Health Impact Assessment of specific PM species and the relation with NO_2

Mike Holland
mike.holland@emrc.co.uk
EMRC
and
COMEAP
(UK Committee on the Medical Effects of Air Pollutants)

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Current approach to quantification

- Treat all particles as equally harmful per unit mass
- Quantify health impacts for $PM_{2.5}$, NO_2, O_3 individually
- Possibly quantify effects of trace pollutants - toxic metals, PAHs and some other organics individually

- Combine estimates in some way to generate overall estimate of damage or benefit
  - HRAPIE: up to 30% cut in NO_2 mortality damage
  - CAO3: Totals with and without NO_2 used in the CBA (made no difference to conclusions from overall CBA)
  - Possible use of 2-pollutant models

Ideal approach to improving air quality

- Target the sources known to cause most harm, recognising
  - Differing levels of toxicity (etc.) of pollutants including different fractions of $PM_{2.5}$
  - Different composition of pollutants from each source
  - Costs of targeting specific sources, specific pollutants and specific fractions of $PM_{2.5}$

- Doing so would optimise the efficiency of pollution control strategies if based on reliable evidence

Problems…

- Mass of $PM_{2.5}$ does not account for:
  - The toxicity of different components of PM
    - Metals, organics, secondary inorganics…
  - The toxicity of different size fractions of PM

- Addition of impacts across pollutants should account for covariance in pollutant exposure
  - PM+NO_2 likely to overestimate impact
  - PM or NO_2 likely to underestimate impact
  - Results of 2-pollutant models are variable

Example of 2-pollutant models

- ELAPSE study
  - https://www.healtheffects.org/system/files/brunekreef-rr-208-report_0.pdf

Table 8. Hazard Ratios for Associations Between Air Pollution and Natural-Cause Mortality in Two-Pollutant Models[a,b]

Pollutant	Single Pollutant HR (95% CI)	HR (95% CI) Adjusted for $PM_{2.5}$	HR (95% CI) Adjusted for NO_2	HR (95% CI) Adjusted for BC	HR (95% CI) Adjusted for O_3
$PM_{2.5}$	1.130 (1.106, 1.155)	NA	1.083 (1.054, 1.113)	1.092 (1.062, 1.123)	1.089 (1.061, 1.117)
NO_2	1.086 (1.070, 1.102)	1.050 (1.031, 1.070)	NA	1.074 (1.038, 1.112)	1.053 (1.032, 1.074)
BC	1.081 (1.065, 1.098)	1.039 (1.019, 1.060)	1.012 (0.977, 1.048)	NA	1.044 (1.024, 1.065)
O_3	0.896 (0.878, 0.914)	0.935 (0.913, 0.957)	0.940 (0.914, 0.966)	0.930 (0.906, 0.955)	NA

[a] NA = Not applicable; N = 325,367. HR (95% CI) presented for the following increments: $PM_{2.5}$, 5 μg/m³; NO_2, 10 μg/m³; BC, 0.5 × 10^{-5}/m; O_3, 10 μg/m³. Main model adjusted for cohort ID, age, sex, year of baseline visit, smoking (status, duration, intensity, intensity squared), BMI, marital status, employment status, and 2001 neighborhood-level mean income.

[b] Two-pollutant models of BC and NO_2 are difficult to interpret because of high correlation between BC and NO_2.

  - But results are variable between studies – other examples would show different levels of adjustment

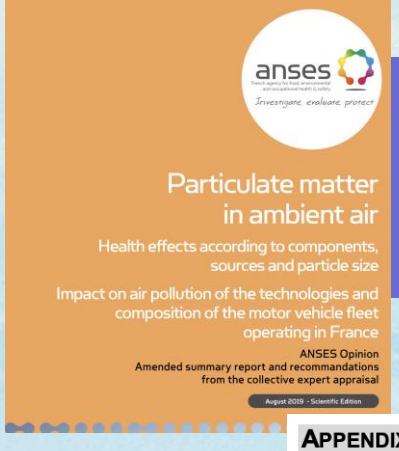
COMEAP
COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS

All COMEAP reports and statements available at:
https://www.gov.uk/government/collections/comeap-reports

Committee on the Medical Effects of Air Pollutants

Statement on the evidence for health effects associated with exposure to non-exhaust particulate matter from road transport

- Given that non-exhaust particles have a different composition (eg higher metal concentrations) and size distribution from those emitted in vehicle exhausts, they may have different toxicological properties and health consequences.
- However…
  - Toxicological studies suggest that tyre and brake wear particles have the potential to induce biological effects at higher concentrations, but it is unclear whether real world exposures are high enough for this to be of concern for health.
  - Epidemiological studies of health impacts are not consistent.
  - Several of the studies reviewed found no associations of health outcomes with non-exhaust particles, others did report significant associations but covered a considerable range of different health effects.
  - However, the likely strong correlation of non-exhaust particles with exhaust pollutants in ambient air makes it difficult to identify an independent effect of non-exhaust particles.



ANSES report

https://www.anses.fr/fr/system/files/AIR2014SA0156RaEN.pdf

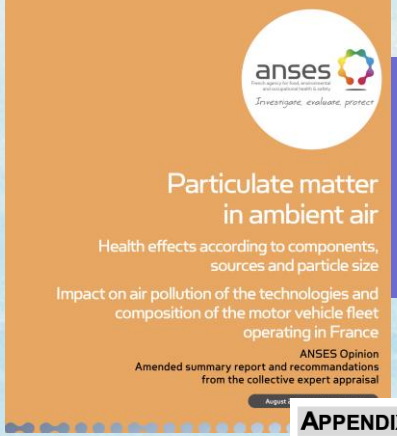
APPENDIX 4 – SUMMARY OF ACCUMULATED EVIDENCE AND LEVELS OF EVIDENCE FOR HEALTH EFFECTS OF EXPOSURE TO AMBIENT AIR PARTICULATE COMPONENTS FROM HUMAN STUDIES IN REVIHAAP AND SINCE REVIHAAP

Component		In REVIHAAP: Reported level of evidence	In REVIHAAP: Associations reported†	Since REVIHAAP and up to February 2016: Highest level of evidence for an adverse health effect[θ] (number of publications in the body of evidence)		Summary of the accumulated evidence: Evidence or associations reported in REVIHAAP | Evidence for an adverse health effect since REVIHAAP
Ultrafine particles (<100 nm)		Evidence still limited	Yes	🫀	Moderate (14) (+Exp.*)	+ | ↗
Coarse particles ($PM_{2.5-10}$)		Suggestive evidence	Yes	🫁 ☠	Moderate (44)	+ | ↗
Carbonaceous materials	Black carbon, elemental carbon	Sufficient evidence	Yes	🫁 🫀 ☠	High (78) (+Exp.*)	+ | ↗
	Organic carbon	Increasing information	Yes	🫁 🫀 ✚	High (37) (+Exp.*)	+ | ↗
	SOAs (source factor€)	Ø	Yes		Inadequate (7) (+Exp.*)	+ | →
	PAHs	Ø	Ø	🫁	Low (4)	Ø | →
Secondary inorganic aerosols	SIAs (source factor€)	Ø	Yes	🫁	Low (6)	+ | →
	Sulphate	Ø	Yes	🫁	High (48)	+ | ↗
	Nitrate	Ø	Yes	🫀	High (25) (+Exp.*)	+ | ↗
	Ammonium	Ø	Ø	🫁 🫀 ☠	Low (14)	Ø | →
Transition metals	Nickel	Ø	Yes§	🫁	High (34) (+Exp.*)	Ø | ↑
	Zinc	Ø	Yes§	🫀	Moderate (31)	Ø | ↑
	Copper	Ø	Yes§	🫀	Low (31)	Ø | →
	Vanadium	Ø	Yes§	🫀	High (32)	Ø | ↑
	Iron	Ø	Ø	🫀	High (31)	Ø | ↑
Silica		Ø	Ø	🫁 ☠	Moderate (30)	Ø | ↑
Endotoxins		Ø	Ø	🫀	Moderate (5)	Ø | ↑
Oxidative potential		Ø	Ø	🫁 🫀	Low (6)	Ø | →

In REVIHAAP:
+ : effect reported;
–: no effect reported;
Ø: not examined.

| Since REVIHAAP:
↑ : new evidence for an adverse health effect;
↗ : confirmation of the health effect;
→ : no new evidence for an adverse health effect (i.e. a "low" or "inadequate" level of evidence for an adverse effect);
↘ : evidence of no health effect;
Ø: no studies identified.

☠ all-cause mortality 🫁 respiratory health 🫀 cardiovascular health ✚ all-cause hospitalisations. θ The purpose of assessing the level of evidence for an adverse health effect is to conclude whether the observed association between a given component of ambient air particulate matter and a given health category (e.g. cardiovascular health) is highly, moderately or faintly plausible. It may also be concluded that a component has no effect on health, or that there is too little evidence available in the literature to be able to reach any conclusion. *Abbreviations: SIAs: secondary inorganic aerosols; SOAs: secondary organic aerosols; PAHs: polycyclic aromatic hydrocarbons. * The conclusions based on animal experimental studies support this evidence of effects. † Yes if association reported, no if no association reported, Ø if not reported. § The reported associations do not specifically concern this element and include other transition metals in mixtures. Defined according to a statistical method of allocating components into categories or sources (e.g. positive matrix factorisation).*



ANSES report

https://www.anses.fr/fr/system/files/AIR2014SA0156RaEN.pdf

APPENDIX 5 – SUMMARY OF ACCUMULATED EVIDENCE AND LEVELS OF EVIDENCE FOR HEALTH EFFECTS OF EXPOSURE TO SOURCES OF AMBIENT AIR PARTICULATE MATTER FROM HUMAN STUDIES IN REVIHAAP AND SINCE REVIHAAP

Source category		In REVIHAAP: Reported level of evidence	In REVIHAAP: Associations reported†	Since REVIHAAP and up to February 2016: Highest level of evidence for an adverse health effectθ (number of publications in the body of evidence)	Accumulated evidence: Evidence or associations reported in REVIHAAP | Evidence for an adverse health effect since REVIHAAP	
Road traffic	Traffic-related $PM_{2.5}$	Probable association	Yes	Moderate (16) (+Exp.*)	+	↗
	Traffic-related black carbon	Sufficient evidence∓	Ø	High (4)	+	↗
	Diesel engine exhaust	Ø	Yes	Moderate (3) (+Exp.*)	+	↗
	Gasoline exhaust	Ø	Yes	Moderate (1)	+	↗
	Road dust, crustal material	Probable association	Yes	High (16)	+	↗
	Brake dust	Ø	Ø	Low (1) #	Ø	→
Combustion	Coal combustion	Solid evidence‡	Yes	High (7) (+Exp.*)	+	↗
	Combustion of petroleum products	Influence on health	Yes and no	Moderate (10)	+	↗
Industry	Industry	Ø	Yes	Low (5)	+	→
	Metallurgy	Ø	Yes	Inadequate (10) #	+	→
Biomass combustion		Probable association	Yes§	Inadequate (4) #	+	→
Desert dust		Ø	Yes	Moderate (3)	+	↗
Sea salt and sea spray		Suggestion of no effect	Yes and no	Low (9)	–	→
Hazardous waste site		Ø	No	Inadequate (1)	–	→
Rural environment and agriculture		Ø	Ø	Ø	Ø	Ø

In REVIHAAP:
+ : effect reported;
–: no effect reported;
Ø: not examined.

Since REVIHAAP:
↑ : new evidence for an adverse health effect;
↗ : confirmation of the health effect;
→ : no new evidence for an adverse health effect (i.e. a "low" or "inadequate" level of evidence for an adverse effect);
↘ : evidence of no health effect;
Ø: not examined.

all-cause mortality; respiratory health; cardiovascular health; neurological health. θ The purpose of assessing the level of evidence for an adverse health effect is to conclude whether the observed association between a given source of ambient air particulate matter and a given health category (e.g. cardiovascular health) is highly, moderately or faintly plausible. It may also be concluded that a source has no effect on health, or that there is too little evidence available in the literature to be able to reach any conclusion. * *The conclusions based on animal experimental studies support this evidence of effects.* † *Yes if association reported, no if no association reported, Ø if not reported.* ∓ *This level of evidence is based on the accumulated evidence for black carbon.* ‡ *This level of evidence is based on the accumulated evidence for sulphate-enriched particles (chemical tracer of coal combustion).* § *In particular, associations between forest fires and health events.* # *Does not consider evidence for health effects that may have been observed for individual chemical components or tracers linked to this source.*

COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS

Guidance

Statement on the differential toxicity of particulate matter according to source or constituents: 2022

- Update to 2015 COMEAP statement taking account of ANSES and USEPA and other sources
  - Different sources and constituents of PM are associated with adverse health outcomes.
  - PM associated with combustion and road traffic received the most attention.
  - Insufficient evidence to conclude that these sources of PM played a significantly greater role than other sources or constituents.
  - The substantial body of recent evidence adds weight to the conclusion that different constituents are likely to have different toxicological actions,
  - But it does not consistently indicate specific components of PM that are more toxic than others.
  - At present, $PM_{2.5}$ remains the most suitable metric for evaluating the health impacts, and hence for regulating PM in the atmosphere.

Looking backwards, looking forwards

- Concerns about lack of account for variation in harm across the mix of different types of PM raised at least 30 years ago, have yet to be resolved
- Evidence of harm from different fractions has grown, but still…
- $PM_{2.5}$ remains the most suitable metric for evaluating the health impacts, and hence for regulating PM in the atmosphere.
- Important questions:
  - What needs to be done to generate sufficient experimental evidence to support separate treatment of different fractions?
  - How long would that take, and what would the pollution climate be by that time (recognising increasing air pollution controls, electrification, etc.)?
- Suggestions
  - Define precise policy questions where current tools are seriously deficient
  - More work on 2-pollutant models to try to resolve the PM-NO_2 overlap
  - Review of pollutant mixes from different sources by toxicologists and others to highlight those that they consider most problematic, accounting for both primary and secondary pollutants