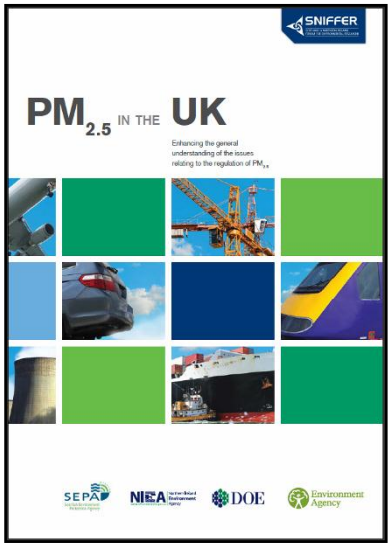
$PM_{2.5}$

an overview of composition, sources & health effects

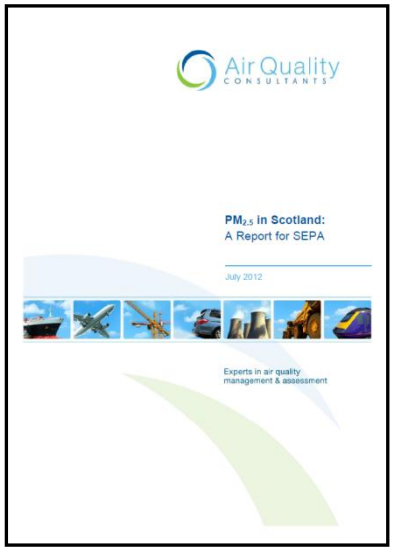
Mathew Heal

School of Chemistry, University of Edinburgh

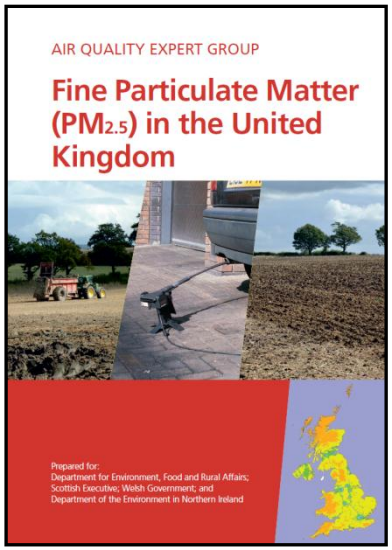
Scottish Air Quality Database and Website Annual Seminar,
18th March 2013



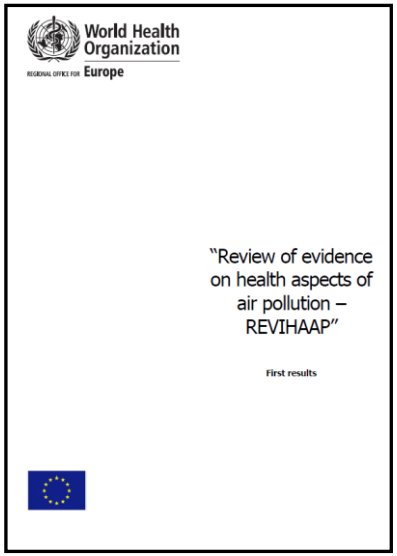
SNIFFER report: $PM_{2.5}$ in the UK
www.sniffer.org.uk



SEPA report: $PM_{2.5}$ in Scotland
www.sepa.org.uk or contact John Lamb

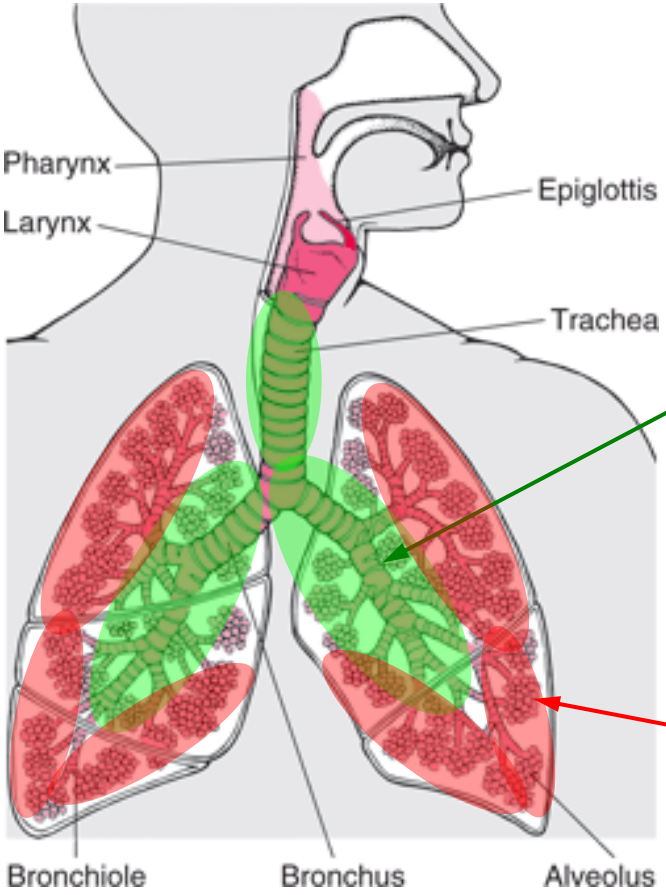


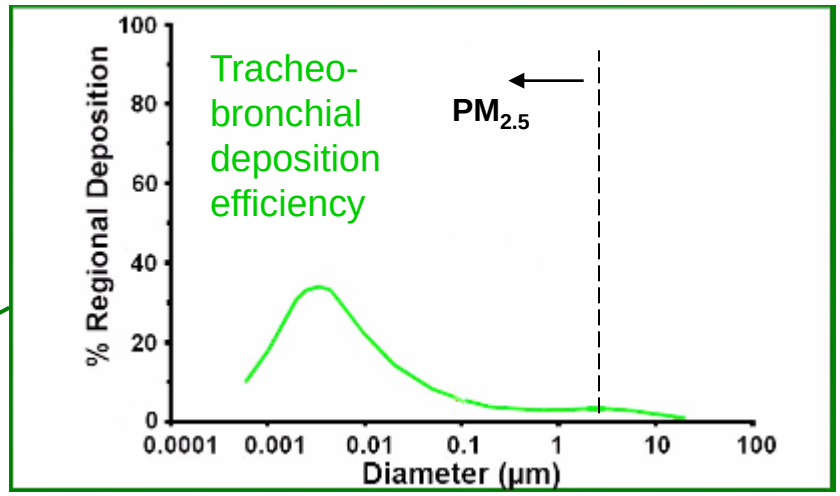
Defra AQEG report: Fine particulate matter in the United Kingdom
www.defra.gov.uk/environment/quality/air/air-quality/committees/aqeg/

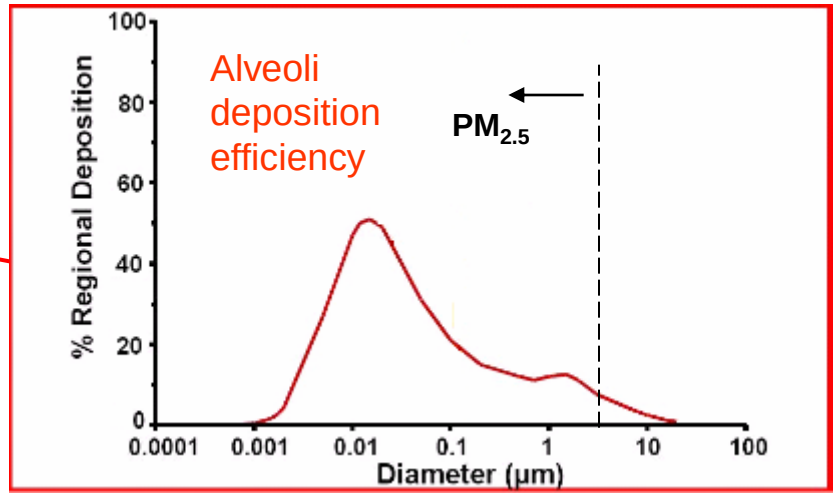


WHO report: Review of evidence of health aspects of air pollution
www.euro.who.int

The potential hazard: deposition in the respiratory system as a function of particle size







Health effects of $PM_{2.5}$ are associated with both short- and long-term exposures

Exposure	Outcome	Expert view on causality
Short-term	Mortality	Causal
Manifest as illness, hospital admission & death	Cardiovascular effects	Causal
	Respiratory effects	Likely to be causal
	Central nervous system	Inadequate evidence
Long-term	Mortality	Causal
Manifest as decrease in life expectancy & chronic illness	Cardiovascular effects	Causal
	Respiratory effects	Likely to be causal
	Cancer, genotoxicity	Suggestive
	Reproductive & neurodevelopment	Suggestive

USEPA (2009)

The quantitative impacts on health from exposure to $PM_{2.5}$

Short-term

Few studies for $PM_{2.5}$ but plenty for PM_{10}

Daily outcome	Relative risk per 10 μg m^{-3} PM_{10}	
All-cause mortality	0.6% (0.4 – 0.8%)	WHO (2004) meta-analysis
Respiratory mortality	1.3% (0.5 – 2.1%)	
Cardiovascular mortality	0.9% (0.5 – 1.3%)	
Hospital admissions	0.8%	

Long-term

Outcome	Relative risk per 10 μg m^{-3} $PM_{2.5}$	
All-cause mortality	6% (2 – 11%)	COMEAP (2009) (from US ACS and 6-cities studies)
Cardiopulmonary mortality	9% (3 – 16%)	
Lung cancer mortality	8% (1 – 16%)	

- Health impacts dominated by long-term exposures

What do these health risks mean in practice?

(for the UK)

Short-term

For PM_{10} levels in the UK in 2001, the IGCB estimate:

6,800 deaths brought forward

6,700 each for respiratory and cardiovascular hospital admissions

Long-term

For $PM_{2.5}$ levels in the UK in 2008, the IGCB estimate:

~6 months average loss of life expectancy

Other average loss of life expectancies:

1-3 months due to road traffic accidents

2-3 months due to passive smoking

Is there a safe level for exposure?

None yet identified at population level

But variability in individual susceptibility very likely

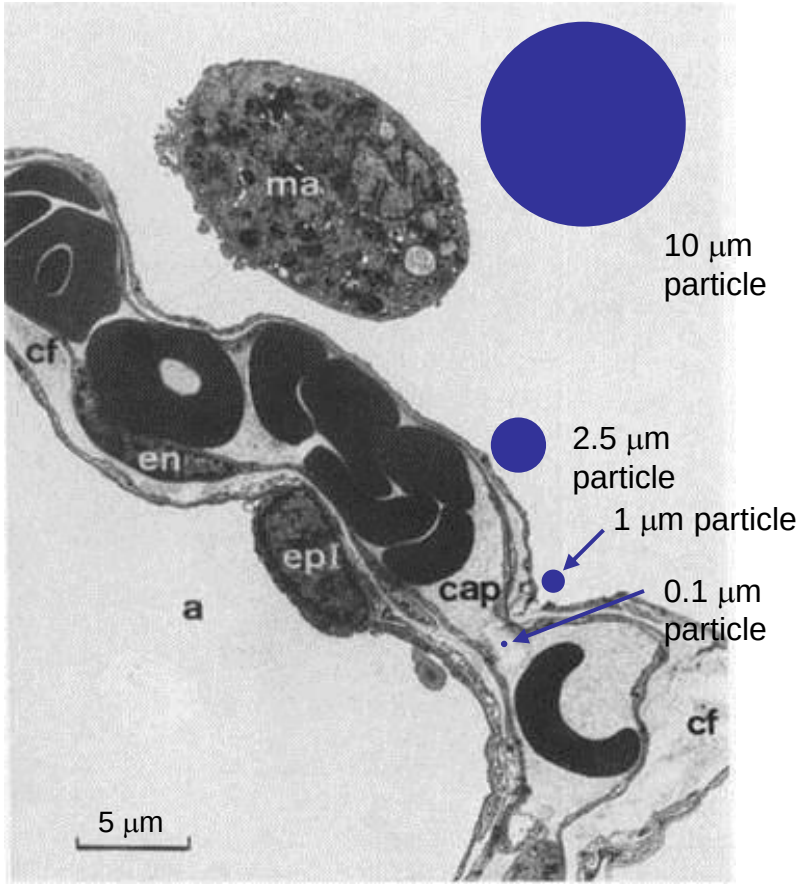
- effects expected to be greatest amongst children, the elderly and those with pre-existing disease

Are specific components/sources of $PM_{2.5}$ more toxic?

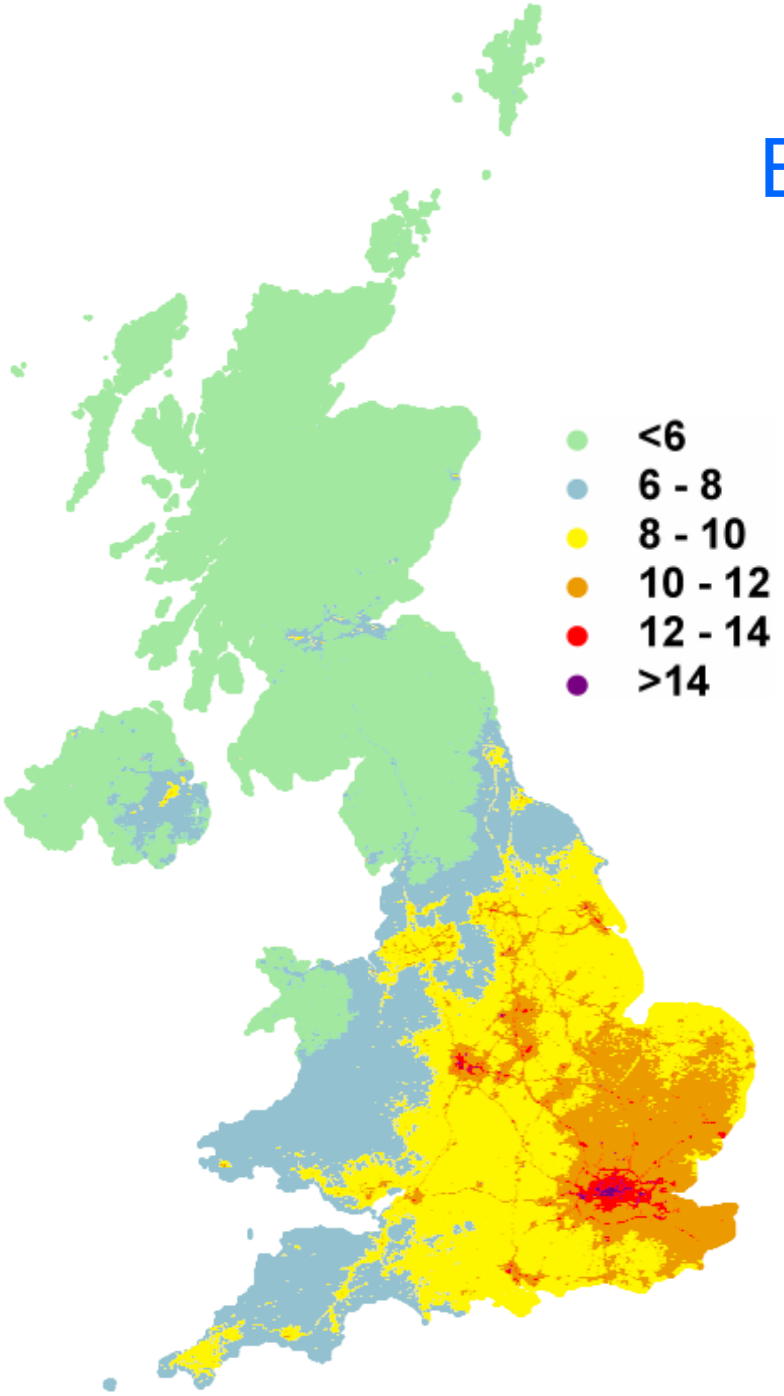
A cautious yes – for example:

- redox-active and/or toxic transition metals
- redox-active and/or toxic organic species
- ultrafine (< 0.1 μm) particle numbers
- particles from road traffic or combustion

The current WHO review says more on this



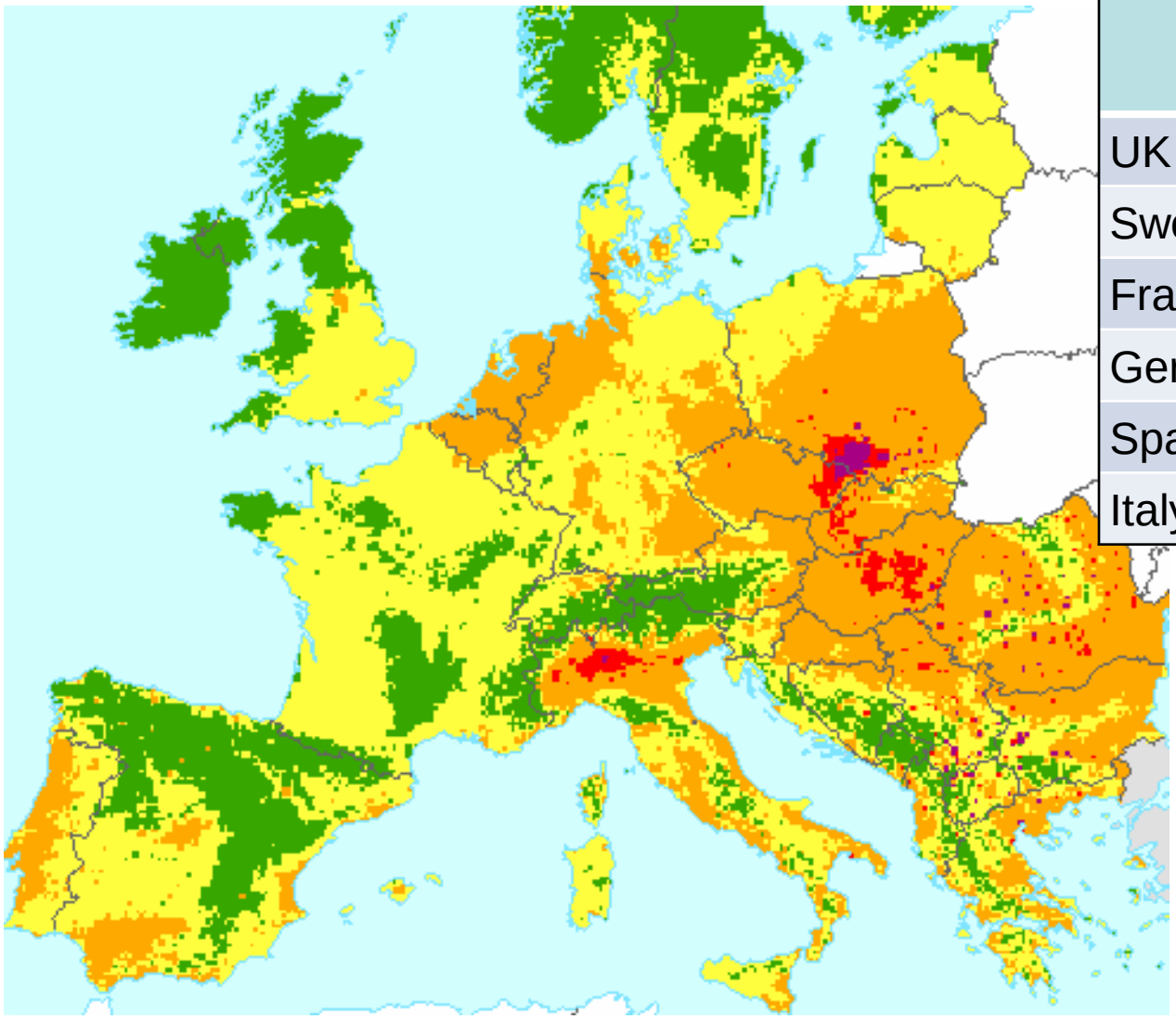
Exposure: PCM-modelled $PM_{2.5}$ in the UK



	Population-weighted $PM_{2.5}$ exposure μg m^{-3}
Scotland	5.5
Wales	8.3
Northern Ireland	6.4
Inner London	14.1
Outer London	13.4
Rest of England	10.6

(2010)

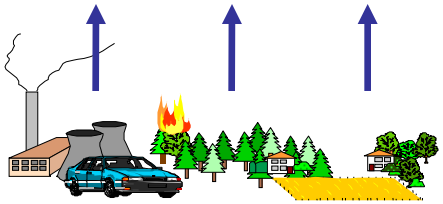
$PM_{2.5}$ across Europe



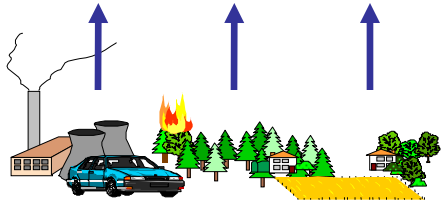
	Population-weighted $PM_{2.5}$ exposure µg m^{-3}
UK	10.3
Sweden	11.0
France	13.2
Germany	15.3
Spain	16.1
Italy	23.8

(2010)

Composition and sources



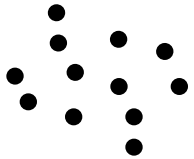
PRIMARY PARTICLE & GAEOUS EMISSIONS



DISPERSION

GAS & PARTICLE CHEMISTRY

Mixture of primary & secondary inorganic and organic particles



Receptor

Sources: anthropogenic & natural, local & distant

RECEPTOR MODELLING

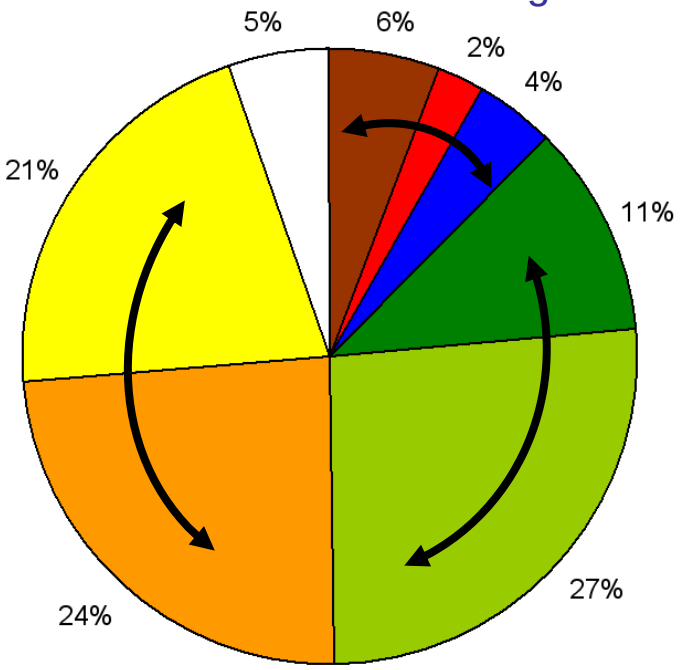
CHEMICAL-TRANSPORT MODELLING

Typical urban background $PM_{2.5}$ composition

Primary, mechanically generated

Secondary inorganic

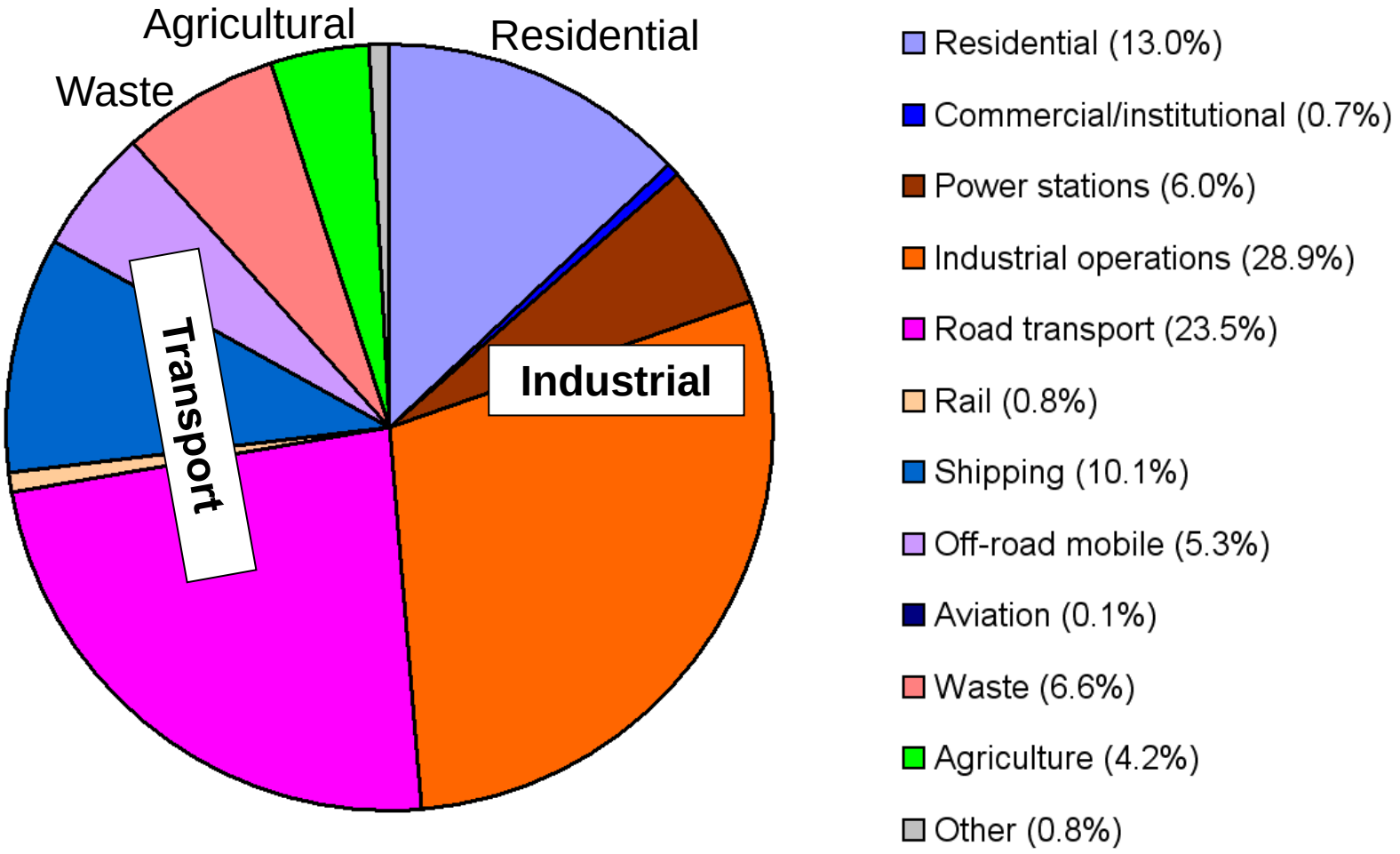
Primary and secondary carbonaceous



- Iron-rich dusts
- Calcium salts
- Sodium chloride
- Elemental carbon
- Organics
- Ammonium Sulphate
- Ammonium & sodium nitrate
- Other

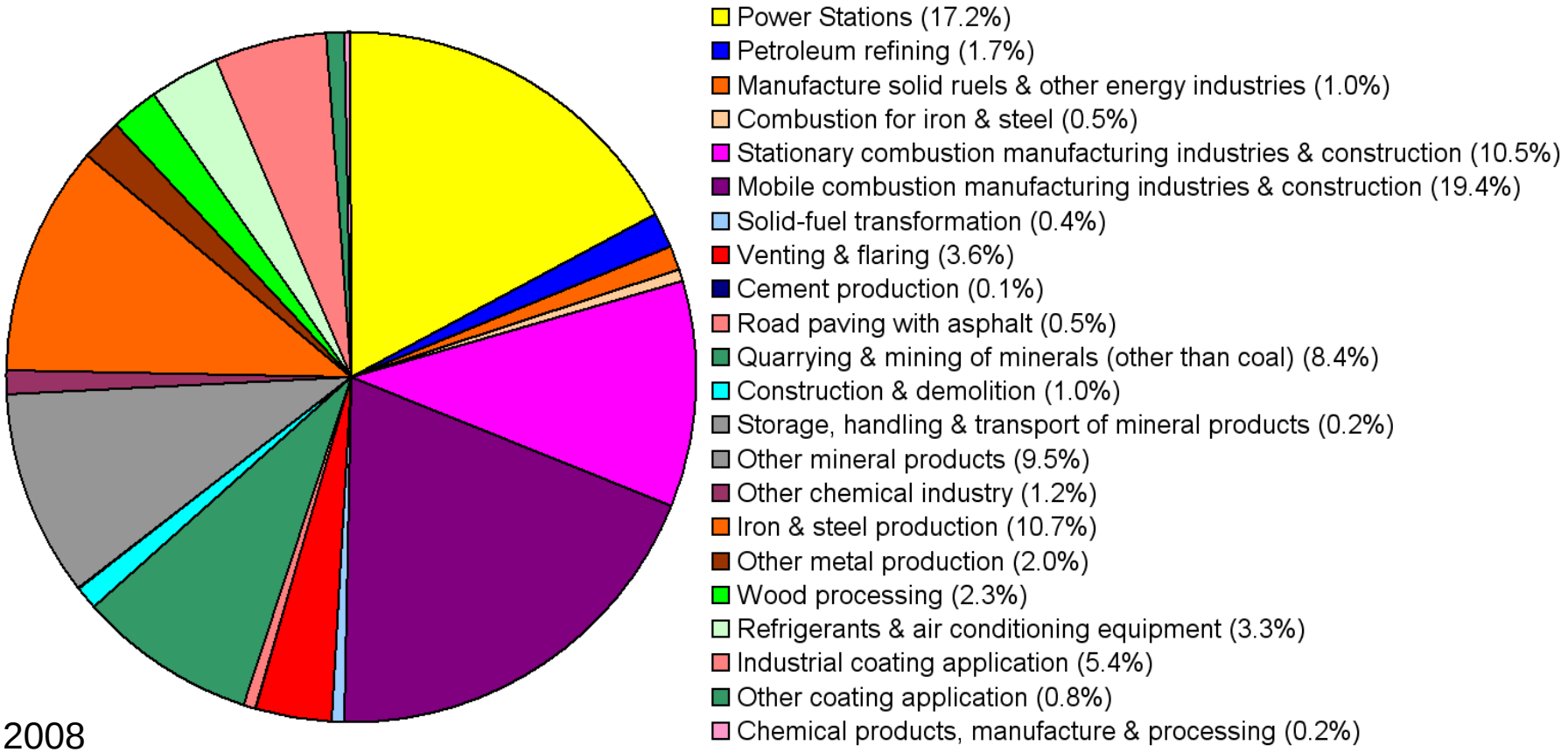
Measurements in Birmingham

NAEI emissions of primary $PM_{2.5}$ in the UK



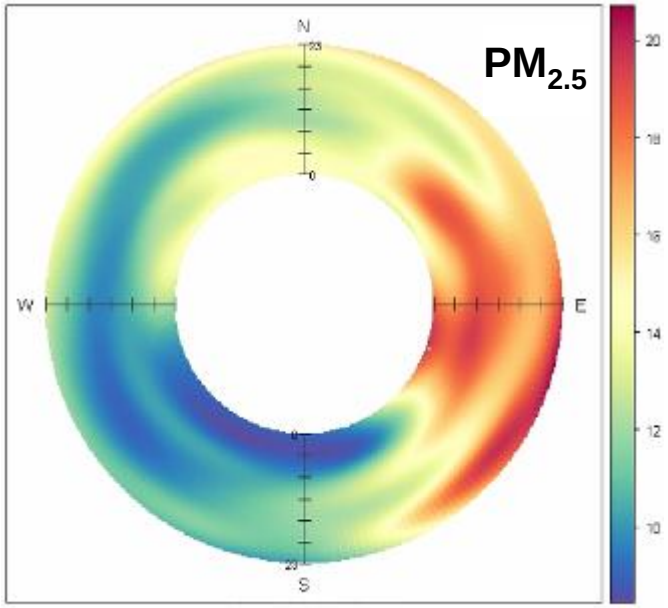
2008

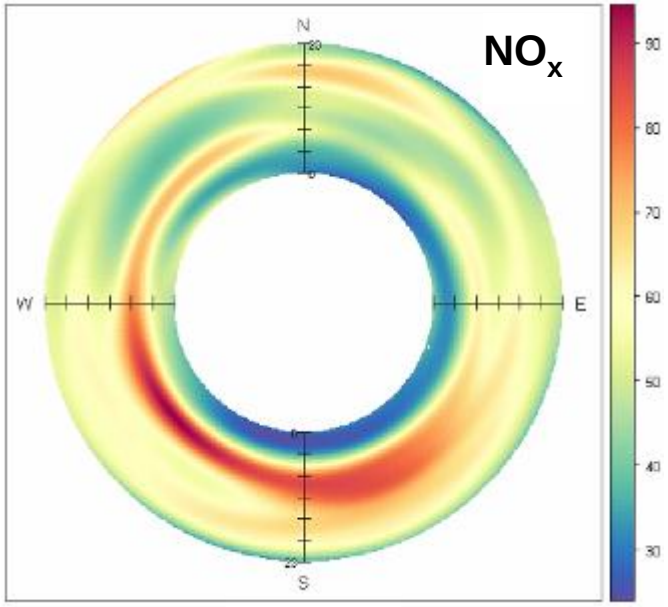
Industrial sources of primary $PM_{2.5}$ in the UK



2008

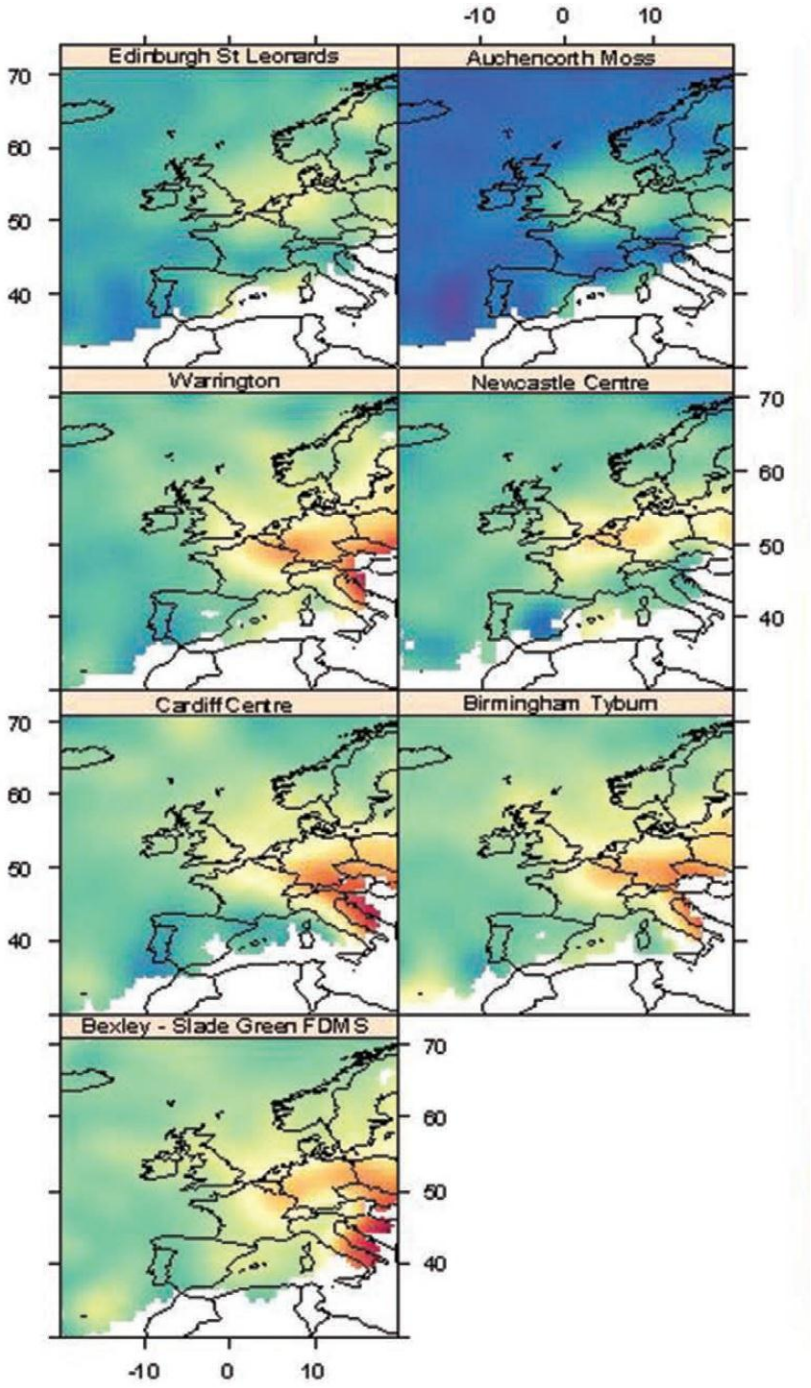
- Primary sources are many and diverse
  $\Rightarrow$ multi-sector abatement required





Polar annulus plots
(direction and time-of-day)

Southampton

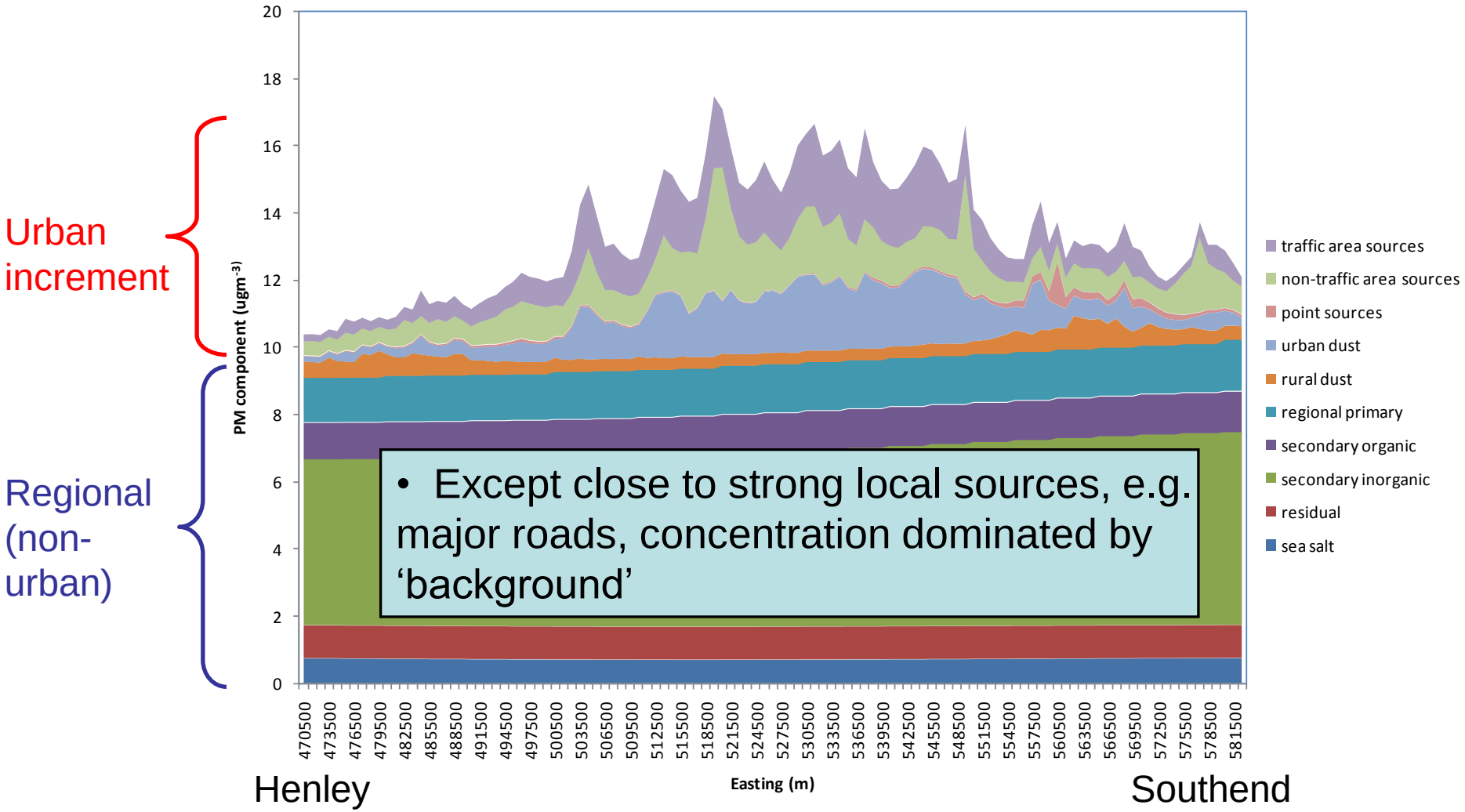


Air-mass back-trajectory analysis for $PM_{2.5}$

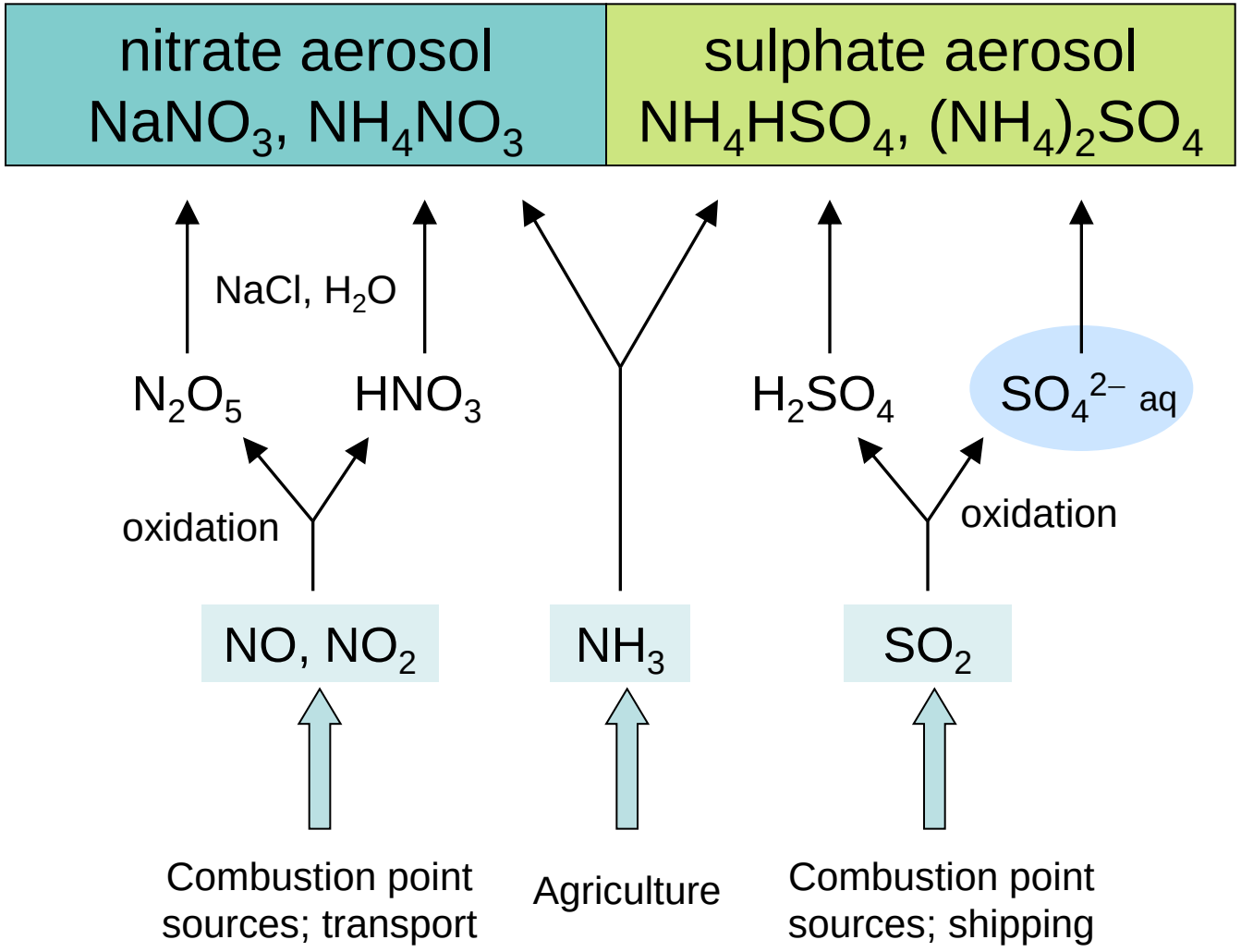
- Important influence of continental sources for elevated $PM_{2.5}$

(2009)

$PM_{2.5}$ source apportionment for west-east transect across London (PCM model)



Formation of secondary inorganic aerosol



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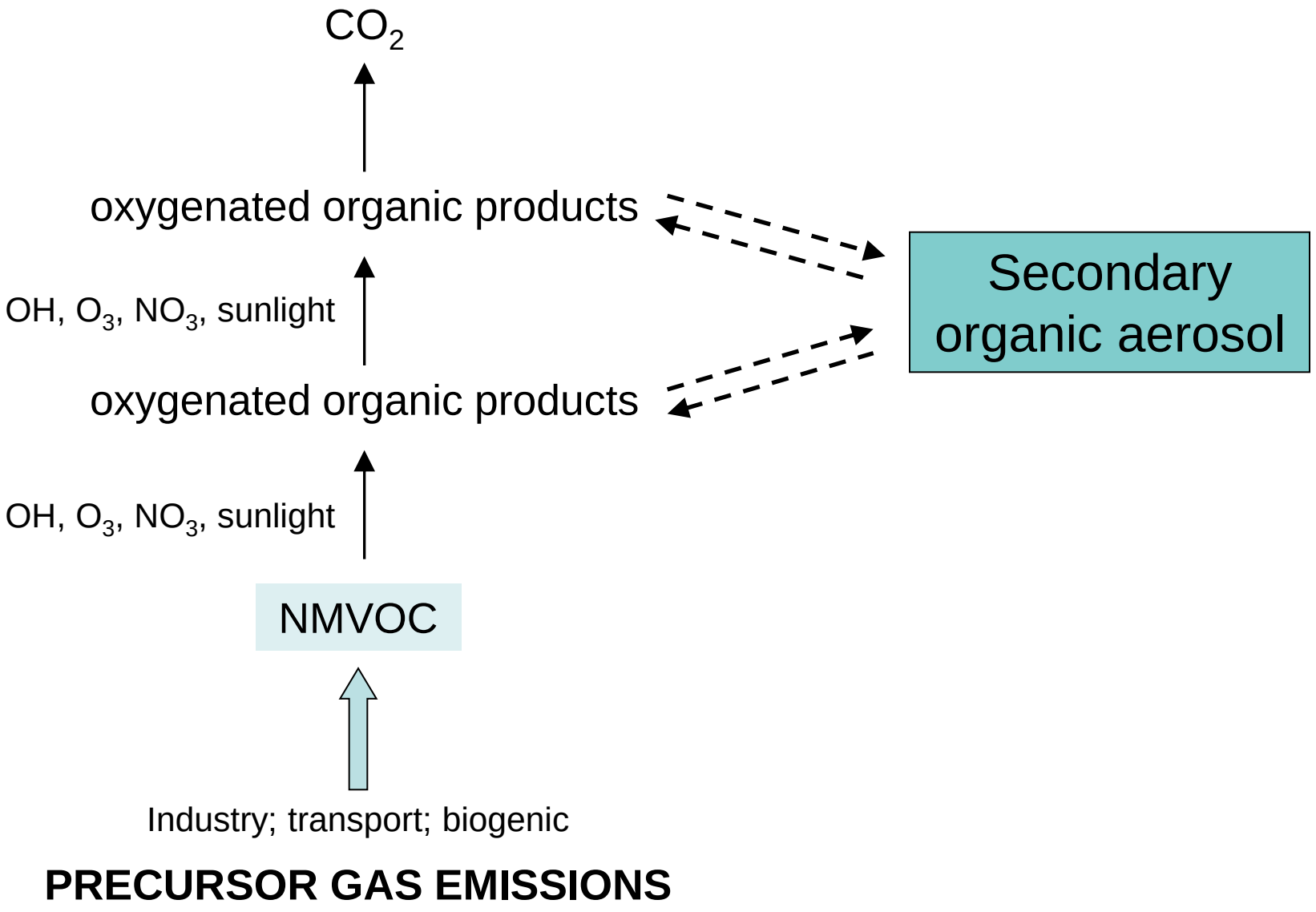
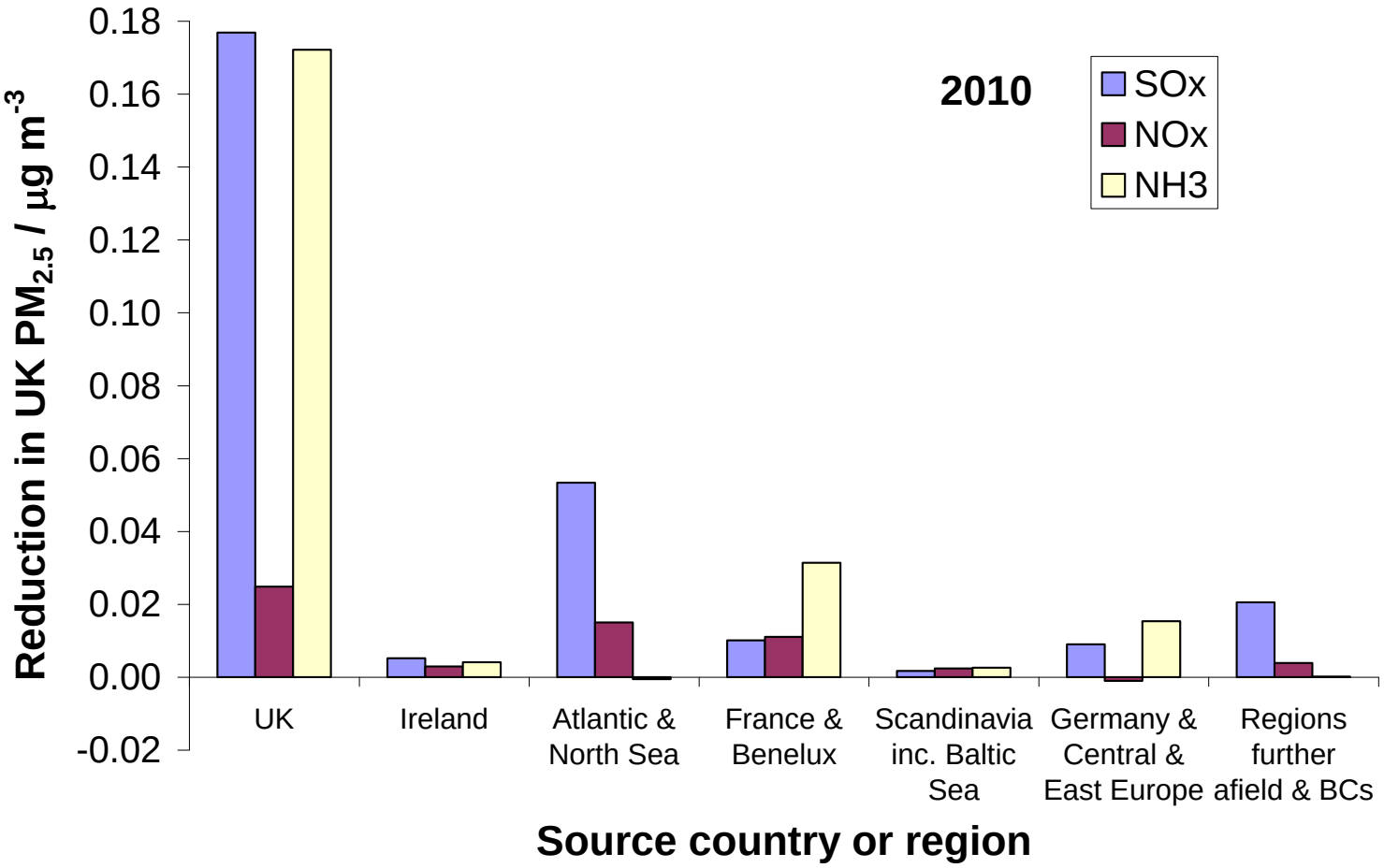
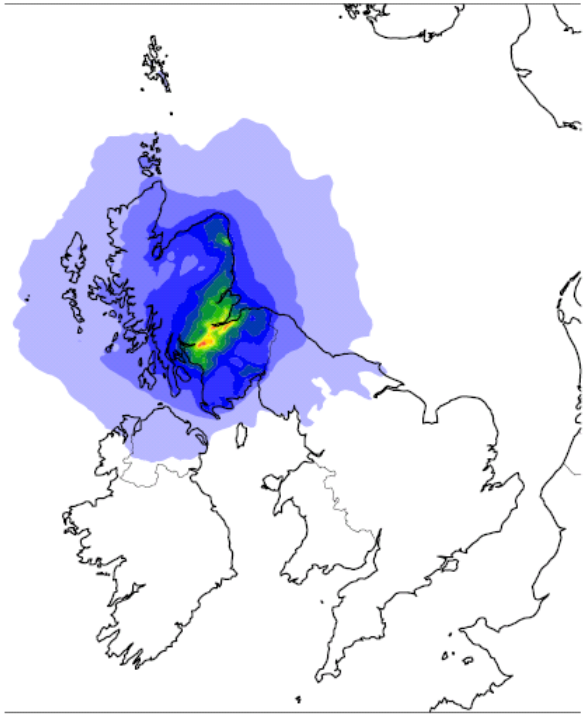


Reductions in UK SIA $PM_{2.5}$ from 15% reductions in SO_x, NO_x or NH_3 emissions in different regions

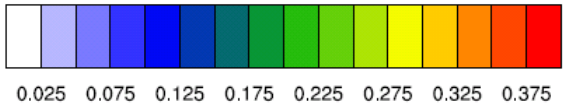


Sensitivity of $PM_{2.5}$ to 30% reduction in all Scottish emissions of primary $PM_{2.5}$, SO_x, NO_x & NH_3

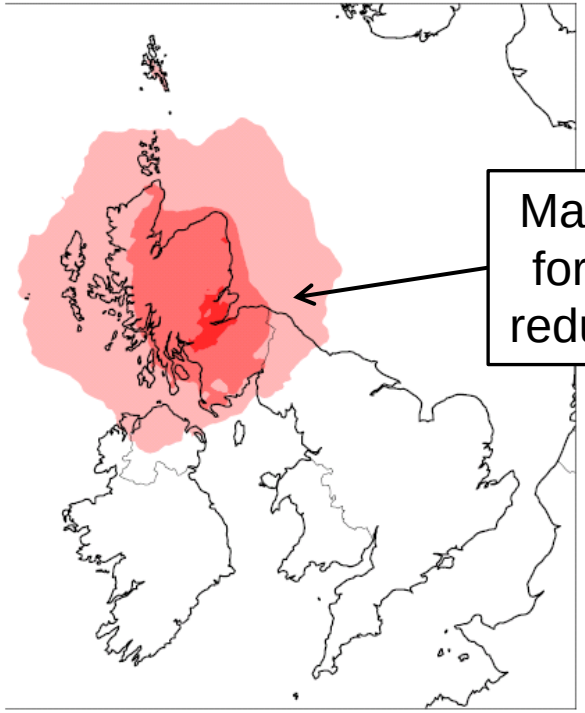
Reduction in $PM_{2.5}$ ($\mu g\ m^{-3}$)



$PM_{2.5}$

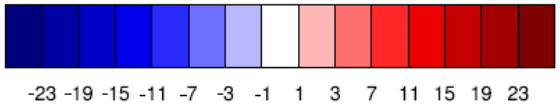


% reduction in $PM_{2.5}$



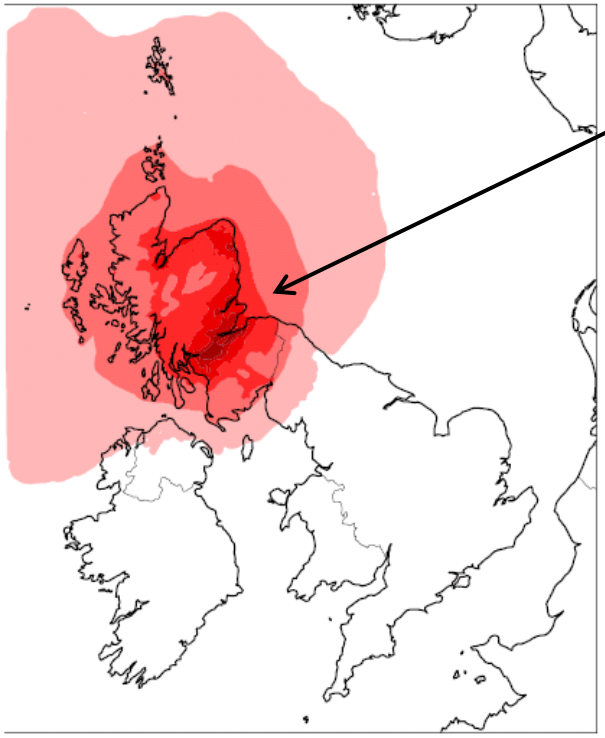
Max reduction ~10%; for most of Scotland reduction is only 3-7%

Δ % $PM_{2.5}$

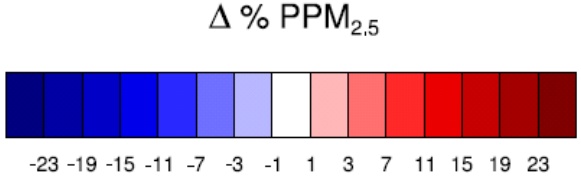


Sensitivity of primary $PM_{2.5}$ to 30% reduction in Scottish emissions of primary $PM_{2.5}$

% reduction in primary $PM_{2.5}$

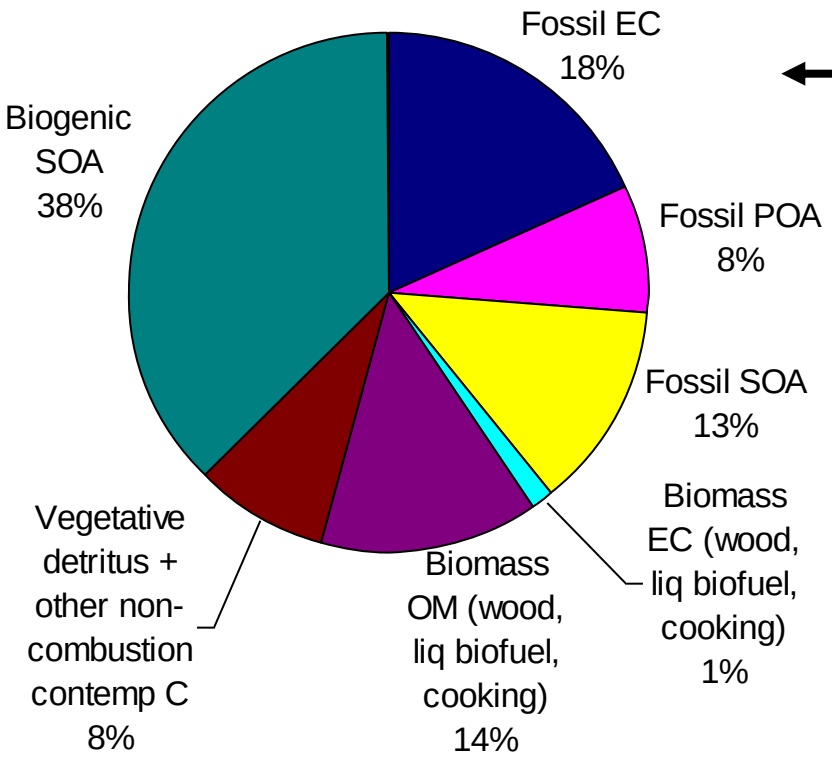


Reductions up to 23% in central belt, 11-15% over most populated areas



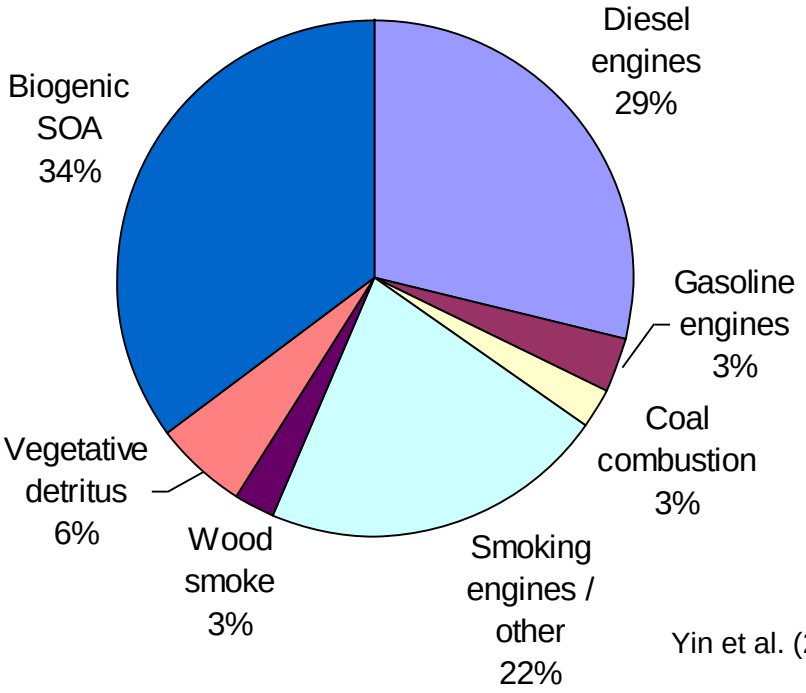
- Response of $PM_{2.5}$ to precursor emissions reductions is highly non-proportional
- Significant contribution to UK SIA from non-UK emissions
- UK SIA is most sensitive to reductions in NH_3 and SO_x – insensitive to NO_x emissions reductions on their own
- Greatest 'local' leverage on $PM_{2.5}$ is via reduction in primary $PM_{2.5}$

Source apportionment of carbonaceous PM



…by carbon-14 (plus some assumptions)

…by organic molecule tracers and PMF-CMB



Yin et al. (2010)

• Significant non-fossil carbon contributions: biomass/biofuel, cooking, primary biological material, and – in particular – biogenic SOA

Some final remarks

- Exposure varies across the UK but is dominated by the background, except adjacent to strong sources
- ~6 month reduction in life expectancy from current exposures; toxic component(s) remain uncertain, but any reduction in $PM_{2.5}$ has potential (and significant) health gain
- Sources of $PM_{2.5}$ are many and diverse making control a challenge; reductions in primary $PM_{2.5}$ remain an effective 'local' lever, reductions in SIA and SOA require considered transnational action
- There are co-benefits from emissions reductions aside from on $PM_{2.5}$ (e.g. on O_3 generation, and on eutrophic, acid & metal deposition)