REVIEW OF EVIDENCE ON HEALTH ASPECTS OF AIR POLLUTION - REVIHAAP

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This presentation:

Summary of the results of the WHO – EC project "Review of evidence on health aspects of air pollution - REVIHAAP"

http://www.euro.who.int/__data/assets/pdf_file/0020/182432/e96762-final.pdf

Plus examples from selected studies.

About REVIHAAP

- WHO project jointly financed by WHO and EC, managed by WHO/ECEH (Marie-Eve Heroux)
- Evidence review in response to 24 key policy questions from the EC
- Timing: 18 months, Sept 2011 – April 2013
- Steering Advisory Committee (8 experts, 2 meetings, multiple TCs)
- Review of evidence and drafting the answers: 29 experts
- External review: 30 experts
- Two expert meetings (Aug 2012 & Jan 2013)
- Full rationales to be published in June 2013
- Followed by a sister project: “Health risks of air pollution in Europe – HRAPIE” - health risk assessment, emerging issues. (June 2012-Aug 2013)

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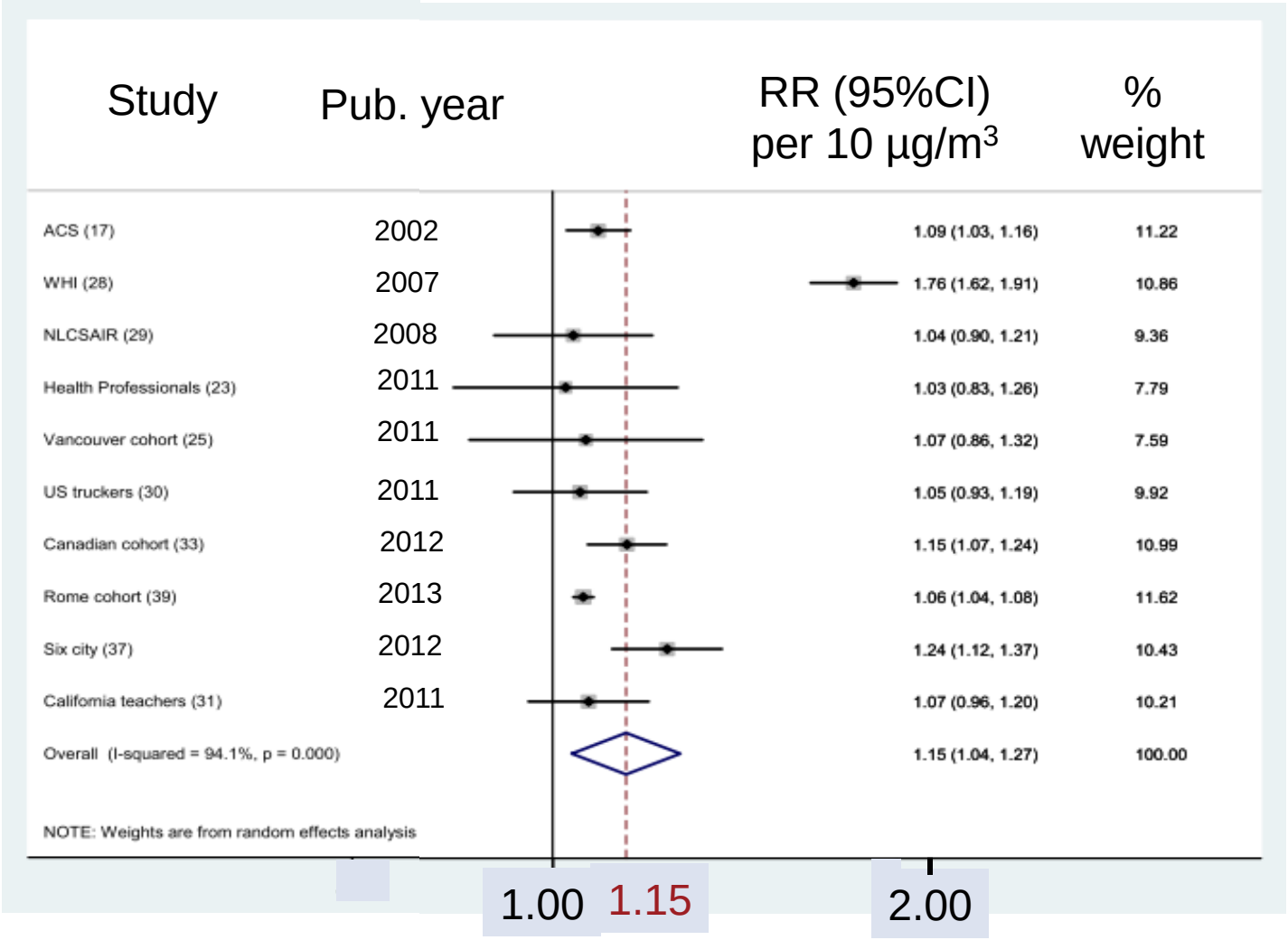
REVIHAAP: selected conclusions on PM (A1)

The scientific conclusions of the 2005 WHO Guidelines about the evidence for a causal link between $PM_{2.5}$ and adverse health outcomes in humans have been confirmed and strengthened and, thus, clearly remain valid.

- New studies on short- and long-term effects;
- Long-term exposures to $PM_{2.5}$ are a cause of cardiovascular mortality and morbidity;
- More insight on physiological effects and plausible biological mechanisms linking short- and long-term $PM_{2.5}$ exposure with mortality and morbidity;
- Studies linking long-term exposure to $PM_{2.5}$ to several new health outcomes (e.g. atherosclerosis, adverse birth outcomes, childhood respiratory disease).

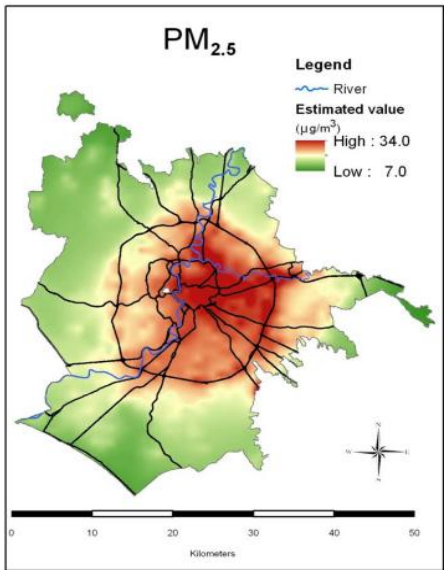
Meta-analysis of the association between long-term exposure to $PM_{2.5}$ and cardiovascular mortality



Hoek et al, EnvHealth 2013

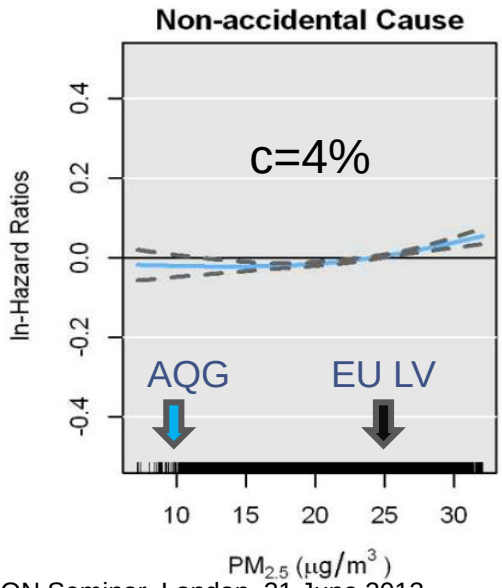
Mortality and long-term exposure to PM2.5

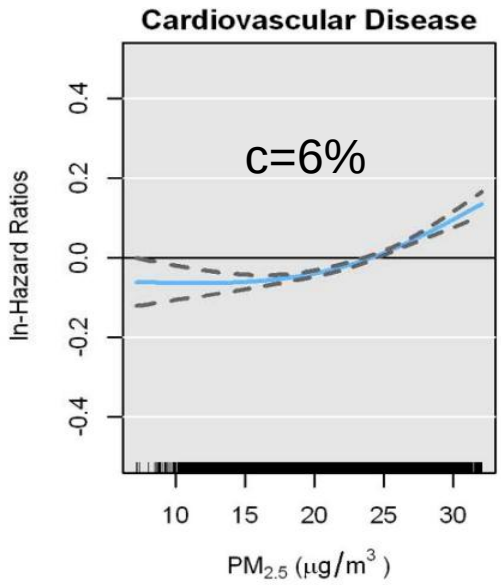
Results of a cohort study in Rome (1.3 million adults followed from 2001 to 2010)

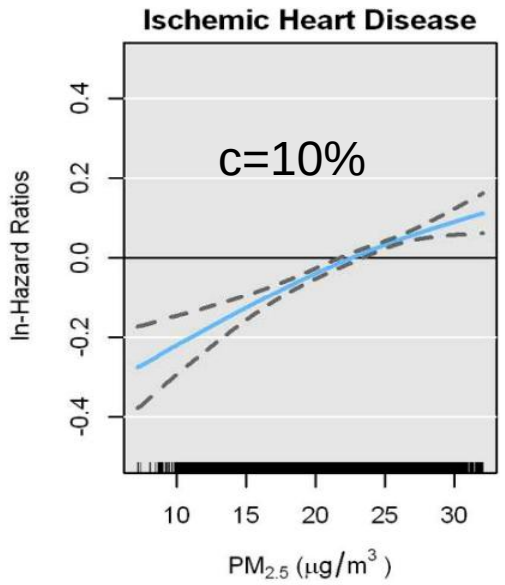


PM2.5: 3-dimensional Eulerian model (1x1 km)

c= % increase in risk per 10 µg/m^3

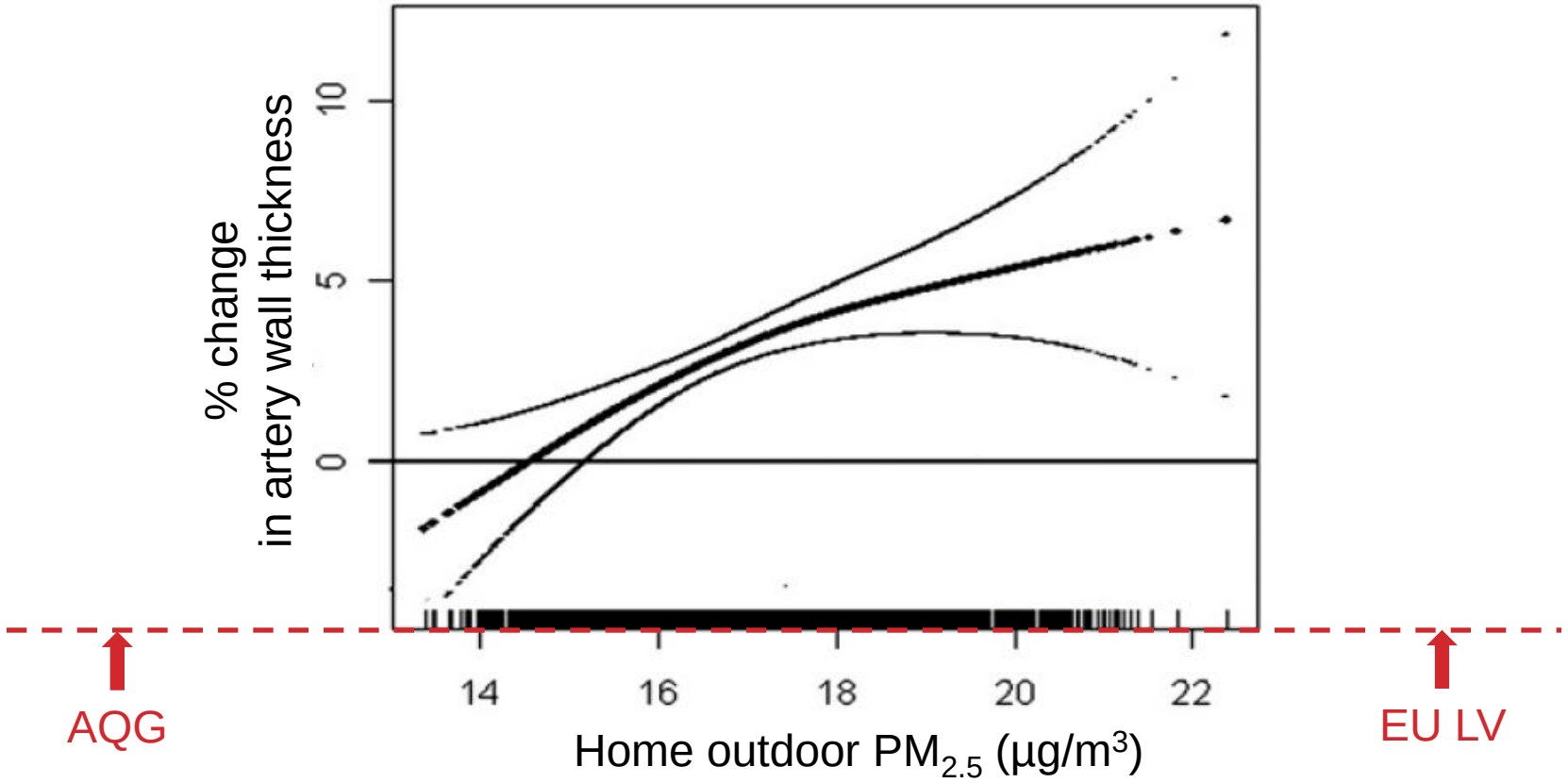






Cesaroni et al. EHP 2013

Carotid artery wall thickness (=risk of atherosclerosis) and long-term $PM_{2.5}$ exposure



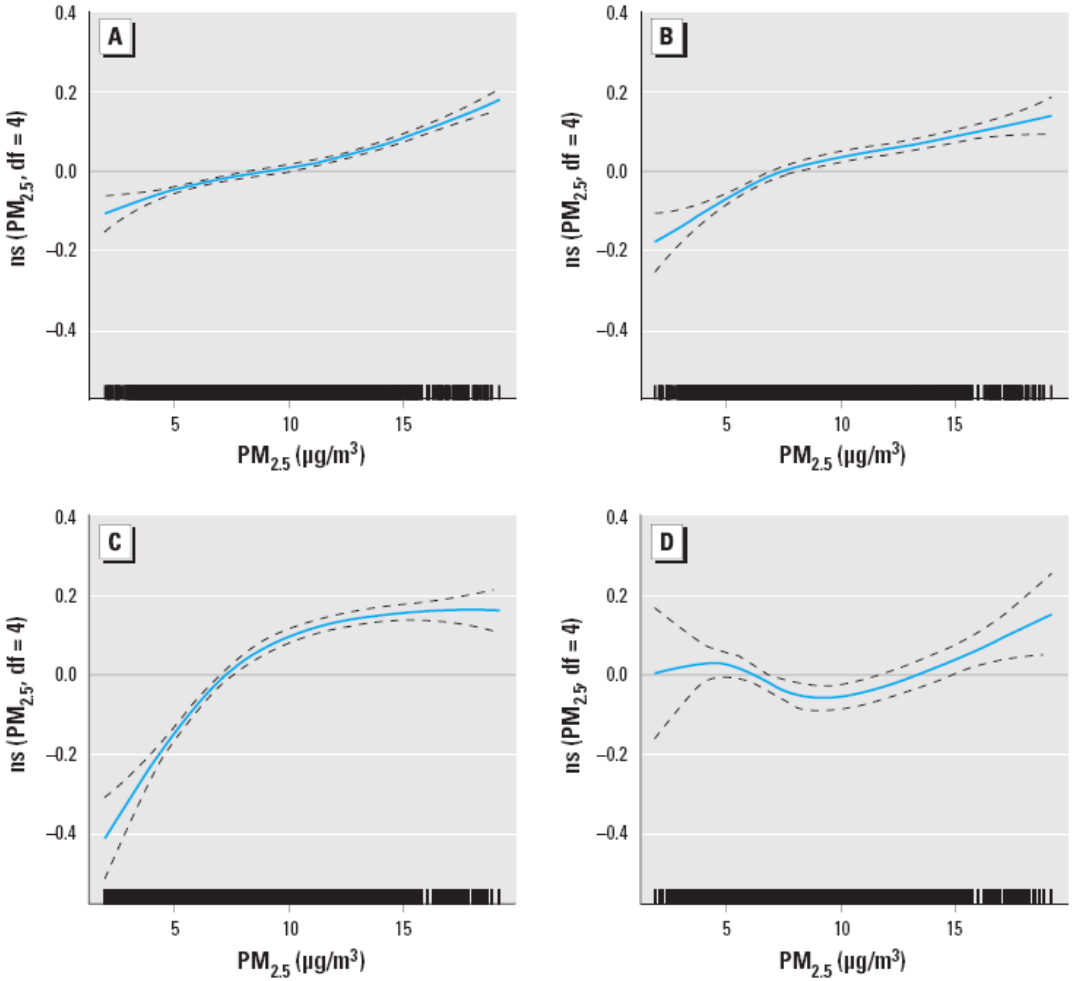
Heinz Nixdorf RECALL study, Ruhr region, Germany

Bauer et al, JACC 2010

Mortality and long-term PM2.5 exposure

Results of a Canadian cohort study (2.1 million adults, 1991-2001)

All non-accidental



Cardiovascular

Ischemic heart disease

Cerebrovascular

PM2.5 estimated from satellite observations + monitoring

Crouse et al, EHP 2012

REVIHAAP: selected conclusions on PM, cont.

- Both short term (such as 24-hour average) and long term (annual means) exposure to $PM_{2.5}$ affect health. (A3)

- Maintaining independent short-term and long-term limit values for ambient PM_{10} in addition to $PM_{2.5}$ to protect against the health effects of both fine and coarse particles is well supported. (A4)

- In the absence of a threshold and in light of linear or supra-linear risk functions, public health benefits will result from any reduction of $PM_{2.5}$ concentrations whether or not the current levels are above or below the (EU) limit values. (A5)

REVIHAAP: selected conclusions on PM composition (A2)

- Black carbon, secondary organic aerosols, and secondary inorganic aerosols may provide valuable metrics for the effects of mixtures of pollutants from a variety of sources. (A2)

- Short-term exposures to coarse particles (including crustal material) are associated with adverse respiratory and cardiovascular health effects, including premature mortality. (A2)
- …

REVIHAAP: selected conclusions on ultrafine PM (A2)

- There is increasing, though as yet limited, epidemiological evidence on the association between short-term exposures to ultrafine (<0.1 µm) particles and cardiorespiratory health, as well as the health of the central nervous system.

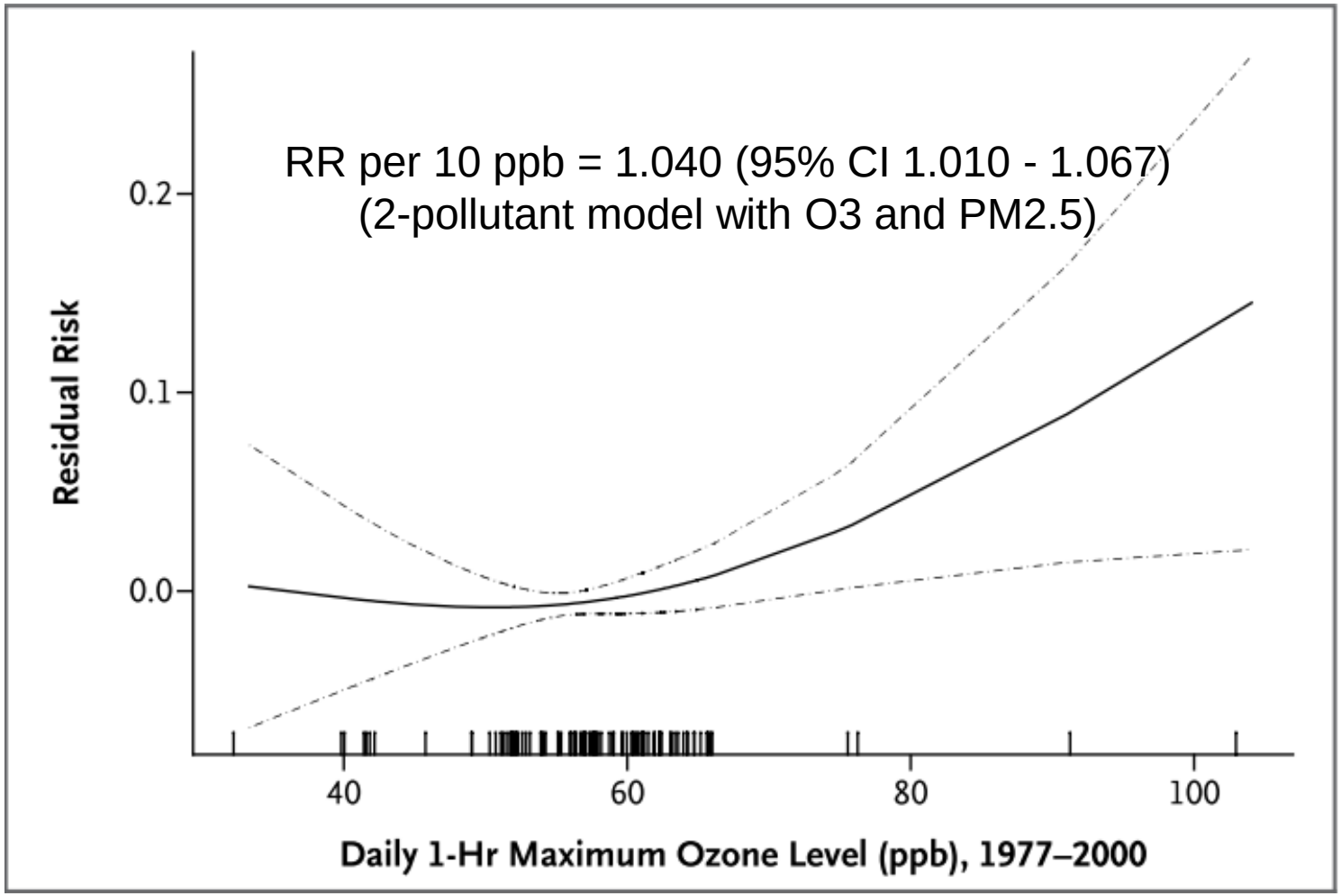
- Clinical and toxicological studies have shown that ultrafine particles (in part) act through mechanisms not shared with larger particles that dominate mass-based metrics, such as $PM_{2.5}$ or PM_{10}.

REVIHAAP: selected conclusions on ozone (B1)

- **New evidence for an effect of <u>long-term</u> exposure to ozone on:**
  - respiratory (and cardiorespiratory) mortality (ACS study);
  - mortality among persons with potentially predisposing conditions (COPD, diabetes, congestive heart failure, and myocardial infarction);
  - asthma incidence, asthma severity, hospital care for asthma and lung function growth.

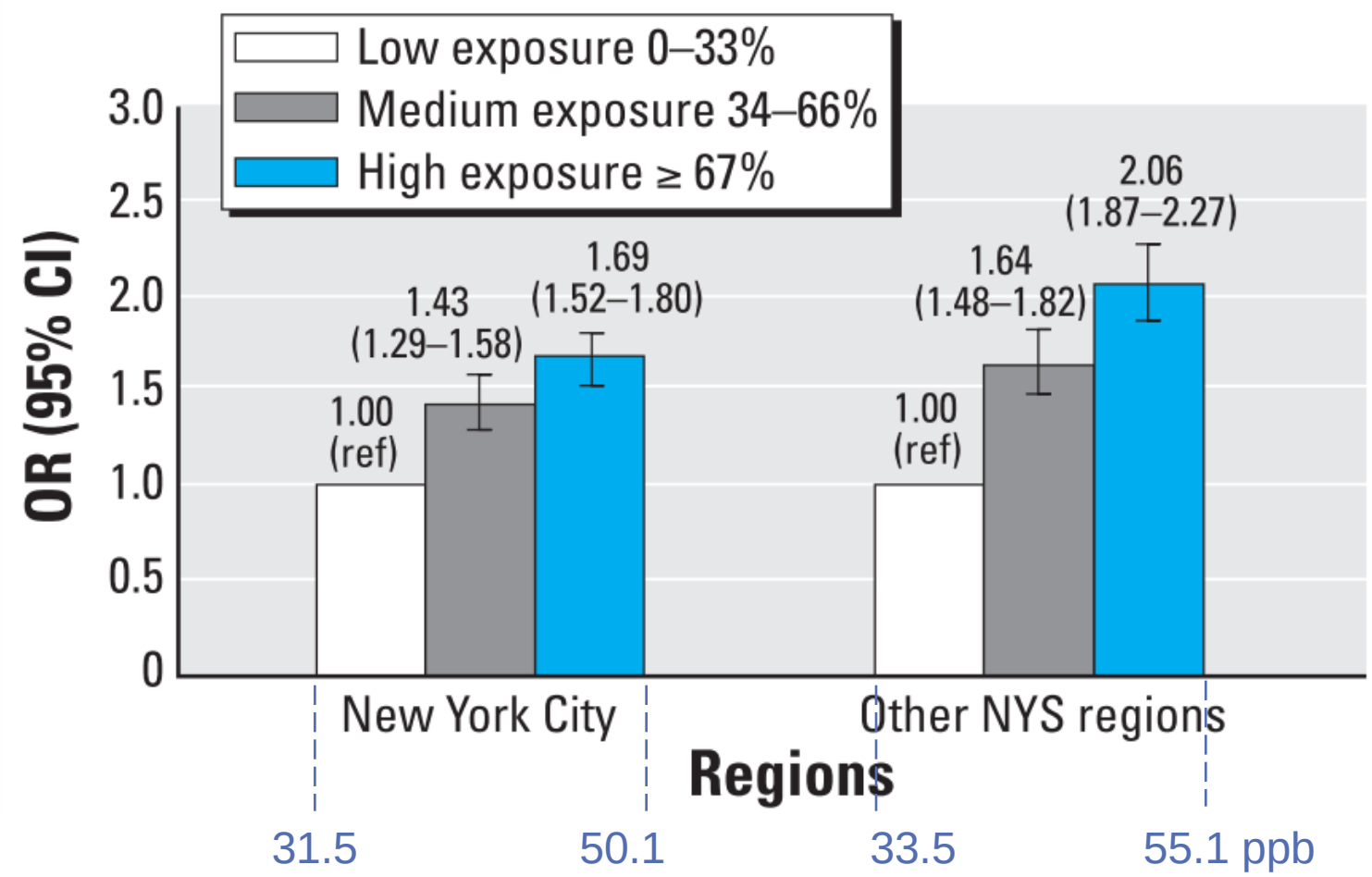
Long term O3 exposure and risk of death due to respiratory causes

ACS cohort of 448 thousand adults followed for 18 years



Jerrett et al, NEJM 2009

Risk of asthma hospital admissions in children and mean O_3 in 1995-1999



O_3 exposure: average of daily max 8-h means in O_3 season (Apr-Oct)

Lin et al, EHP 2008

REVIHAAP: selected conclusions on ozone, cont.

- **Adverse effects of exposure to <u>daily</u> ozone concentrations (maximum daily 1-hr or 8-hr mean) on:**
  - all-cause, cardiovascular and respiratory mortality;
  - respiratory and cardiovascular hospital admissions.

- **The evidence for a threshold for short term exposure is not consistent, but where a threshold is observed, it is likely to lie below 45 ppb (90 µg/m^3) (max 1-hr). (B2)**

Associations between short-term exposure to ozone and mortality and hospital admissions in European cities in the APHENA study

Outcome	Per cent increase in deaths/admissions (95% CI) per 10 μg/m³ increment in daily maximum 1-hour ozone concentrations	
	Single pollutant	Adjusted for PM_{10}
All-cause mortality [a]	0.18 (0.07–0.30)	0.21 (0.10–0.31)
Cardiovascular mortality: 75 years and older [a]	0.22 (0.00–0.45)	0.21 (-0.01–0.43)
Cardiovascular mortality: younger than 75 years [a]	0.35 (0.12–0.58)	0.36 (0.10–0.62)
Respiratory mortality [b]	0.19 (-0.06–0.45)	0.21 (-0.08–0.50)
Cardiac admissions: older than 65 years [a]	-0.10 (-0.46–0.27)	0.64 (0.36–0.91)
Respiratory admissions: older than 65 years [b]	0.19 (-0.28–0.67)	0.32 (0.05–0.60)

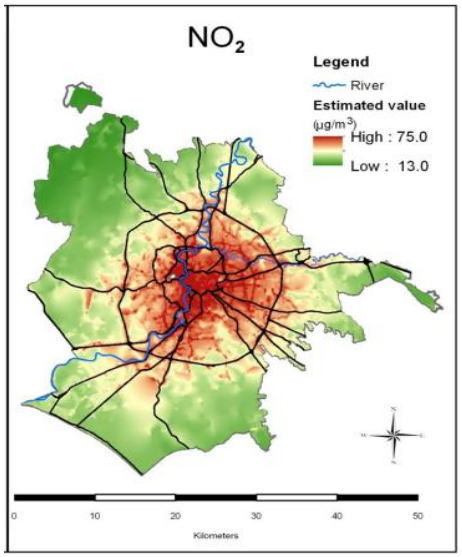
[a] lag 0-1 results; [b] lag 1 results

Katsouyanni et al 2009

REVIHAAP: selected conclusions on NO_2 (C2-3,D1)

- New studies document associations between day-to-day variations in NO_2 and variations in mortality, hospital admissions, and respiratory symptoms;
- New studies showing associations between long-term exposure to NO_2 and mortality and morbidity;
- Both short- and long-term studies have found these adverse associations at concentrations that were at or below the current EU LV (= WHO AQG);
- The associations between NO_2 and short-term health effects in many studies remain after adjustment for other pollutants (including PM_{10}, $PM_{2.5}$, black smoke).
- **… it is reasonable to infer that NO_2 has some direct effects**.
- No evidence to suggest changing the averaging time for the short-term EU limit value (1-hour) (D1)

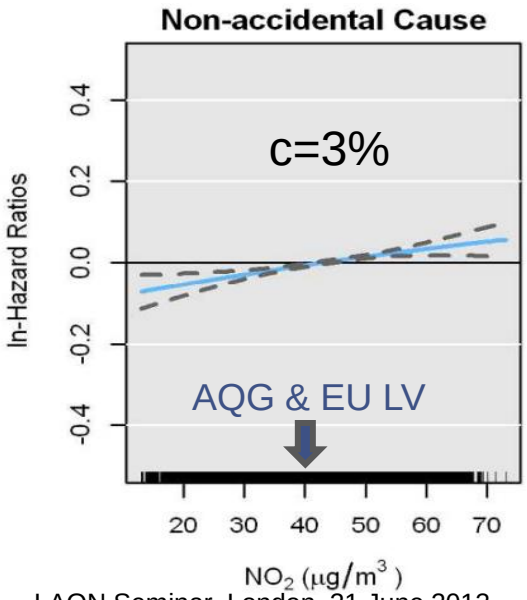
Mortality and long-term exposure to NO_2

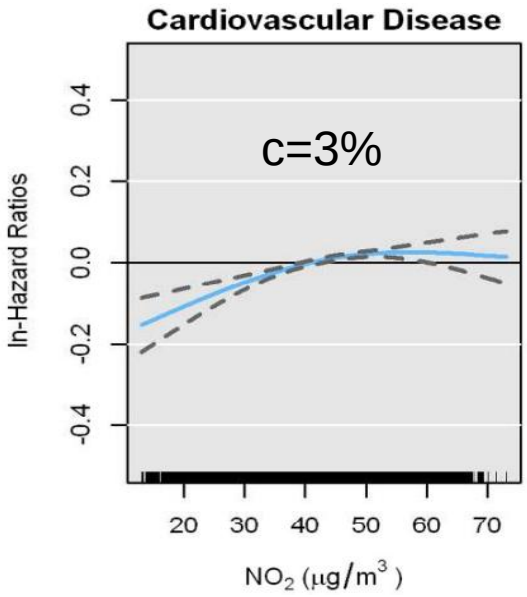


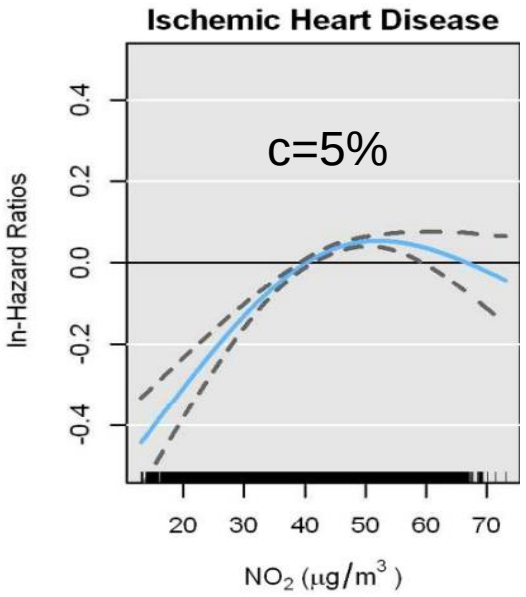
Results of a cohort study in Rome (1.3 million adults followed from 2001 to 2010)

NO_2 estimates: Ogawa samplers in 78 sites, 1-week in Feb, May, Oct 2007 + LUR model
Quintiles of NO_2: 37, 43, 46, 50 µg/m^3

c= % increase in risk per 10 µg/m^3







Cesaroni et al. EHP 2013

ESTIMATES OF RISK OF MORTALITY DUE TO LONG TERM NO_2 EXPOSURE:
SINGLE- AND MULTI- POLLUTANT STUDIES

	Parameter	Total or natural mortality		
		estimate	95%CI	
Gehring, 2006				
NO2 single (per 16 µg/m3)	Rate ratio	1.19	1.02	1.39
with traffic indicator		no changes (data not shown)		
Jerrett, 2009				
NO_2 single (per 4 pbb)	Rate ratio	1.17	1.00	1.36
with traffic indicator		1.13	0.97	1.32
Hart, 2011				
NO2 single (per 8 pbb)	% increase	8.20	4.50	12.10
with PM_{10} and SO_2		7.40	2.40	12.50
Cao, 2011				
NOx single (per 10 µg/m3)	% increase	1.50	0.40	2.50
with TSP		1.40	0.30	2.50
Cesaroni 2013				
NO_2 single (per 10 µg/m3)	Rate ratio	1.03	1.02	1.04
with $PM_{2.5}$		1.02	1.01	1.03
with traffic indicator		no changes (data not shown)		

Source: HRAPIE (F. Forastiere)

REVIHAAP: selected conclusions on health risks of proximity to roads (C1):

- Elevated health risks associated with living in close proximity to roads is unlikely to be explained by $PM_{2.5}$ mass;
- Current evidence does not allow discernment of the pollutants or pollutant combinations that are related to different health outcomes although association with tail pipe primary PM is increasingly identified;
- Toxicological research indicates that non-exhaust pollutants could be responsible for some of the observed health effects.

REVIHAAP: contribution of ambient air pollution to total exposure to regulated air pollutants (C10)

In the absence of tobacco smoke:

- Indoor, occupational and commuting exposures vary more than exposure to ambient air pollution;
- Commuting can increase exposures to PM, NO_2, CO and benzene, and is a major contributor to the exposure to UFP, BC and metals (Fe, Ni and Cu in the underground);
- Ambient air dominates population exposures to NO_2 (no gas appliances), $PM_{2.5}$, BC, O_3, CO and SO_2 (also BaP, As, Cd, Ni and Pb);
- …

REVIHAAP: contribution of ambient air pollution to total exposure to regulated air pollutants (C10) (cont.)

In the absence of tobacco smoke:

- Ambient air, indoor sources and commuting are all important for population exposures to NO_2 (where gas appliances are frequent), benzene and naphthalene;
- The high end of the individual exposures to $PM_{10-2.5}$ and naphthalene originate from indoor sources and commuting;
- Solid fuel fired indoor fireplaces and stoves, where used in suboptimal conditions, dominate the high end of the exposures to $PM_{2.5}$, BC, UFP, CO, benzene and BaP of the affected individuals.

REVIHAAP: Critical data gaps (A7/C9) – selected conclusions on health effects studies

- More epidemiological studies to update E-R functions based on meta-analyses for integrated risk assessment;
- The coordinated application of atmospheric science, epidemiological, controlled human exposure and toxicological studies to advance understanding of the:
  - sources responsible for the most harmful emissions,
  - physical–chemical composition of the pollution,
  - biological mechanisms that lead to adverse effects on health;

…

REVIHAAP: Critical data gaps (A7/C9) – selected conclusions on health effects studies (Cont.)

- Air pollution should be considered to be one complex mix, and conditions under which this mix has the largest effect on human health need to be identified;
- Advances in atmospheric modelling, in conjunction with validation studies that use targeted monitoring campaigns, will provide a more efficient way forward in research on health effects, rather than relying on increasing the number of components measured by routine monitoring networks.

REVIHAAP Conclusions

Considerable amount of new scientific information on health effects of PM, O_3 and NO_2 observed at levels commonly present in Europe, has been published in the recent years. It:

- supports the scientific conclusions of the WHO Air Quality Guidelines updated in 2005;
- indicates that the effects can occur at air pollution concentrations lower than those serving to establish the 2005 Guidelines;
- povides scientific arguments for the decisive actions to improve air quality and reduce the burden of disease associated with air pollution in Europe.

Thank you