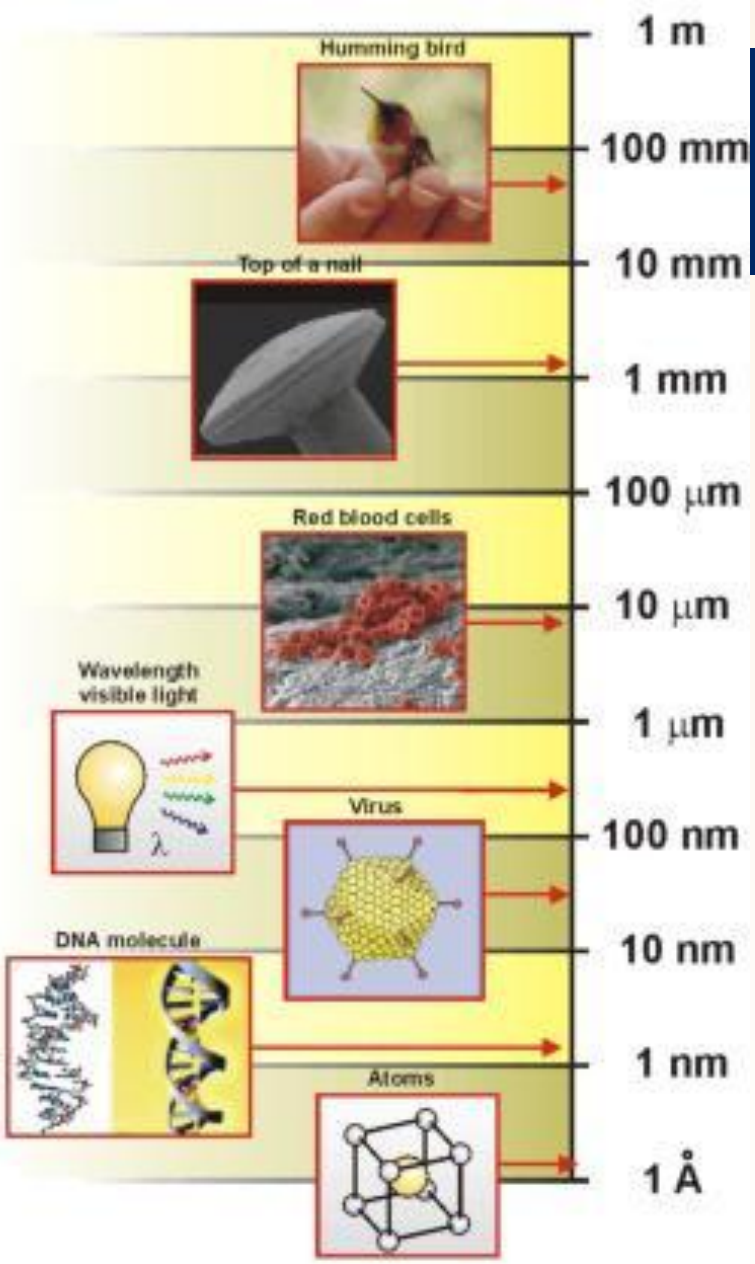
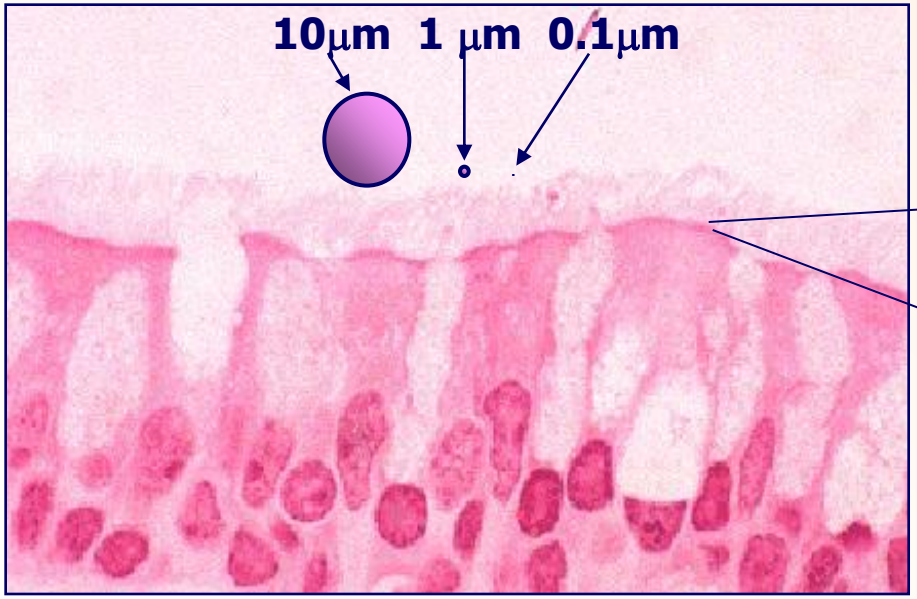


How small are the air pollution particles?

- We're most interested in the effects of fine particles
- (Aerodynamic) diameters up to around 2.5 µm

- Increasing interest in even smaller particles
- 'Ultrafines'/ 'Nanoparticles'

These particles are **small**: Relative size of nanoparticles to cells



Bronchial epithelium

Cilia 0.25μm diameter

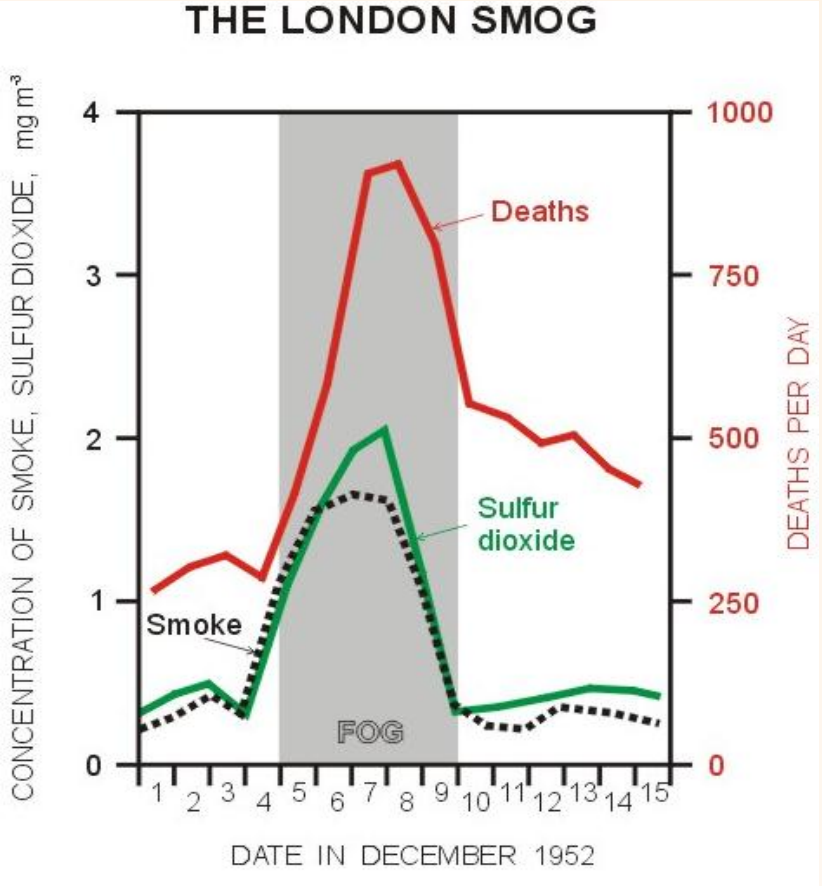
1.0 μm

0.1μm

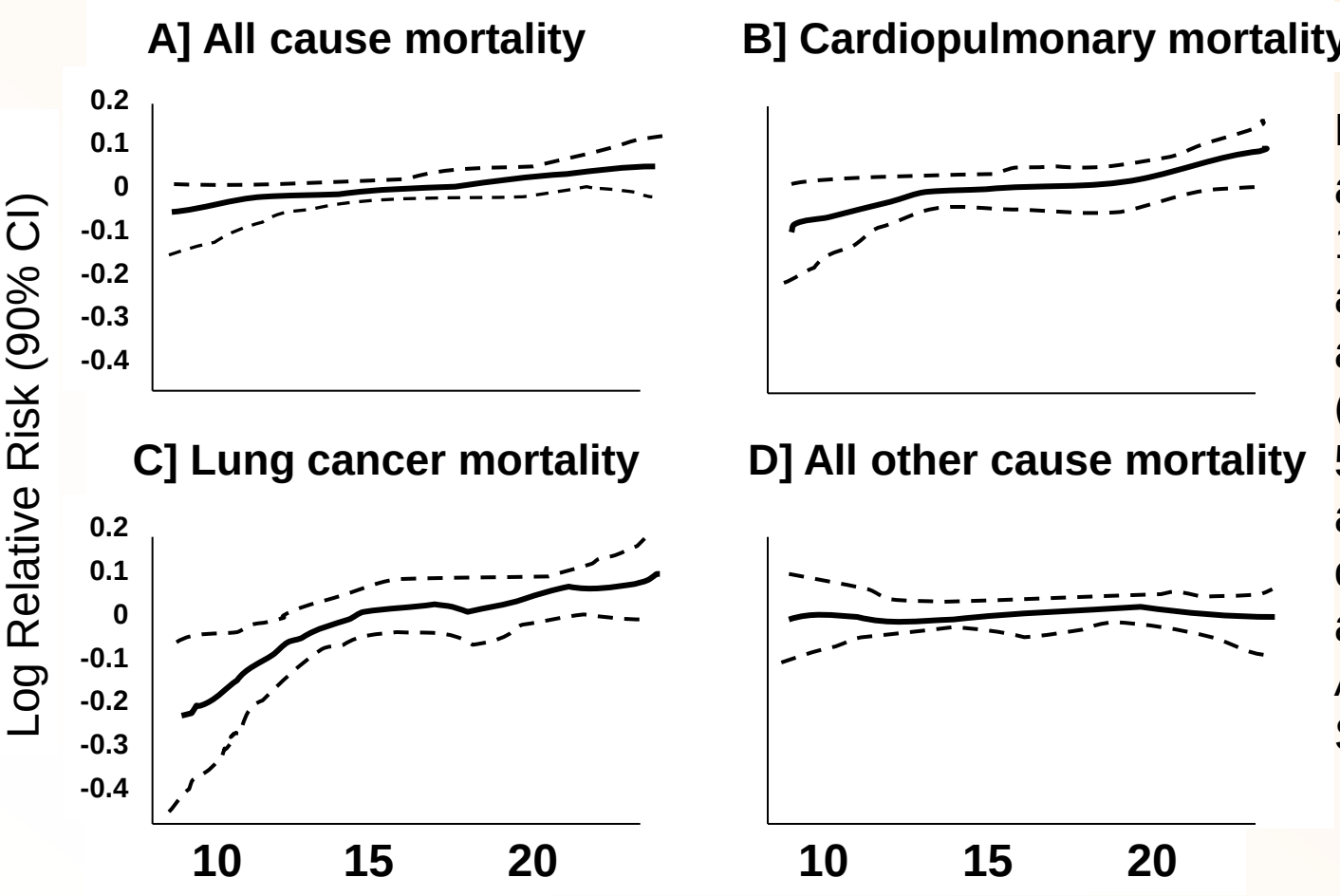
Air Pollution Damages Human Health

- Three main phases of research on air pollution and health
  - 1950s onwards: Air pollution episodes cause increased death and ill-health on the same day or on the days immediately following e.g. London smogs
  - Late 1980s onwards: Daily pollution at 'normal' levels causes increased death and ill-health on the same day or on the days immediately following:
    - US EPA (1995) "Strong evidence of a weak effect", i.e. increase in risk was small
    - COMEAP (1995): Imprudent not to consider PM10 effects as causal
    - There is no known 'safe level' (threshold), for the population as a whole
    - Increased risks of death believed to be among people with pre-existing serious cardio-respiratory disease
  - Mid 1990s onwards: Air pollution has a very wide range of adverse effects on human health; but the dominant effect is increased risk of mortality in adults from long-term exposure, where air pollution is best represented as annual average fine Particulate Matter (PM2.5) (COMEAP, 2009)
    - Again, there is no known 'safe level'
    - The risks seem to apply to the population as a whole, not just those with short remaining life expectancy
    - 6% increase in age-specific death rates per 10 $\mu g.m^{-3}$

London 1952 Smog Episode: Early Example of a ‘Time-series’ Study



$PM_{2.5}$ (μg.m^{-3}) and mortality, Pope et al (2002): Shape of the relationship: ACS Study



Log Relative Risk and CIs of mortality 1982-1998 and annual average ambient particles (PM2.5 in μg.m^{-3}) in 51 US metropolitan areas, based on a cohort of 319,000 adults aged 30+: American Cancer Society study

Health Effects quantified in CAFE CBA and by COMEAP – PM and ozone

- **Chronic (long-term) exposure to particulate matter (PM):**
  - Mortality (PM) and adults – the dominant effect;
  - Infant mortality and PM
  - Development of bronchitis (PM)

- **Acute (short-term) exposure to PM and to O_3**
  - Mortality (O_3) – mortality and PM already included
  - Hospital admissions
    - Respiratory (PM, O_3); Cardiovascular (PM)
  - Days of Restricted Activity; Days off Work (PM, O_3)
  - Visits to GP (PM)
  - Days with symptoms and/or using medication (PM, O_3)
    - In people with chronic lung disease (asthma, COPD)
    - In the general population
- Full CAFE CBA Methodology report (Hurley et al., 2005) on the Web

Is All PM2.5 Equally Toxic?

- Issue is controversial scientifically and is very important practically, on what to control, e.g.
  - Are primary particles, especially primary combustion particles, more harmful per $\mu g/m^3$ than secondary particles?
  - What do we really know about the toxicity of nitrate particles?
  - Is Black Carbon (BC) a better index than PM2.5 for the health effects of PM2.5 from local traffic sources?
- Almost certainly there is variation in toxicity – I have been using 'differential quantification' in research projects since 1998
- BUT the evidence is far from clear-cut and WHO Expert Groups, and others (COMEAP, US EPA) say the evidence is not good enough to quantify
- So now, in quantification work, (i) for primary analysis, I treat all PM as 'equally toxic'; (ii) in sensitivity analyses I 'play' with relative toxicities, to see if results are robust. – 'no regret' policies

WHO views on toxicity of different kinds of PM2.5

- WHO (2004): Answers and Follow-up Answers to CAFE
  - Toxicological studies have highlighted that primary, combustion-derived particles have a high toxic potency; and several other components of the PM mix – including sulphates and nitrates – are lower in toxic potency;

  BUT
  - It is currently not possible to precisely quantify the contributions from different sources and different PM components to health effects.

- WHO (2007) Special 3-Day Workshop:
  - "Current knowledge…does not allow precise quantification or definitive ranking of the health effects of PM emissions from different sources or of individual PM components.
  - Thus, current risk assessment practices should consider particles of different sizes, from different sources and with different composition as equally hazardous to health."

Views on toxicity of different kinds of PM2.5

- WHO (2012) on Black Carbon (BC):
  - "The Task Force recommended that PM2.5 should continue to be used as the primary metric in quantifying human exposure to PM and the health effects of such exposure, and for predicting the benefits of exposure reduction measures."
  - "The use of BC as an additional indicator may be useful in evaluating local action aimed at reducing the population's exposure to combustion PM (for example, from motorized traffic)"

- Transition metals on the surface of small particles
  - Vanadium, Zinc, Nickel…

- In toxicology studies 'pure' sulphates and nitrates do not show much toxic activity. But epidemiological studies show strong relationships
  - Are these acting as a marker of some wider mixture, by source?

Key messages (2) – PM effects are large

7. There is widespread agreement (e.g. WHO, COMEAP, US EPA) that when public health effects are aggregated across the population…
   - The most serious is an increased risk of death in adults from long-term exposure, especially to fine particles (PM2.5).
   - This is estimated as a 3% increase in all-cause mortality per 5 $\mu g.m^{-3}$ PM2.5 (population average Scotland) – varies by specific cause.
8. Because everyone is exposed the aggregate population effects are large. COMEAP (2010) estimated these as equivalent to about 1,500 deaths per year (2.8%) in Scotland, if air pollution were the sole cause – much more than Road Traffic Accidents (RTA).
9. It's not easy to communicate the size of the problem because unlike RTA there are not identifiable 'victims'.
10. There are many other effects of PM – from long-term exposure, and from the 'triggering' effects of day-to-day levels, including effects of coarse PM also.

COMEAP (2010): In Scotland, mortality effect of current anthropogenic PM2.5

- From COMEAP (2010) – results for Scotland
  - Loss of life expectancy 3-4 months – average population-weighted PM2.5 was 5.0 µg/m^3
  - Attributable deaths – if air pollution were the sole cause, equivalent to
    - 1,560 in 2008, out of 55,500 (2.8%);
    - Average Years of Life Lost (YOLL) – >12y, per death
    - Total population time lost – 19,000 years of life, per annum
  - Air pollution one cause among many, increasing the risk of common causes of death
    - Plausibly >10,000 individuals affected with on average <2 years
  - All with large uncertainty bands
  - Most of this mortality attributable to annual average PM2.5 at <7µg/m^3, i.e. with somewhat greater uncertainty

- Nevertheless, a significant public health problem
  - RTAs, 2007: 281 deaths, 2375 serious injuries
  - Alcohol-specific deaths: In 2003, 2882 deaths “in which alcohol played a part”

Key messages (3) – Gases and sources

11. There are ‘triggering’ effects of ground-level ozone. These seem to be independent of (additive to) those of PM. There may effects of longer-term exposure.
12. There are associations of NO2 with a wide range of health outcomes, including mortality and hospital admissions and development of asthma in children.
    - It is unclear to what extent the most serious effects (e.g. on mortality) are a direct effects of NO2 *per se* or reflect NO2 as a marker of traffic pollution.
    - In studies of short-term exposure NO2 seems to be picking up an effect different from PM. Also, NO2 is a precursor of secondary PM (nitrates) and ozone.
13. Action on air pollution needs to be both local and international. Traffic is the main source – both local and transboundary.
14. There is a separate but related literature on the adverse health effects of living close to major roads – not just combustion PM – non-tailpipe emissions also…

Close to major roads

- Many studies of traffic pollution in cities examine not only the role of individual pollutants – PM and its constituents, NO2, etc. – but also distance from major roads
- Major review by the US Health Effects Institute (2010)
- There are adverse effects of living close to major roads, i.e. within 100-500m.
- Air pollution seems to be a contributory factor.
  - The specific cause is unknown
  - It seems not to be PM2.5 – because PM2.5 generally doesn't show much gradient close to major roads
  - Things that do vary include NO2, ultrafine PM, Black Carbon, coarse PM (from non-combustion dust).

Inequalities in air pollution and health

- Differences in exposure to air pollution
  - Background concentrations of individual pollutants (e.g. PM2.5)
  - The nature of the air pollution mixture
  - Personal exposures, for a given background concentration, e.g. closeness to major roads
- Differences in relative risks, per unit exposure (µg/m3)
  - expressed as % change in risk of adverse health effect
  - Higher risks with lower educational status (ACS)
- Differences in background rates of mortality or morbidity
  - the same exposure and same % change imply bigger impacts whenever background rates of death and disease are bigger

Policy responses: UK and EU

- Pollution episodes are damaging:
  - Control the peaks / Eliminate the episodes / Ban domestic coal burning
  - Largely successful – thought the problem was solved
- 'Normal' daily (24-hr average) PM10 is damaging
  - Implication: Control annual average PM10;
  - EPAQS (1995): Control both high days and annual average
  - PM10 has been reduced
- Biggest effect is annual average PM2.5 on mortality
  - Exposure reduction strategies for PM2.5
  - [But AQMAs still defined largely on exceedances of daily levels and of NO2.]
- EU reviewing its Air Quality Directives in 2013 – DG Environment with WHO and others

Some issues re. control of PM

What does the shape of the relationship imply for control?

- Shape is linear, no threshold – everybody exposed, no completely safe level
  - The big gains are in reducing annual average PM
  - Leading to exposure reduction strategies

- Is there a need in addition to control peaks – to limit individually high days?
  - Many people think there is, because individual high days cause damage to health
  - I'm not convinced that there is added benefit *on health grounds* to control of high days, provided that annual average is kept low enough.

To what extent is local air quality under local control?

- Limited: There is a need to co-operate…

Why control NO_2?

Possible reasons include (i) It is in itself a hazardous gas; (ii) It is a precursor of other pollutants; (iii) It is an indicator of traffic pollution

- It used to be that reducing NO_2 implies reducing the transport mixture as a whole; but now NO_2 can be controlled without controlling the mixture as a whole…
- While NO_2 *per se* damages health, the direct health benefits from control of annual average NO_2 are not easily quantified, and may be small.
- However, there seems to be an effect of NO2, or of something that co-varies with it, not explained by co-variation in PM – i.e. adjustment for PM does not remove the NO2 relationships.
- There are indirect benefits, via control of nitrate PM and ozone. These indirect benefits can be substantial.

It may be that, from a public health viewpoint, control of annual average NO_2 is best seen as part of a strategy to control PM2.5 and ozone, rather than as an end in itself.

Ongoing work – next steps

- Major ongoing projects in Europe
  - ESCAPE – Cohorts in Europe – long-term exposure and mortality; cardiovascular disease; lung disease; birth outcomes
  - TRANSPHORM – Traffic-related PM in cities; better speciation of PM; Health Impact Assessment of policies
- Reviews in support of EC Review of Air Quality Directives 2013
  - Review of major EU research findings 2005-2012 – David Fowler
  - WHO Answers to EU questions: REVIHAAP
- Ongoing work of COMEAP and other expert groups

THANKS…

- To many colleagues and collaborators over 20+ years:
  - Brian Miller (IOM) on air pollution and mortality
  - Colleagues from COMEAP, WHO etc.
  - Colleagues from EU projects: ExternE, CAFE, HEIMTSA, INTARESE, TRANSPHORM, URGENCHE…
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- To them and to many others for useful discussions: including SEPA colleagues – Anne Conrad, Colin Gillespie, John Redshaw, John Lamb…
- Opinions are my own – others not necessarily implicated
- And thanks to you all…