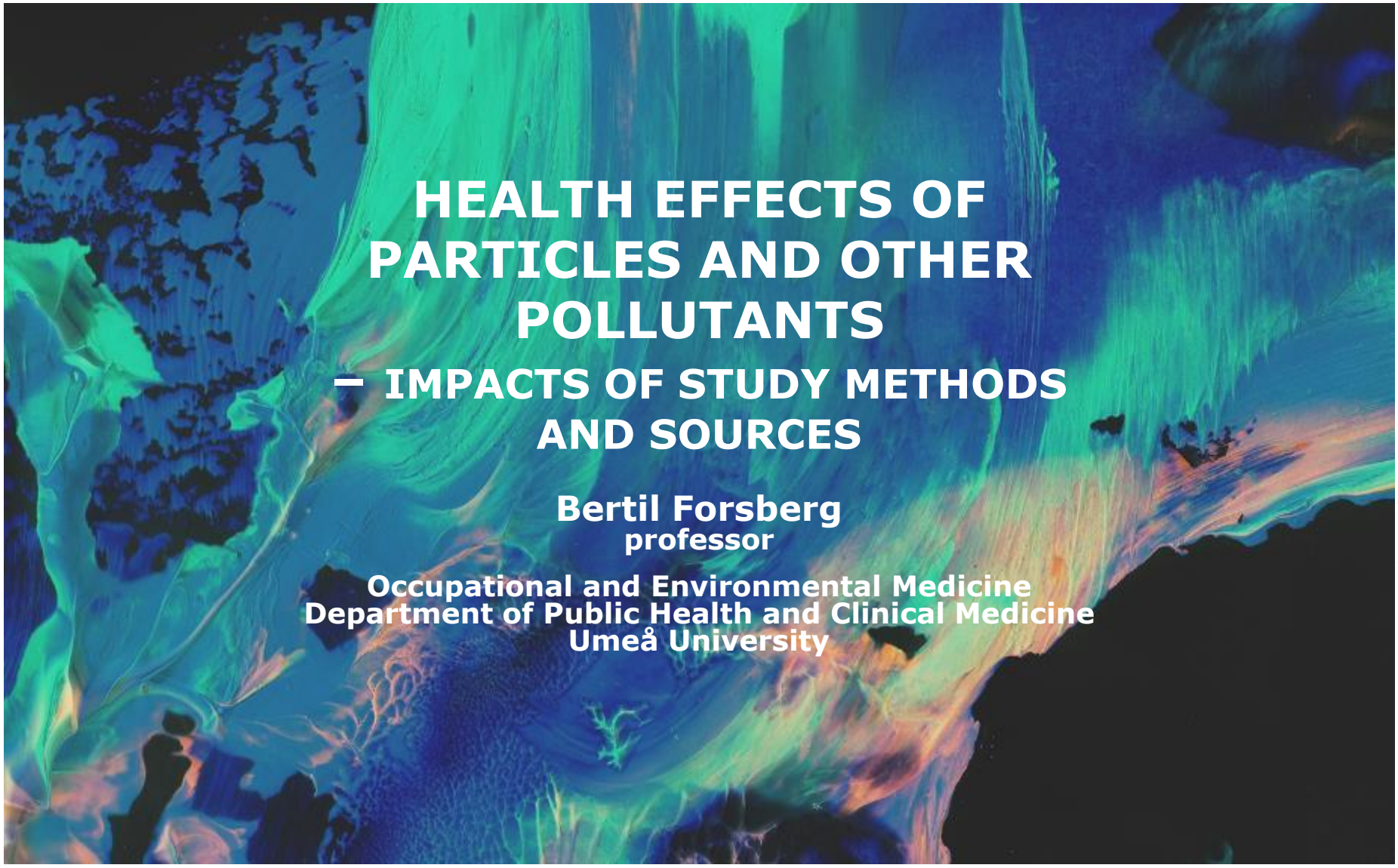

HEALTH EFFECTS OF PARTICLES AND OTHER POLLUTANTS

– IMPACTS OF STUDY METHODS AND SOURCES

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A MAJOR HEALTH PROBLEM

- Ambient PM globally over 4 mi deaths 2016 (GBD Study, Lancet 2017)
- Ambient PM is the 6th and 7 th leading risk factor for DALYs in women and men

A GROWING PROBLEM – OR?

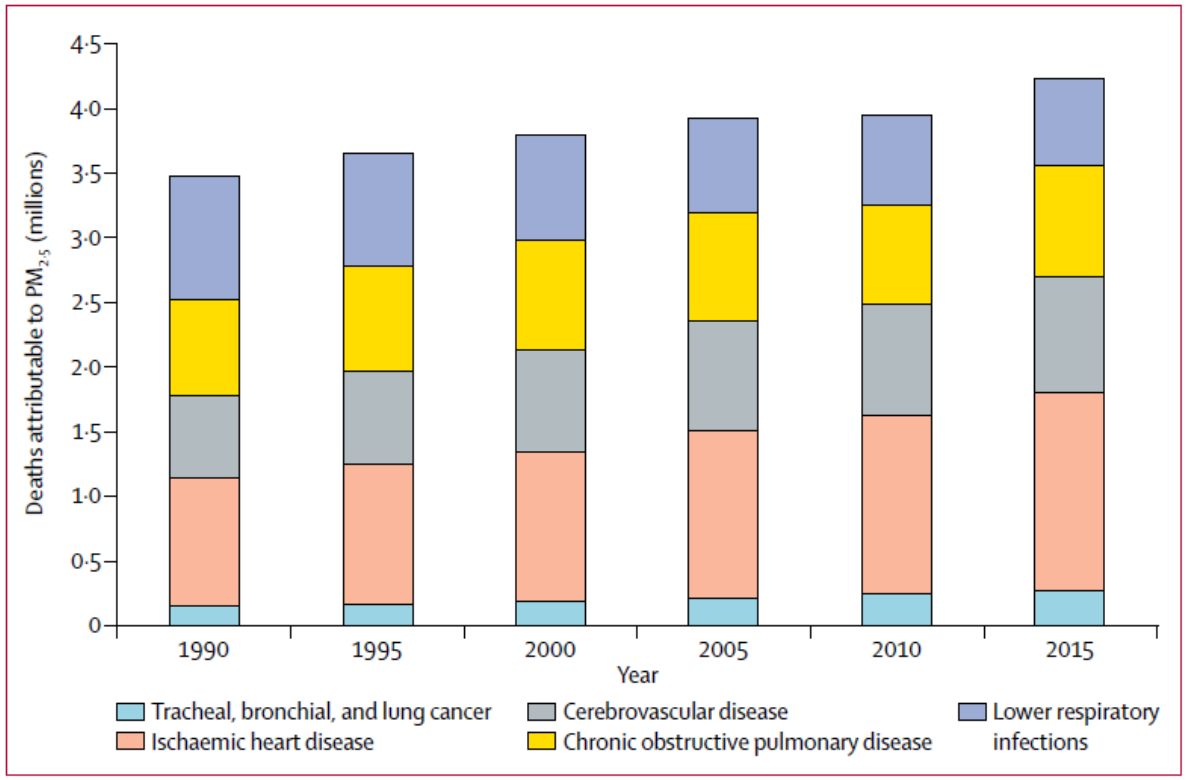

Figure 4: **Deaths attributable to ambient particulate matter pollution by year and cause**
$PM_{2·5}$=particle mass with aerodynamic diameter less than 2·5 μm.

(Cohen et al, 2017)

IMPACT OF EXPOSURE IS REDUCED BY DECREASE IN MORTALITY RATES

- The absolute number of PM2.5 related deaths was globally estimated to increase from 3.5 to 4.2 mi (1990-2015)
- This was the result of:
  - an estimated increase in PM2.5 exposure (pop weigthed)
  - a growing and ageing population
  - a decrease in age-standardized total mortality rates
- However, globally age-standardized mortality rates due to PM2.5 decreased

MORE THAN ABOUT LUNGS – AND EVEN MORE?

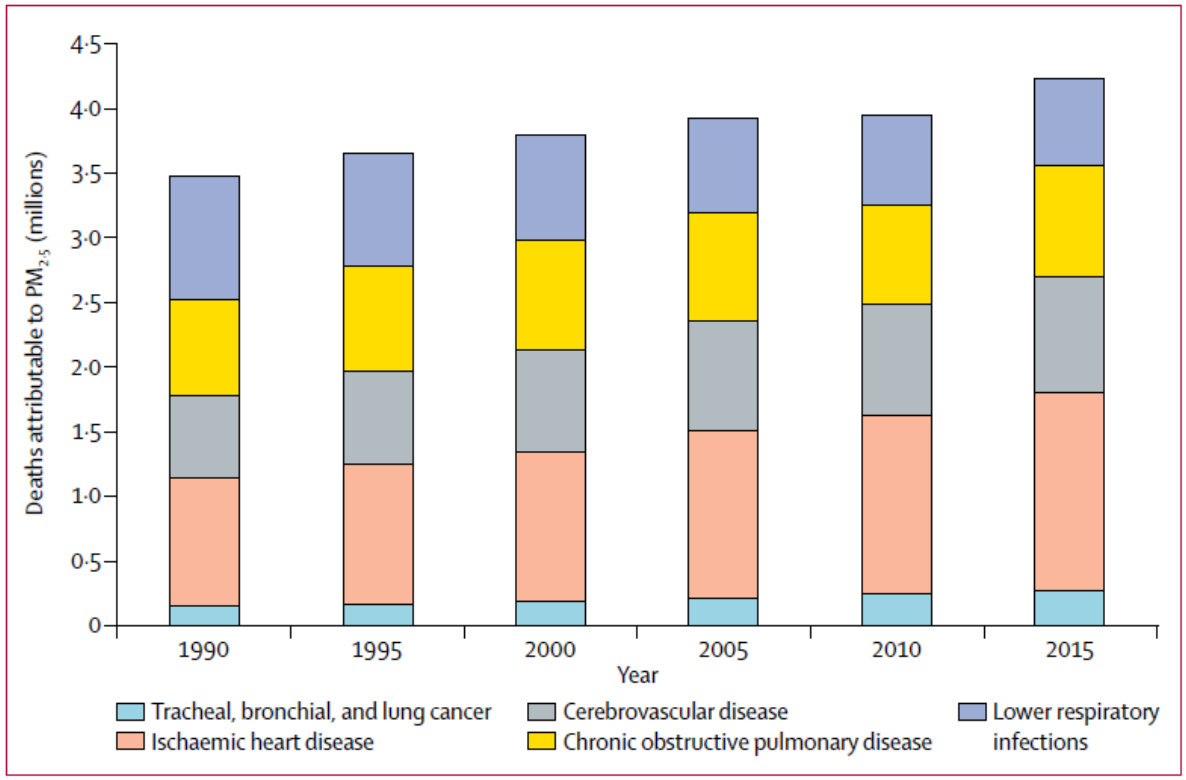

Figure 4: **Deaths attributable to ambient particulate matter pollution by year and cause**
$PM_{2.5}$=particle mass with aerodynamic diameter less than 2·5 µm.

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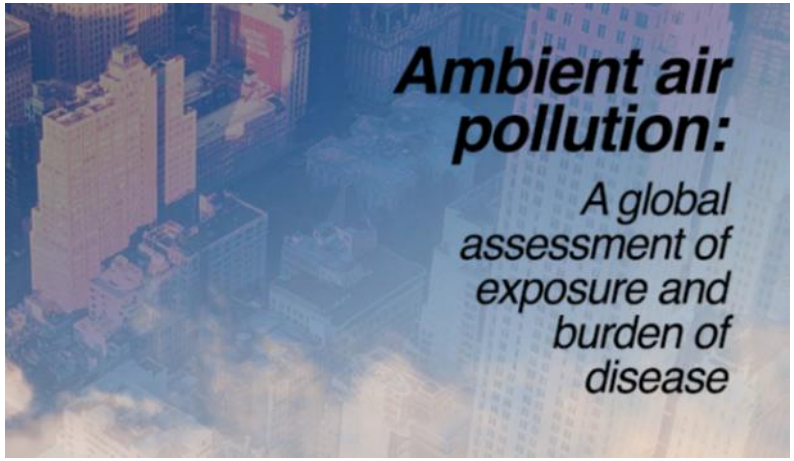
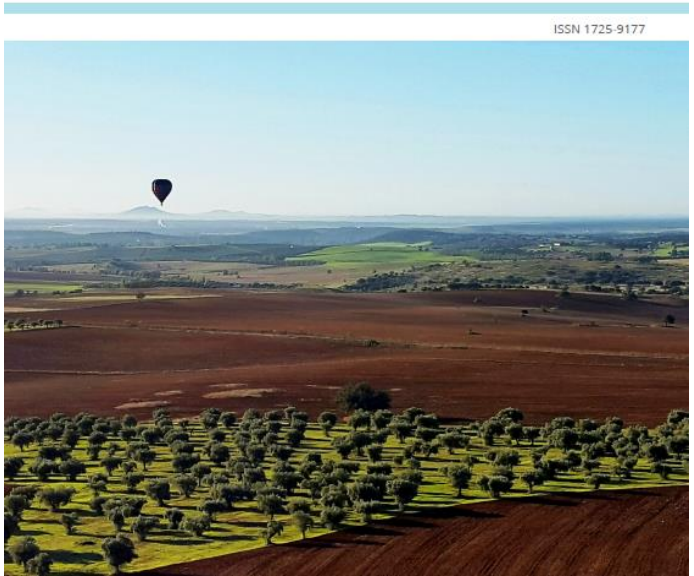
HOW WELL DO STUDIES REFLECT HEALTH EFFECTS AND IMPACTS?



Depend on outcomes and study methods

Estimated effects of sources and pollutants



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HEALTH IMPACT ASSESSMENTS OF AIR POLLUTION EXPOSURE

- Global and large scale assessments have used RRs from between city analyses or studies with poor spatial resolution, typically from ACS: 6% increase in mortality per 10 µg/m3 PM2.5 (Pope et al, 2002)
- Local assessments and those focussed on specific emissions e.g. diesel particles or wood smoke particles have often used the same RRs
- This has long been questioned (Forsberg et al, 2005), and studies have suggested higher RRs for local sources and a finer spatial resolution (Jerrett et al, 2005)
- Reports from WHO and EPA have concluded that toxicity likely is different, e.g. for BC, but not possible to recommend specific RRs for different particle types, components or sources
- More recent studies have suggested that NO2 has own effects on mortality in addition to the effects associated with PM2.5 and some assessments now include both

HOW TO ESTIMATE LOCAL EFFECTS OF LESS TRAFFIC POLLUTION?

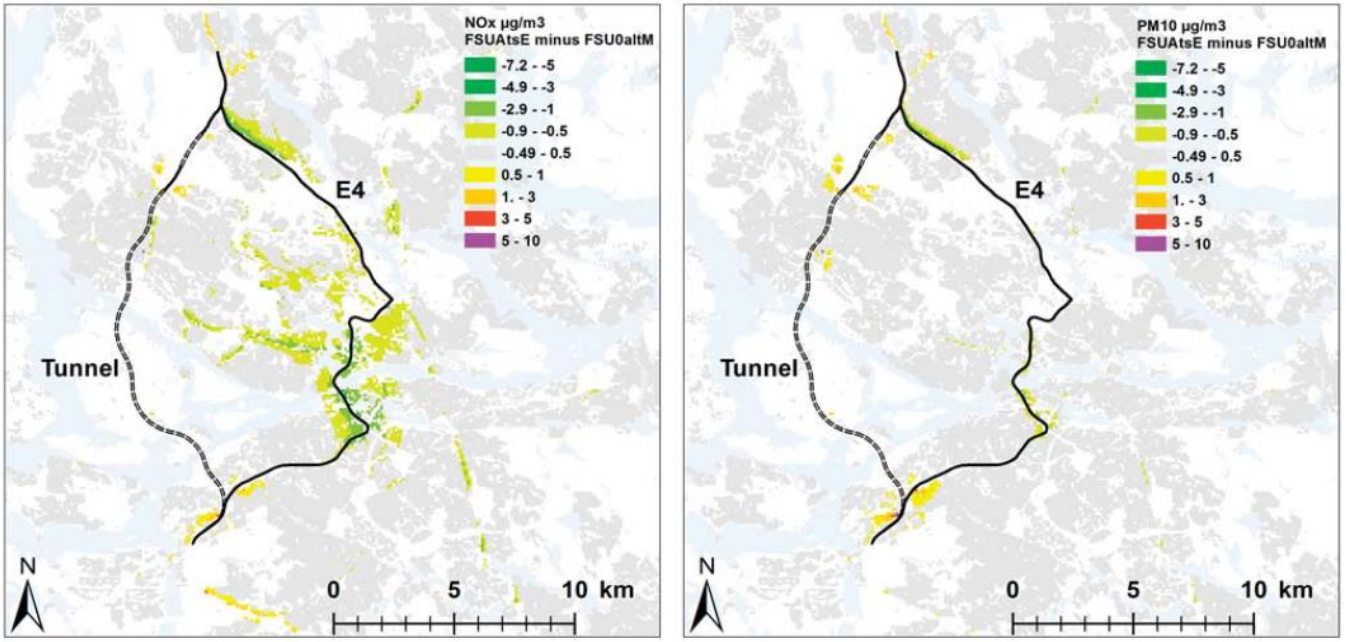

Figure 4. Change in NO_X and PM_{10} annual levels with the bypass Förbifart Stockholm. PM, particulate matter.

Journal of Exposure Science and Environmental Epidemiology (2015) 25, 524–531

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IMPORTANT EFFECTS OF STUDY METHODS AND DATA SOURCES

- Early cohort studies assigned all individuals in a city the same exposure, which may be ok for pollutants with large variations between regions and small within cities (e.g. sulphate), but not for those with high spatial variability (e.g. BC)
- Modern cohort studies use home address (or at least a fine spatial resolution) when exposure is assigned from dispersion models or LUR models with data on local sources, e.g. traffic
- Using such methods the risk related to within-city contrast can be studied instead of between-city contrasts
- Hybrid models with Global chemical transport models and/or satellite data (absorption/scattering of specific wavelengths) are now used with monitoring data or land use data

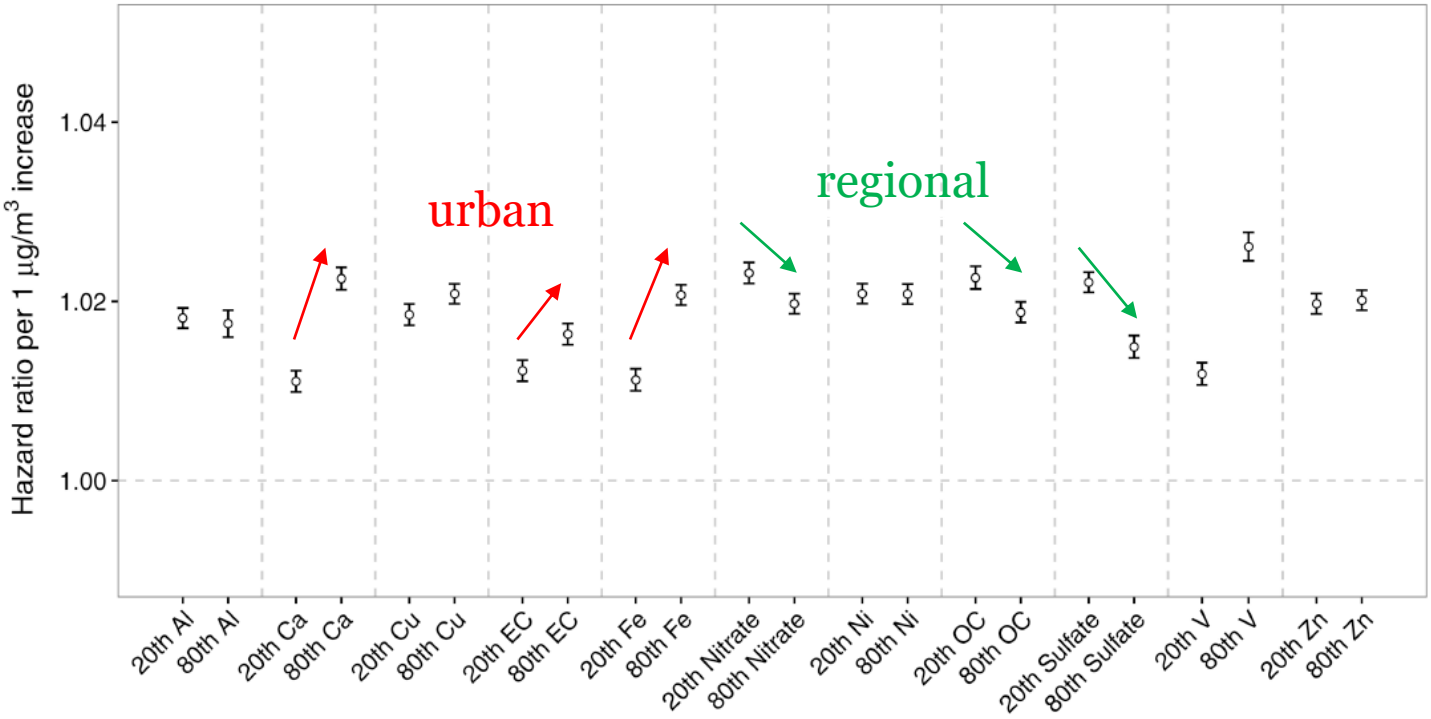
A HYBRID MODEL FOR PM2.5 AND COMPONENTS IN SOUTH EAST US

(WANG Y ET AL, 2017)

- Mortality in persons aged 65+
- Satellite data + monitoring data + land use data where use in three stages to predict PM2.5 and components in 1*1 km grids
- Exposure was estimated based on the zip code
- RR (HR) was much higher than in between-city comparisons in US: 21% per 10 µg/m3 PM2.5 (95% CI 19-22)
- RR was higher in urban areas, and below EPA standard (12) RR 33% per 10 µg/m3 (95% CI 31-35)
- The risk associated with PM2.5 changed with proportion of components such as EC and sulfate

PM2.5 RR CHANGED WITH HIGH VS LOW PROPORTION OF COMPONENTS

(WANG Y ET AL, 2017)



EXPOSURE TO REGIONAL AND NEAR SOURCE PM2.5 AND MORTALITY
(TURNER ET AL, 2016)

- A Hybrid LUR model was used to study long-term exposure and mortality in the large prospective ACS Cohort
- Regional and ”near source” PM2.5 were simultaneously included in the model, producing very different RRs per 10µg/m3

Table 2. All-Cause and Cause-Specific Mortality Hazard Ratios in Relation to Each 10-Unit Increase in Air Pollutant Concentrations, 1982–2004 Follow-up in American Cancer Society Cancer Prevention Study II Cohort, United States (n = 669,046)

Cause of Death	ICD-9 Codes; ICD-10 Codes	Deaths (*n*)	Multipollutant Model Data, Fully Adjusted HR* (95% CI)			
			HBM O_3	Regional $PM_{2.5}$	Near-Source $PM_{2.5}$	LUR NO_2
All-cause mortality	All	237,201	1.02 (1.01–1.04)	1.04 (1.02–1.06)	1.26 (1.19–1.34)	1.01 (1.00–1.03)
Diseases of the circulatory system	390–459, 250; I00–I99, E10–E14	105,039	1.03 (1.01–1.05)	1.07 (1.04–1.10)	1.41 (1.29–1.54)	1.03 (1.01–1.05)

HOW CONSISTENT ARE RESULTS REGARDING COMPONENTS?

- Some studies report that traffic-related pollutants indicated by BC/EC pose a greater risk than PM2.5 from other sources
- Other studies report that other constituents of PM2.5 such as sulfate have more robust associations – methods may explain!
- Some papers focus on the RR per µg/m3 change, others presents RR for a relative change (IQR). What is most relevant depends on the question!
- Policy options usually mean that we discuss µg/m3

REVIEW OF STUDIES ON PARTICLE METRICS AND DAILY MORTALITY

(ATKINSON ET AL, 2015)

Table 2. Random effects summary estimates for particle metrics and all-cause and cause-specific mortality.

Pollutant	*Disease*	*All SC/MC*[a]	*Selected SC/MC*[b]	*RE (95% CI)*[c]	I^2 *(%)*[d]
SO_4^{2-}	All-cause	14/4	9/3	0.15 (0.06, 0.25)	71
	Cardiovascular	9/1	8/1	0.21 (–0.01, 0.44)	42
	Respiratory	8/1	7/1	0.23 (–0.07, 0.52)	38
NO_3^-	All-cause	6/1	5/1	0.17 (0.12, 0.23)	0
	Cardiovascular	6/1	5/1	0.11 (–0.12, 0.35)	70
	Respiratory	4/1	3/1	0.15 (–0.29, 0.59)	68
EC	All-cause	6/1	5/1	1.30 (0.17, 2.43)	92
	Cardiovascular	5/1	4/1	1.66 (0.52, 2.81)	97
	Respiratory	4/1	3/1	1.09 (–1.59, 3.85)	99
OC	All-cause	4/1	3/1	0.37 (–0.19, 0.94)	99
	Cardiovascular	5/1	4/1	0.56 (0.01, 1.10)	97
	Respiratory	4/1	3/1	0.57 (–1.11, 2.28)	98

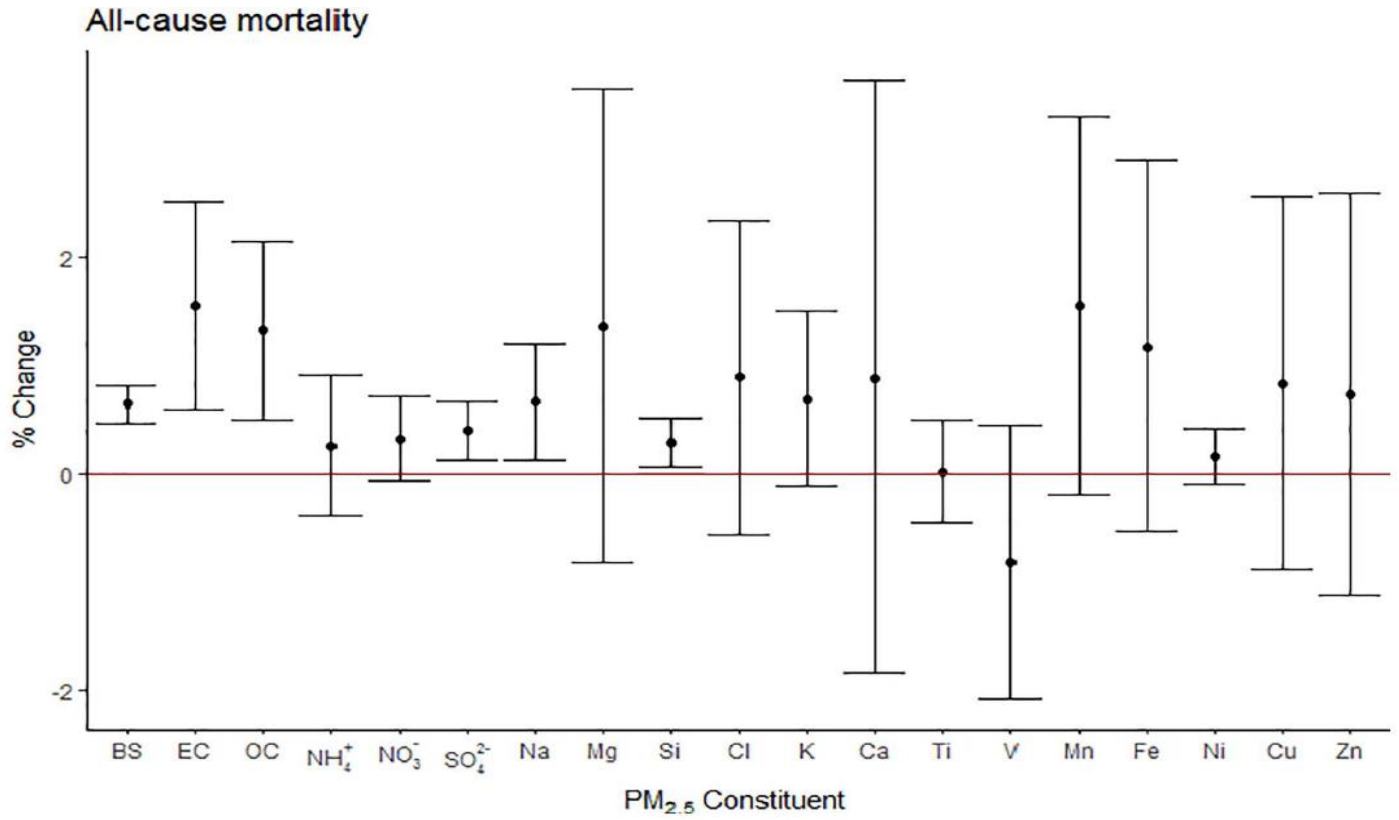
[a]Number of estimates available from all single/multi-city studies. [b]Number of estimates from single/multi-city studies selected for meta-analysis (see Methods for details of estimate selection protocol). [c]Random effects summary estimate expressed as percentage of change in the number of deaths per 1 $\mu g/m^3$ (95% confidence interval). [d]I^2 percentage of between-city variability attributed to heterogeneity.

REVIEW OF STUDIES ON PM CONSTITUENTS AND DAILY MORTALITY
(ACHILLEOS ET AL, 2017)

- Systematic review, (up to July 2015), PRISMA guidelines
- 38 studies, 129 city-specific estimates
- 75 estimates adjusted for total PM2.5 (two-pollutant model)

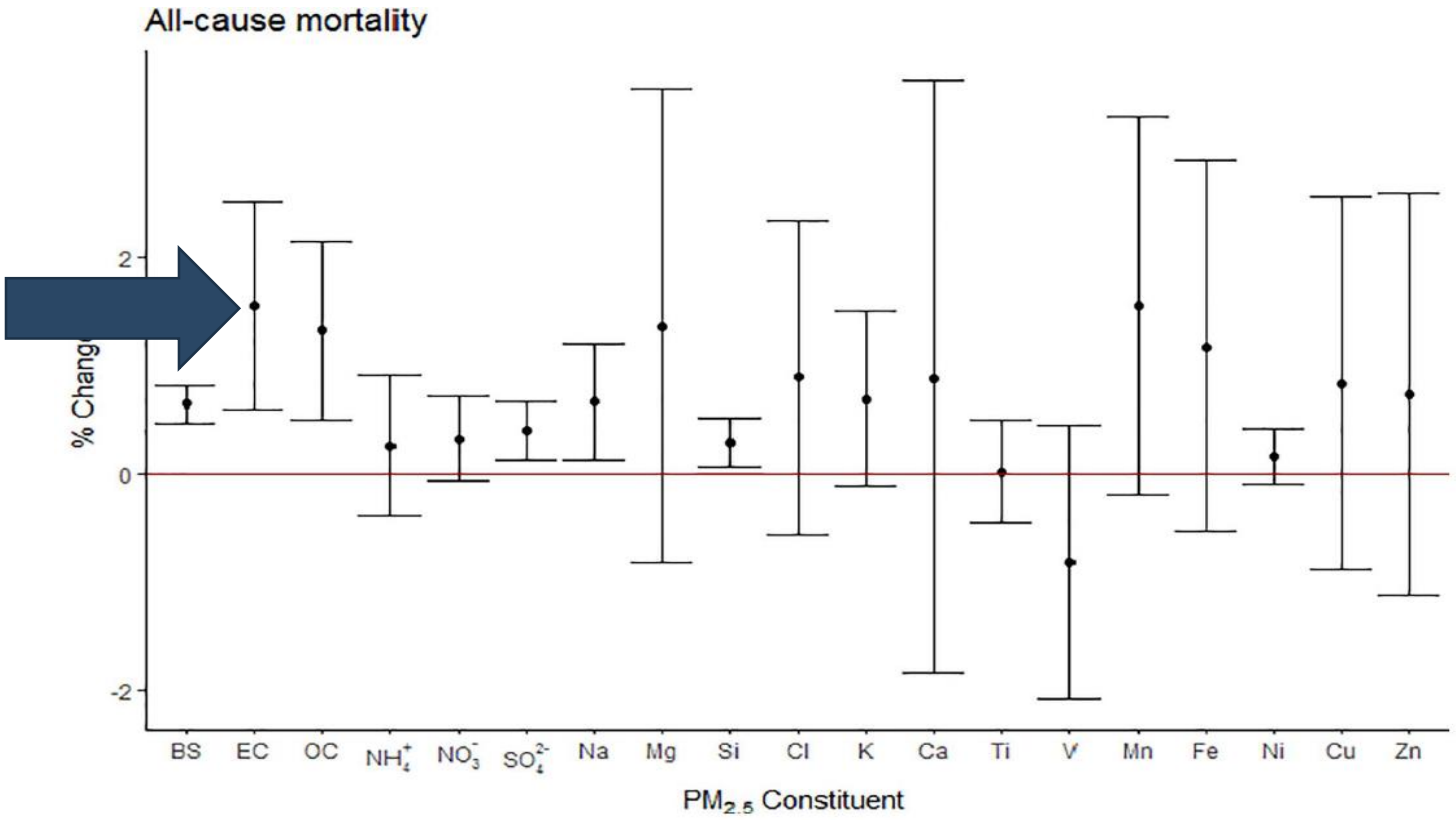
MEAN % CHANGE IN DAILY MORTALITY PER IQR INCREASE

(ACHILLEOS ET AL, 2017)



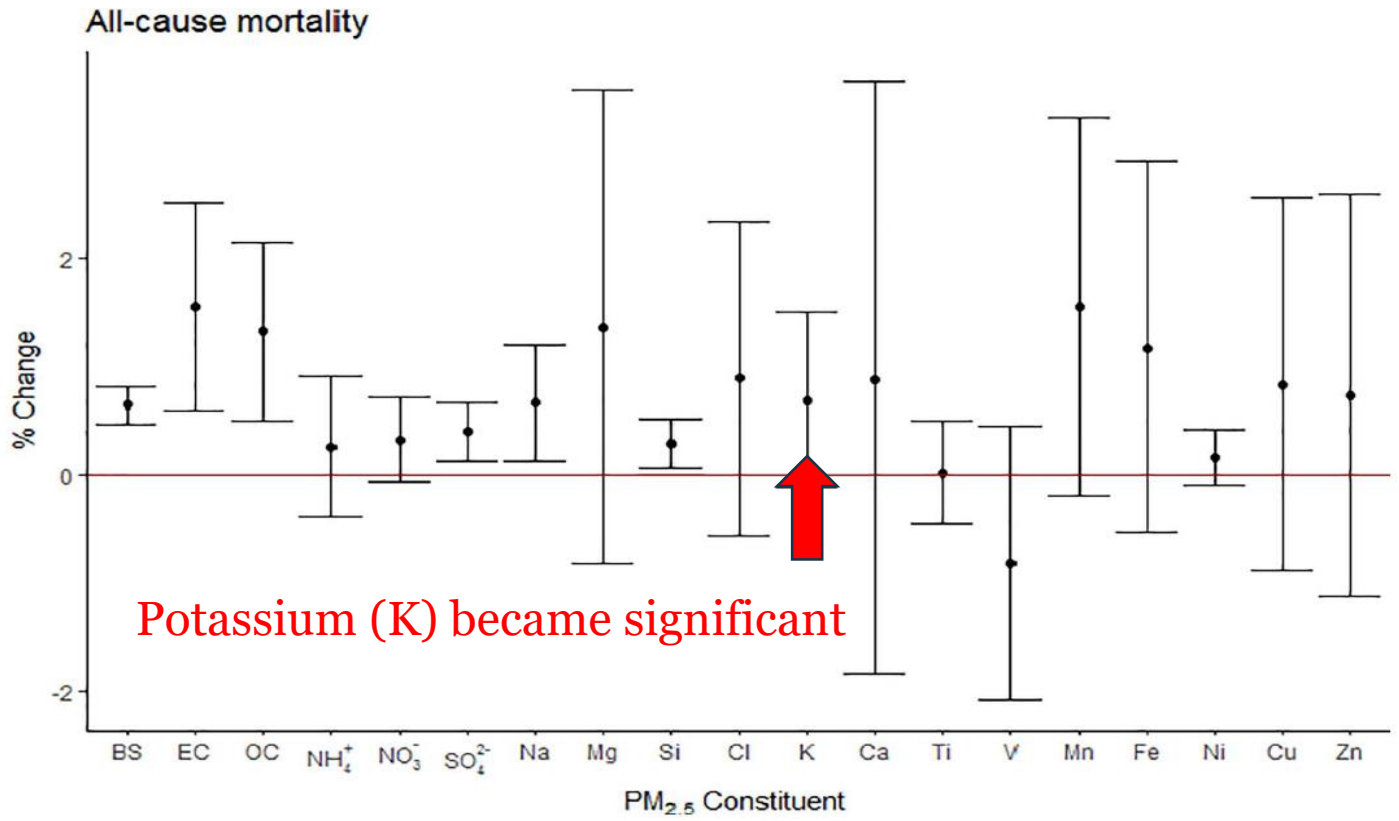
EC REMAINED SIGNIFICANT WHEN INCLUDING TOTAL PM2.5 IN MODEL

(ACHILLEOS ET AL, 2017)



POTASSIUM BECAME SIGNIFICANT WHEN INCLUDING TOTAL PM2.5 IN MODEL

(ACHILLEOS ET AL, 2017)



CONCLUSIONS FROM REVIEW
(ACHILLEOS ET AL, 2017)

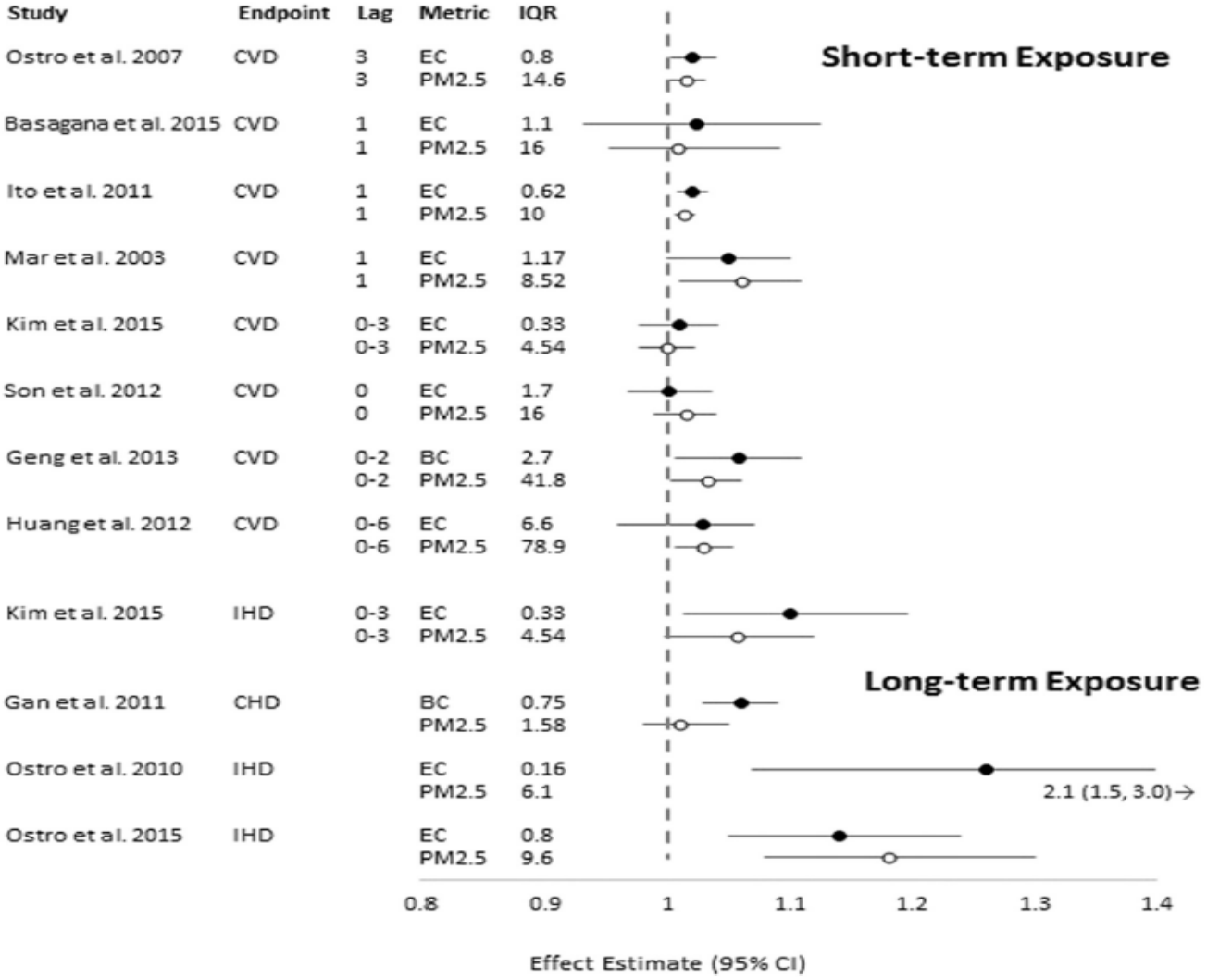
- Similar results in both models (single/with tot PM2.5) are most convincing
- EC and potassium had a stronger short-term effect on mortality than other constituents
- EC reflects mostly traffic exhaust
- K is a wood combustion indicator

REVIEW OF STUDIES ON EC/BC AND PM2.5 AND DIFFERENT CVD EFFECTS
(LUBEN ET AL, 2017)

- Systematic review to evaluate if risks for CVD effects are greater for BC or EC than for PM2.5, compared across cities
- Concludes that the review demonstrates generally similar (”modest”) risk for BC or EC and PM2.5
- However, estimates are presented for an IQR increase, and not per µg/m3

RR PER IQR AND PER UG/M3 WOULD GIVE DIFFERENT CONCLUSIONS

(LUBEN ET AL, 2017)



REVIEW OF LONG-TERM EXPOSURE TO FINE PM AND MORTALITY
(HOEK ET AL, 2013)

- A similar review had been presented by WHO and Janssen et al (2011)
- Spatial scale in studies vary between county and home address but the effect of resolution was not further analyzed
- The pooled estimate for PM2.5 was 6% per 10 µg/m3
- The pooled estimate for EC was 6% per 1 µg/m3
- And a pooled estimate for NO2 was 5% per 10 µg/m3

IS NO2 ITSELF RESPONSIBLE FOR HEALTH EFFECTS? OR IS ONLY AN INDICATOR OF OTHER POLLUTANTS?

- The predominant views in the literature has been that the association between PM and mortality is causal and robust
- NO2 has usually been seen as mainly an indicator correlated to many other traffic-related pollutants
- The discussion of the role of NO2 becomes increasingly more important

ARE THERE LONG-TERM EFFECTS OF NO2 ON MORTALITY?
(FAUSTINI ET AL, 2014)

- Review of studies (23) on long-term exposure to NO2 and mortality, including also estimates for PM
- The pooled risk estimates per 10 μg/m3 were 4,1% for NO2 and 4,5% for PM2.5
- Minimal difference between the single-pollutant and the multi-pollutant results for NO2 were found.
- Authors conclude. “Health impact assessments relying only on PM2.5, and not considering NO2, would be neglecting an important source of the adverse effects of today’s pollution mixture.”

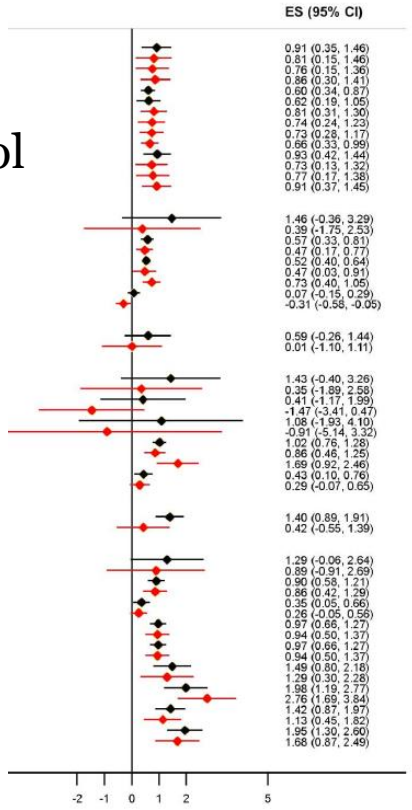
ARE NO2 SHORT-TERM EFFECTS INDEPENDENT OF PM?
(MILLS ET AL, 2016)

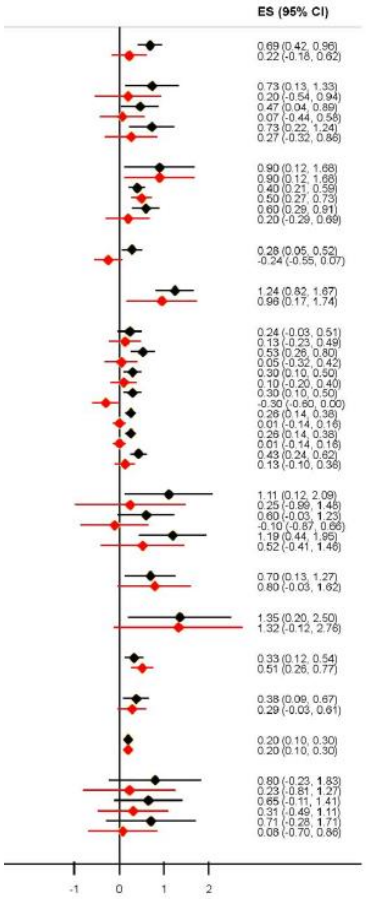
- Systematic review of time-series studies with both single-pollutant and two pollutant estimates for NO2 and PM
- For daily mortality 32 pairs from 26 cities from 5 WHO regions were included
- Meta-analysis gave summary estimates - RR per 10 μg/m3, and NO2 was the more robust in two-pollutant models

NO2 (L) 0.78 → 0.60%
PM2.5 (R) 0.74 → 0.54% (NS)

Black = single pol

Red = both pol





DIFFERENT RESULTS FOR WITHIN AND BETWEEN CITY NO2 EXPOSURE
(CROUSE ET AL, 2015)

- A study of independent and joint effects on mortality of within- and between-city NO2 contrasts in a Canadian population based cohort
- LUR-model was used to estimate annual city-mean and post code mean NO2 concentrations
- Most of the association was determined by within-city contrasts, as opposed to by between-city contrasts in NO2.

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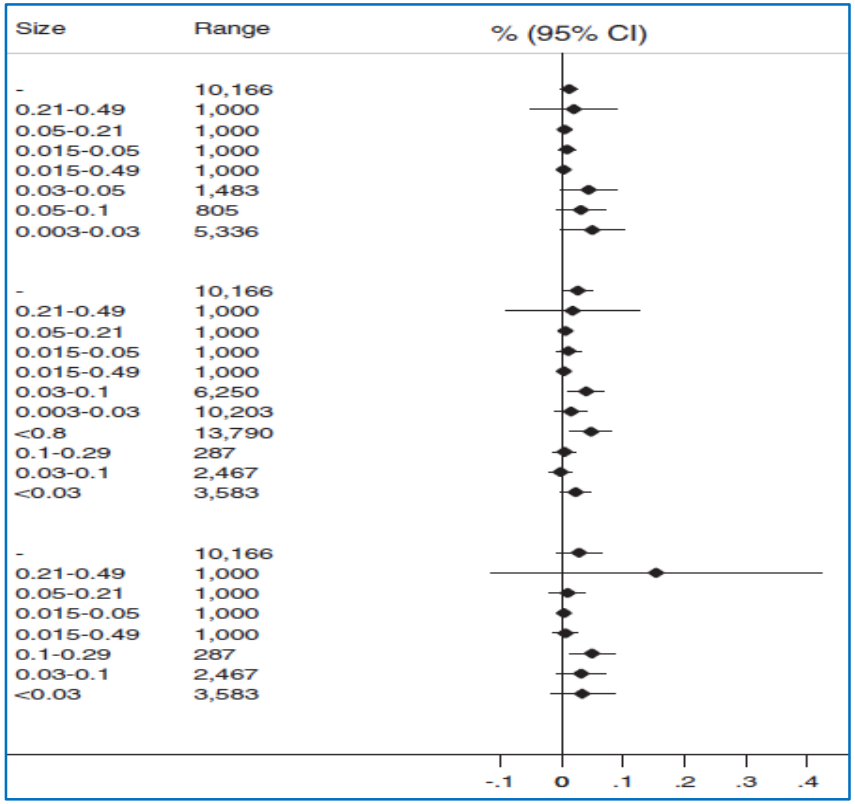
Model exposure metric	*Non-accidental causes*		
	Hazard ratio	*95% Confidence interval*	
NO_2+personal[a] and contextual[b] covariates, with city random effe			
Overall[c]	1.05	1.04	1.07
Between[d]	0.99	0.96	1.03
Within[d]	1.06	1.04	1.07

Per 5 ppb

THE SUPPORT IS WEAK FOR ULTRA-FINES AND DAILY MORTALITY

- A review by Atkinson et al (2015) included 15 studies of UFP and mortality, and found little consistent evidence of an association between (as in other reviews)

- % per IQR increase single-pollutant



EVIDENCE FOR MORE TYPES OF ADVERSE HEALTH EFFECTS

- We repeatedly add new outcomes to the list of adverse effects of air pollution … cardiovascular, metabolic, prenatal…
- During the last few years we have seen a growing number of studies on ageing, cognitive decline and dementia
- Also several studies on air pollution and child behavior and neurodevelopment have recently been published

ELSEVIER

NeuroToxicology

Full length article

Exposure to air pollution as a potential contributor to cognitive function, cognitive decline, brain imaging, and dementia: A systematic review of epidemiologic research

Melinda C. Power[a,b], Sara D. Adar[c], Jeff D. Yanosky[d], Jennifer Weuve, MPH, ScD[e,f,*]

CONCLUSIONS

- The results from a HIA depend much on the choice of RR
- As some sources, such as traffic and RWC, vary on a small scale, it is important in the epidemiological analysis of long-term exposure to assess exposure on a fine spatial scale
- Within-city contrasts in exposure are generally associted with higher RRs than between-city contrasts
- Some exposure models lack a relevant predictor variable for wood smoke
- EC/BC show high RRs per mass concentration, more typical per IQR, results for other PM components/elements vary
- RRs for UFP are often *n s* and sensitive to adjustment for NO2 or PM2.5
- Both short-term and long-term effects of NO2 usually remain after adjustment for PM (if spatial resolution not too bad)
- More types of adverse health effects are added to the long list

Thank you!

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