I danni d’organo e gli effetti noti e meno noti

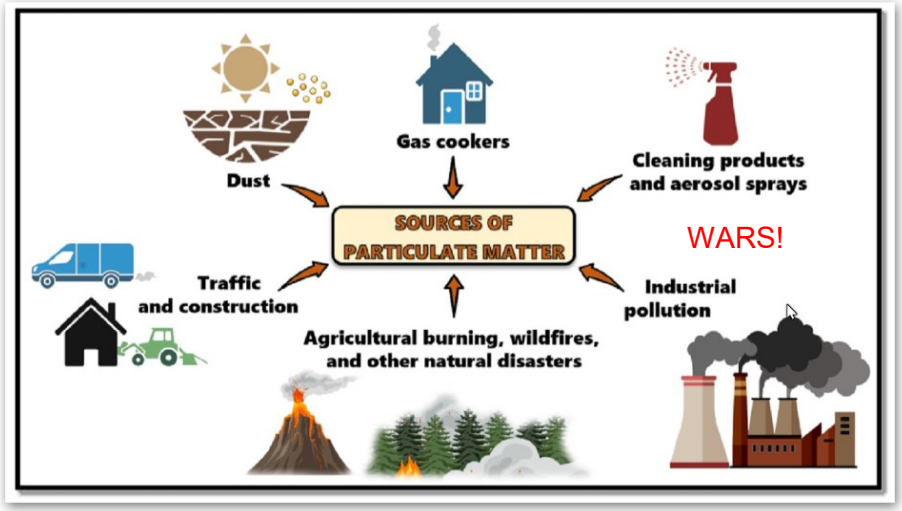
CUORE E VASI

Roberto Pedrinelli



INQUINAMENTO ATMOSFERICO (AIR POLLUTION)

- Una miscela di materia solida, liquida gassosa sospesa nell'aria originata da
  - FONTI NATURALI: vulcani, tempeste di polvere o incendi;
  - ATTIVITÀ UMANE (particolato antropogenico) derivate dalla combustione di combustibili fossili, traffico, processi industriali, guerre e il fumo di tabacco (come inquinante in ambienti chiusi).
- L'inquinamento atmosferico rappresenta un importante fattore altamento patogeno per la salute umana





INQUINAMENTO ATMOSFERICO E MORTALITA' TOTALE



Public Health

MORTALITY IN THE LONDON FOG INCIDENT, 1952

W. P. D. LOGAN
M.D. Glasg., Ph.D. Lond.
CHIEF MEDICAL STATISTICIAN, GENERAL REGISTER OFFICE

TABLE I—DEATHS REGISTERED IN GREATER LONDON FROM WEEK ENDED NOV. 15, 1952, TO JAN. 10, 1953, COMPARED WITH THE ANNUAL AVERAGE FOR CORRESPONDING WEEKS OF THE FIVE PREVIOUS YEARS, 1947–52

Year	Deaths in week ended								
	Nov. 15	Nov. 22	Nov. 29	Dec. 6	Dec. 13	Dec. 20	Dec. 27	Jan. 3	Jan. 10
1952	1565	1699	1902	2062	4703	3138	2234	2977	2634
1947–52 (av.)	1747	1708	1809	1805	1852	1914	1923	2303	2213
	0,89	0,99	1,05	1,14	**2.54**	**1,64**	1,16	1,29	1,19



Lancet. 1953 Feb 14;1(6755):336-8.

Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study

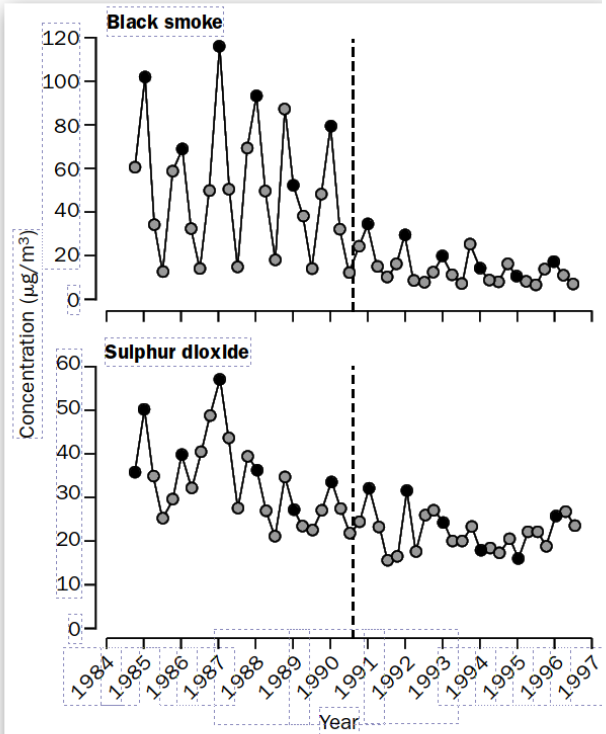
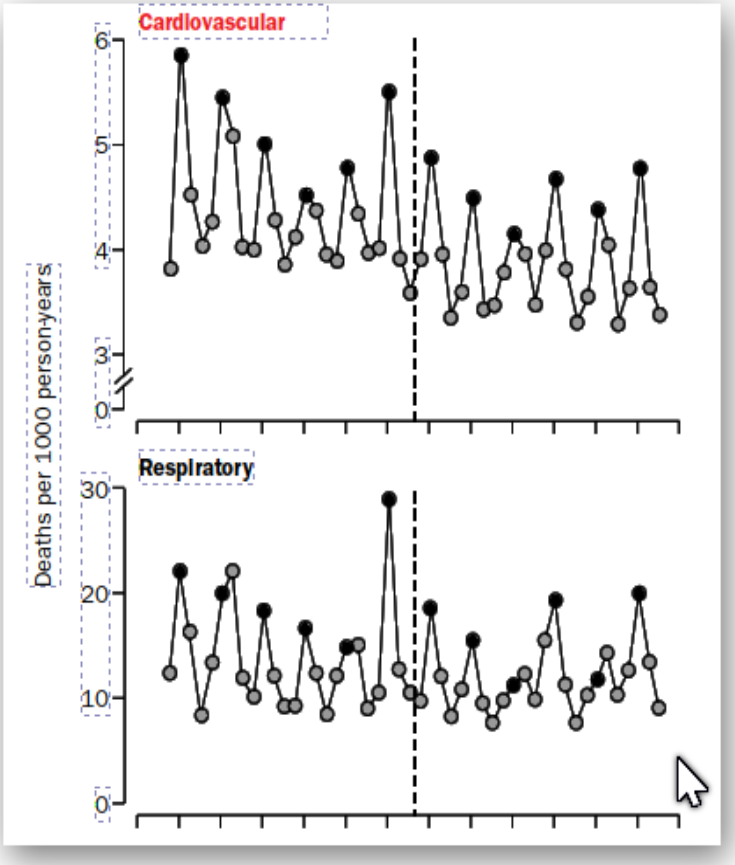


Figure 1: **Seasonal mean black smoke (upper) and sulphur dioxide (lower) concentrations, September 1984–96**
Vertical line shows date sale of coal was banned in Dublin County Borough. Black circles represent winter data.





- Roberto Pedrinelli : L'impatto sul sistema cardiovascolare

Lancet 2002; 360:1210–14

PARTICULATE MATTERs (PM9

PM

- Tra le componenti della miscela solida, liquida gassosa sospesa nell'aria le PM rappresentano una fonte patogena di primaria rilevanza
- Particelle carboniose derivate dalla combustione incompleta associate a sostanze chimiche organiche adsorbite e metalli reattivi
- In base alle dimensioni, il PM viene suddiviso in:
  - (a) grossolano (PM10, diametro <10 µm),
  - (B) FINE (PM2.5, DIAMETRO <2.5 µM),

PM 2.5

- Penetrano nelle piccole vie aeree e negli alveoli e da qui nella circolazione sistemica
- Al di sopra di certe concentrazioni aumentano morbilità e mortalità CV oltre che di patologie cardiometaboliche che a loro volta ne amplificano il rischio



Table 1. Recommended levels and interim targets (in μg/m³) for an annual averaging time

Pollutant	Interim target				AQG (2021)	AQG (2005)
	1	2	3	4		
$PM_{2.5}$	35	25	15	*10*	**5**	10
PM_{10}	*70*	50	30	*20*	**15**	20
NO_2	*40*	30	20		**25**	40

AQG: WHO air quality guidelines.



PM2.5 E PATOLOGIE CARDIOVASCOLARI



Epidemiological time series studies of $PM_{2.5}$ and daily mortality and hospital admissions: a systematic review and meta-analysis

R W Atkinson,[1] S Kang,[1] H R Anderson,[1,2] I C Mills,[3] H A Walton[2,]

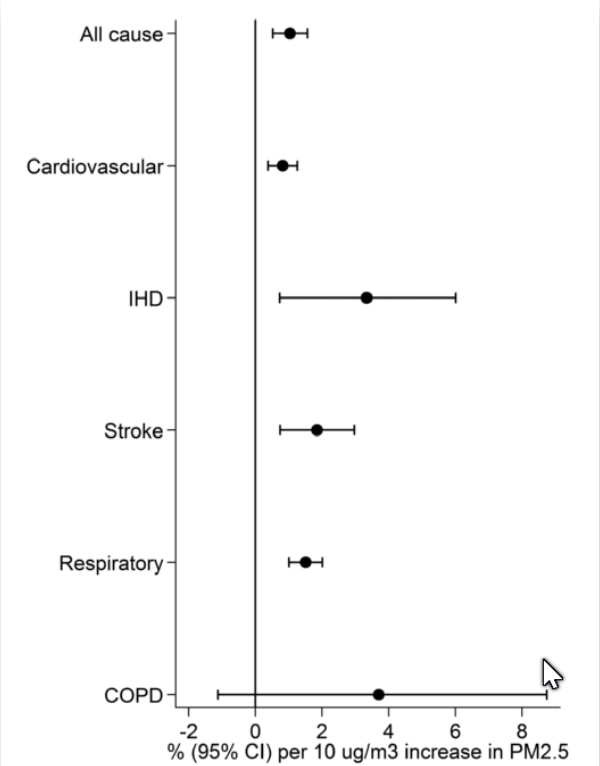


Figure 1 Summary estimates (95% confidence intervals) for all-cause and cause-specific mortality.

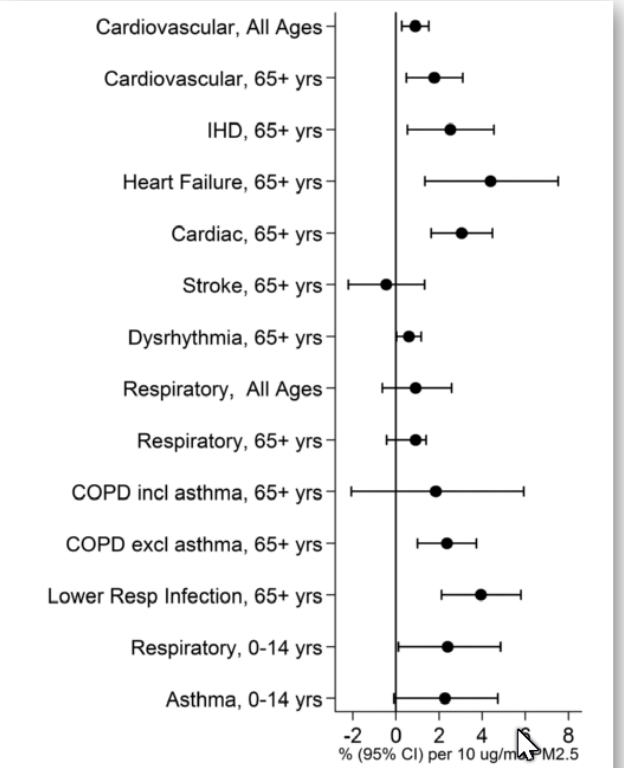


Figure 2 Summary estimates (95% confidence intervals) for cardiovascular and respiratory hospital admissions.



Roberto Pedrinelli : L'impatto sul sistema cardiovascolare

Thorax 2014;69:660–665.

Long-Term $PM_{2.5}$ Exposure and Risks of Ischemic Heart Disease and Stroke Events Review and Meta-Analysis

Stacey E. Alexeeff, PhD; Noelle S. Liao, MPH; Xi Liu, MPH; Stephen K. Van Den Eeden, PhD; Stephen Sidney, MD, MPH

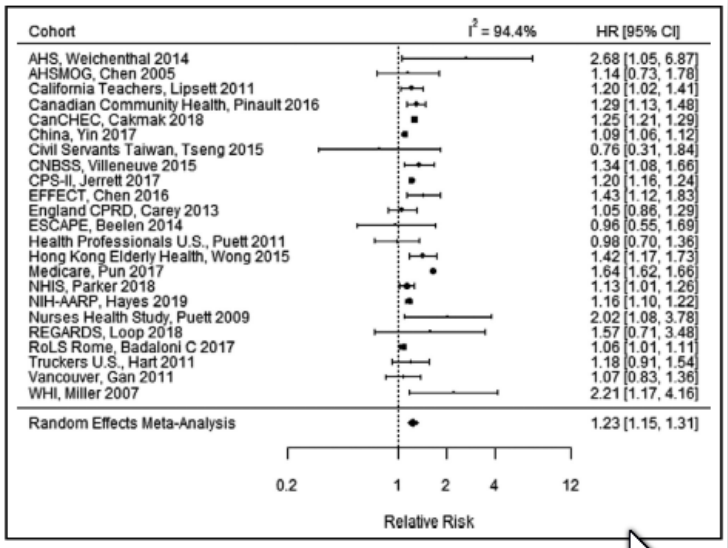


Figure 1. Meta-analysis of the relative risk of ischemic heart disease mortality per 10-μg/m³ increase in long-term fine particulate matter <2.5 μm in diameter exposure, combining the effects of 23 studies.[11]

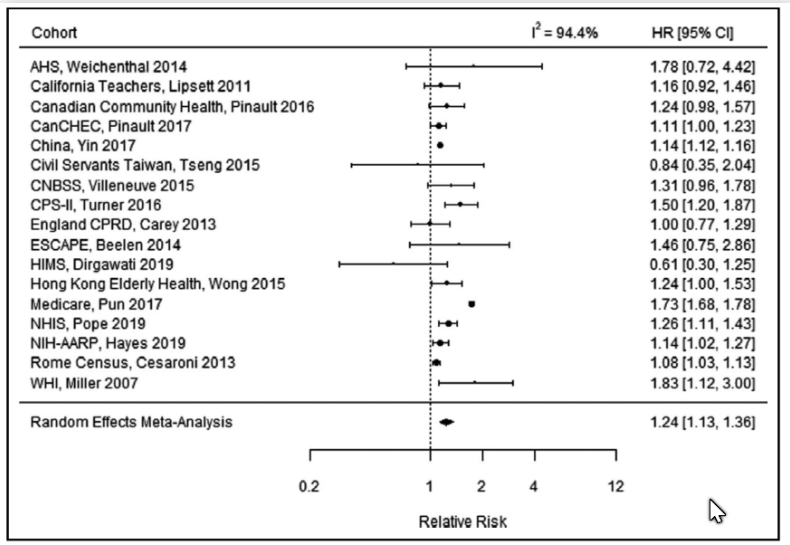


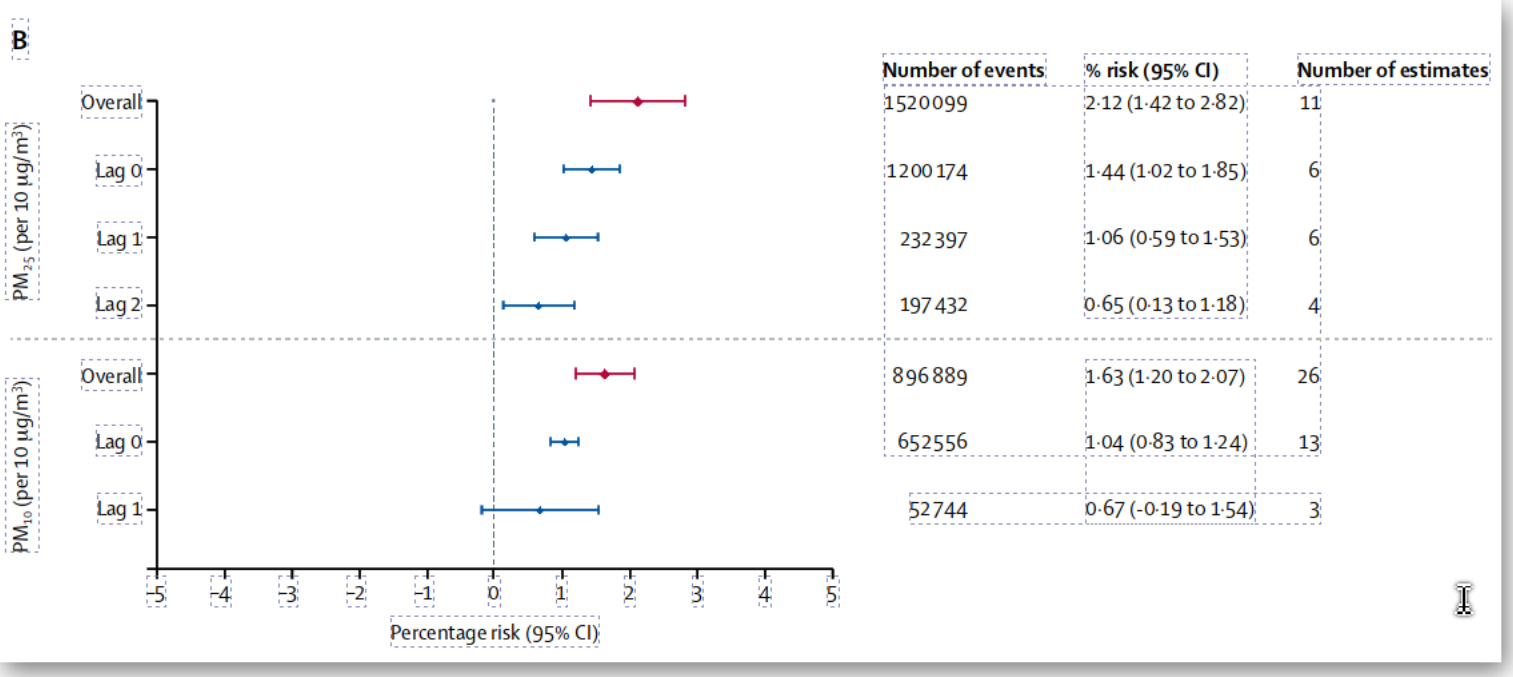
Figure 3. Meta-analysis of the relative risk of cerebrovascular mortality per 10-μg/m³ increase in long-term fine particulate matter <2.5 μm in diameter exposure, combining the effects of 17 studies.[13]



J Am Heart Assoc. 2021 Jan 5;10(1):e016890

Global association of air pollution and heart failure: a systematic review and meta-analysis

Anoop S V Shah, Jeremy P Langrish, Harish Nair, David A McAllister, Amanda L Hunter, Ken Donaldson, David E Newby, Nicholas L Mills





NB: Stime basate su eventi acuti associati all'esposizione a breve termine di PM2.5

Lancet 2013; 382: 1039–48

Global association of air pollution and heart failure: a systematic review and meta-analysis

Anoop S V Shah, Jeremy P Langrish, Harish Nair, David A McAllister, Amanda L Hunter, Ken Donaldson, David E Newby, Nicholas L Mills

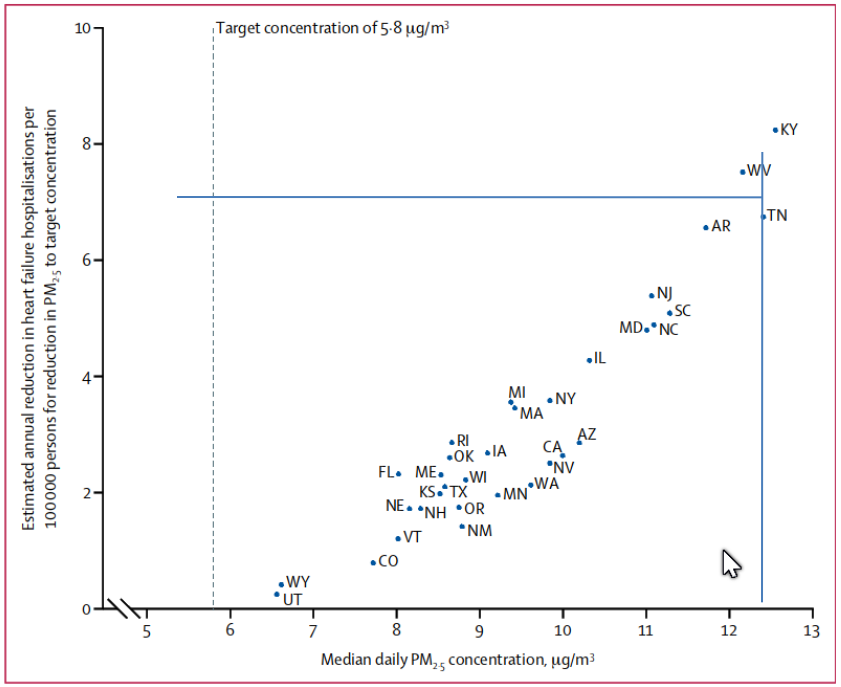


Figure 3: Median daily $PM_{2·5}$ concentrations and estimated impact of a reduction in $PM_{2·5}$ to a target concentration on heart failure hospitalisation per US state

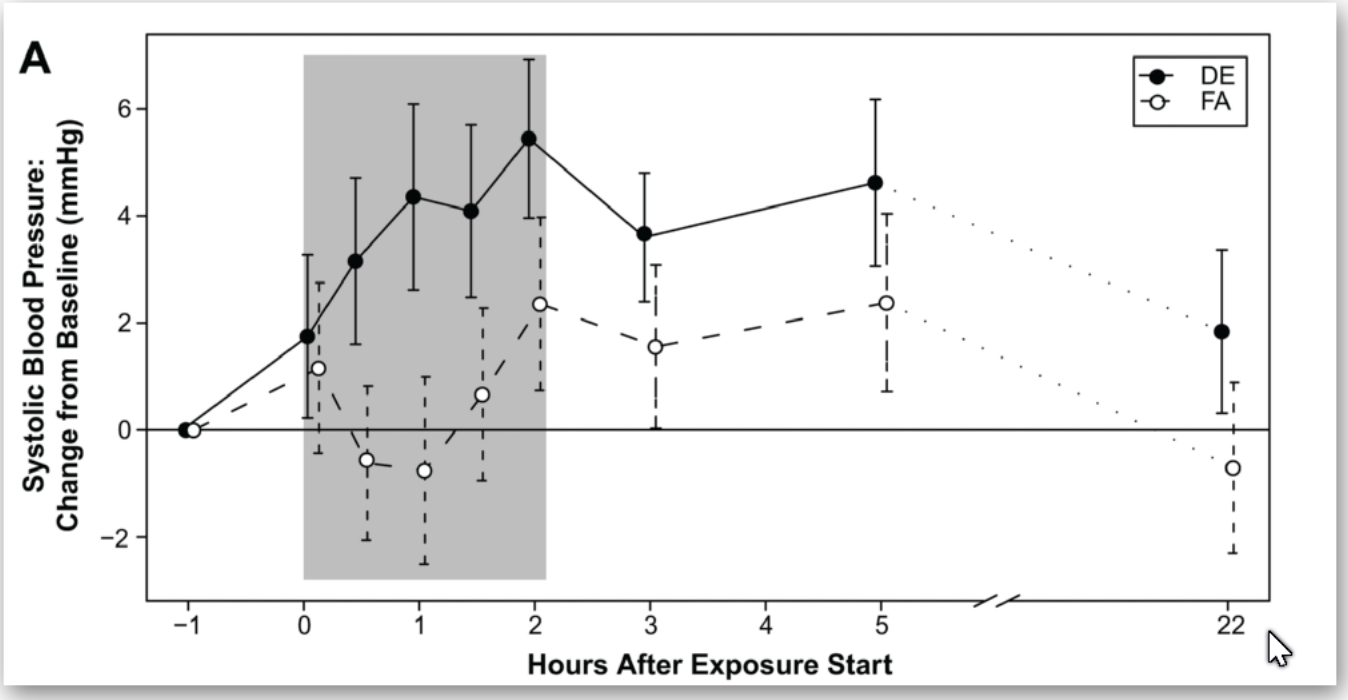


Lancet 2013; 382: 1039–48

PM2.5 E MODULAZIONE PRESSORIA SISTEMICA



BLOOD PRESSURE RESPONSE TO CONTROLLED DIESEL EXHAUST EXPOSURE IN HUMAN SUBJECTS



DE Diesel Exhaust
FE Filtered Air

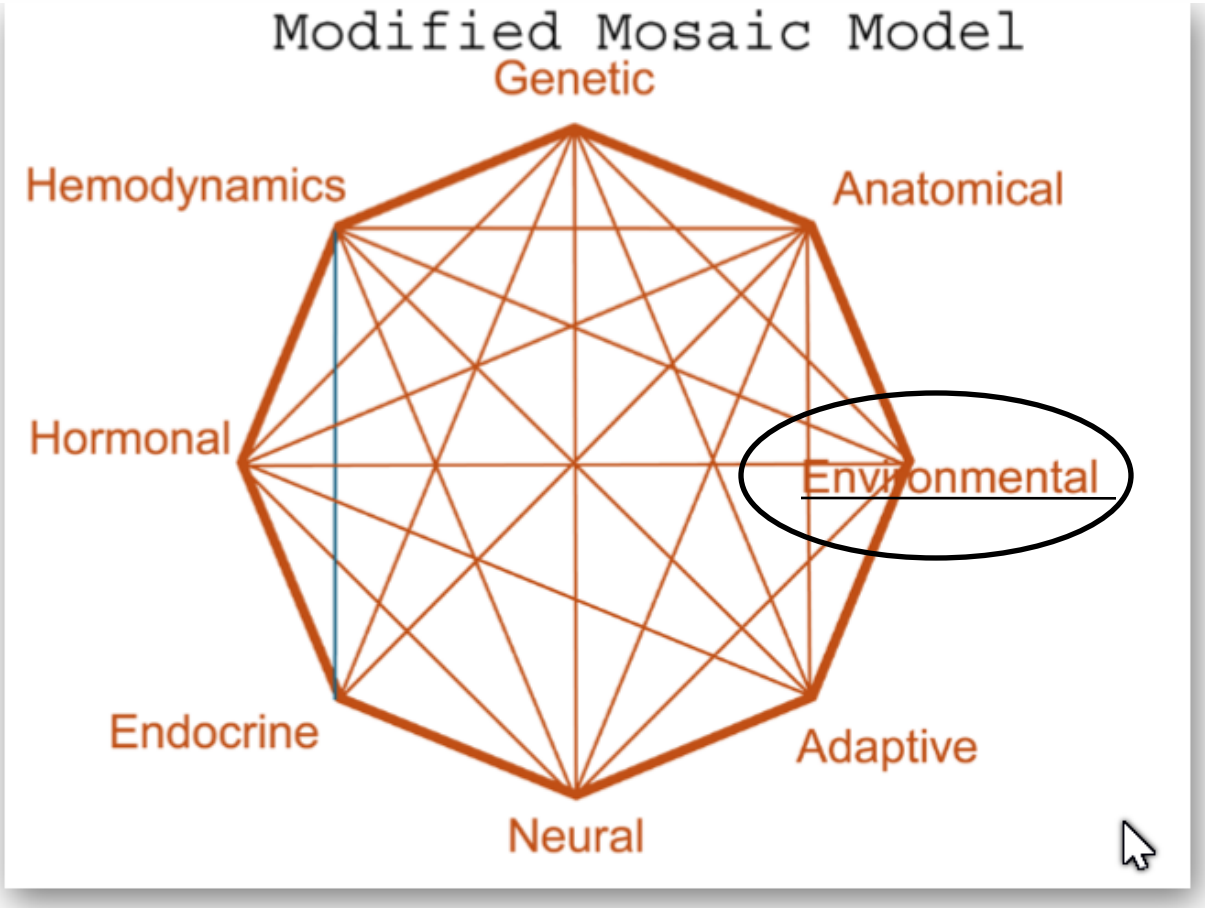
Hypertension. 2012 ;59:943-8.



REVIEW

Environmental Hypertensionology and the Mosaic Theory of Hypertension

Sanjay Rajagopalan, Robert D. Brook, Thomas Münzel





Hypertension. 2025;82:561–572.

INDOOR (HOUSEHOLD) POLLUTION

Il fumo di sigaretta



Original Investigation

An experimental investigation of tobacco smoke pollution in cars

Taryn Sendzik, Geoffrey T. Fong, Mark J. Travers, & Andrew Hyland

Nicotine & Tobacco Research, Volume 11, Number 6 (June 2009)

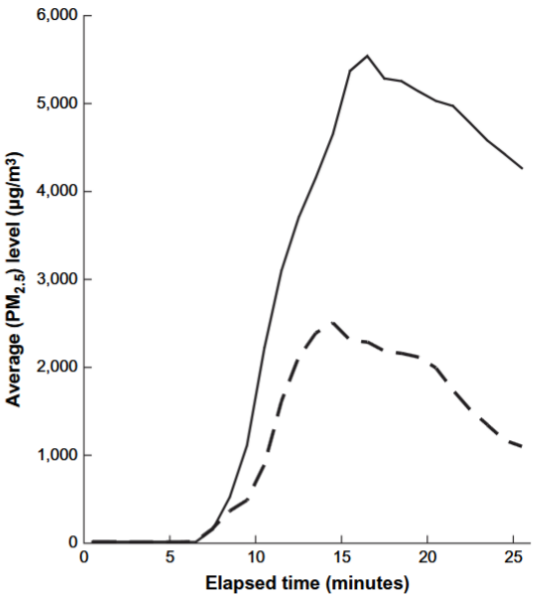


Figure 2. Average levels of $PM_{2.5}$ measured in Conditions 1 and 2. (a) Average for Condition 2 excludes data collected in car 4 due to cigarette relighting. (b) The EPA 24-hr exposure limit is 35 µg/m³. Solid line = Condition 1, one cigarette smoked, engine off, and windows closed. Broken line = Condition 2, one cigarette smoked, 20-min drive, and windows closed.

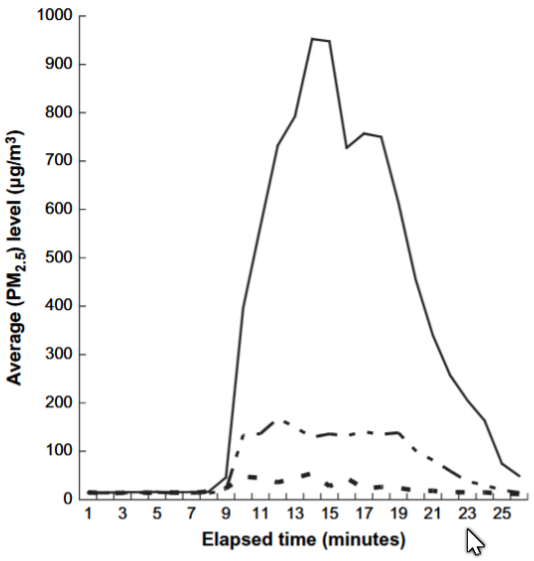


Figure 3. Average levels of $PM_{2.5}$ measured in Conditions 3, 4, and 5. (a) Average for Condition 5 excludes data collected in car 4 due to cigarette relighting. (b) Average for Condition 4 excludes data collected in car 4 due to machine failure. (c) The EPA 24-hr exposure limit is 35 µg/m³. Solid line = Condition 3, one cigarette smoked, 20-min drive, and all windows open. Long–short dashed line = Condition 4, one cigarette smoked, 20-min drive, and driver's window half-open. Dashed line = Condition 5, one cigarette smoked, 20-min drive, and air conditioner on.



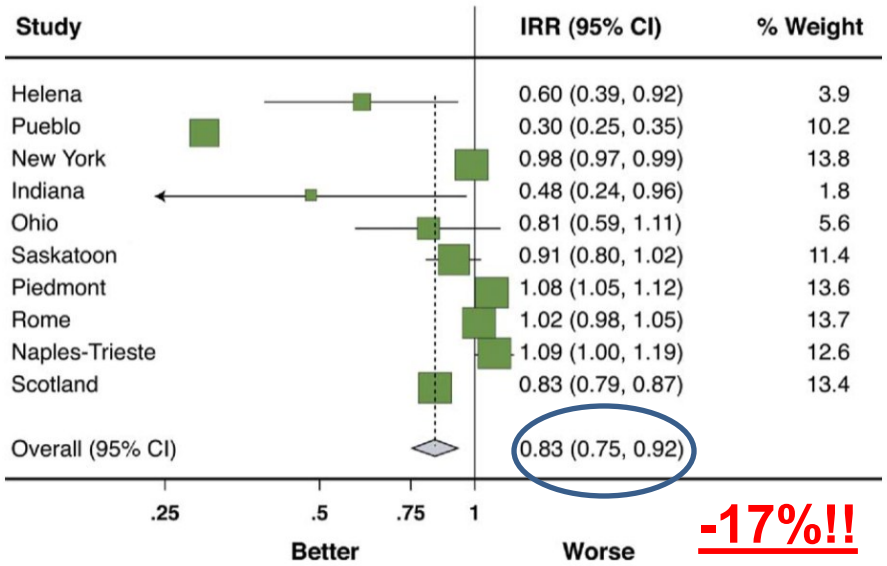
Nicotine Tob Res. 2009 Jun;11(6):627-34.

QUARTERLY FOCUS ISSUE: PREVENTION/OUTCOMES

Cardiovascular Effect of Bans on Smoking in Public Places

A Systematic Review and Meta-Analysis

David G. Meyers, MD, MPH,*† John S. Neuberger, DrPH, MPH, MBA,† Jianghua He, PhD‡
Kansas City, Kansas



-17%!!

Figure 1 Effects of Community Smoking Bans on Incident Acute Myocardial Infarction (Person-Year Approach)

Meta-analysis results for 11 studies in 10 geographic locations. CI = confidence interval; IRR = incidence rate ratio.



J Am Coll Cardiol. 2009; 54:1249-55

PM2.5 E DIABETE



Estimates, trends, and drivers of the global burden of type 2 diabetes attributable to $PM_{2·5}$ air pollution, 1990–2019: an analysis of data from the Global Burden of Disease Study 2019

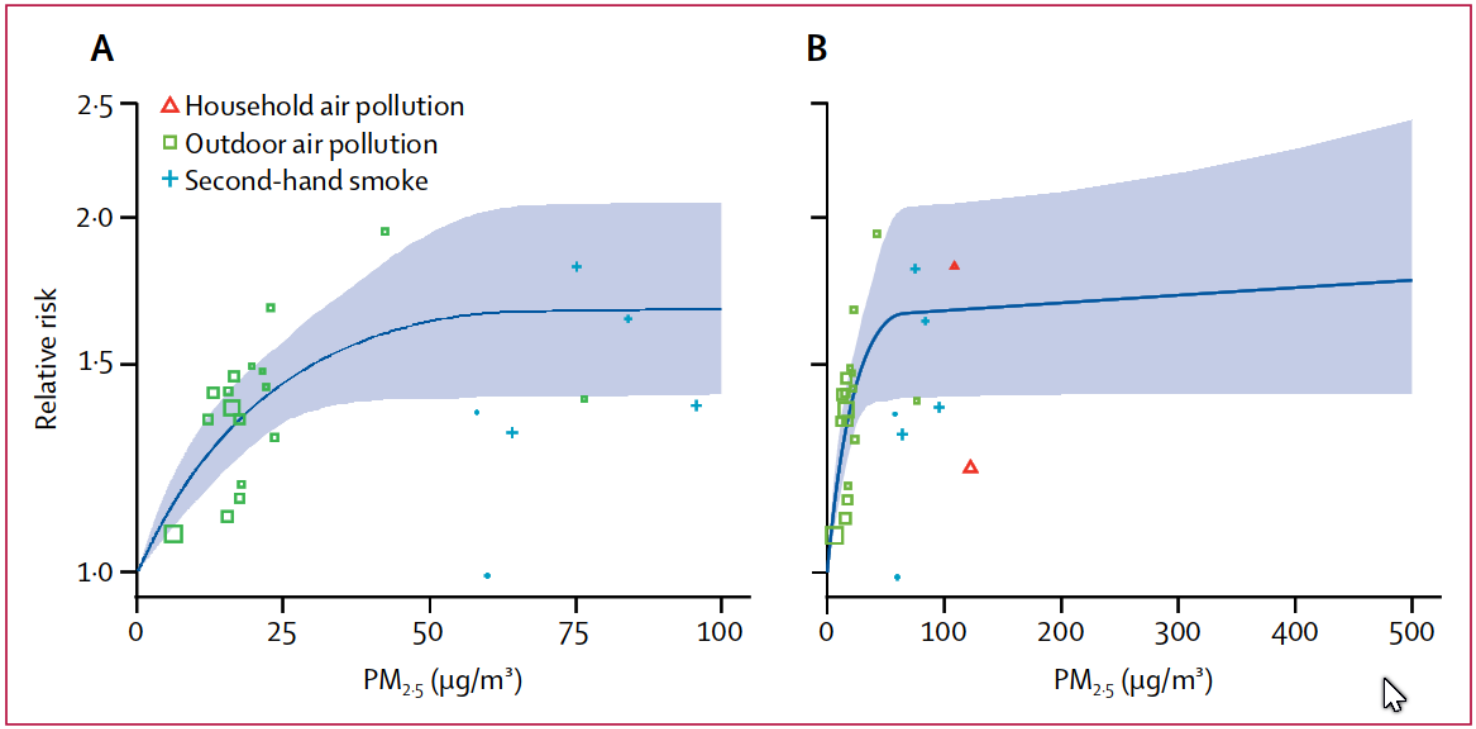


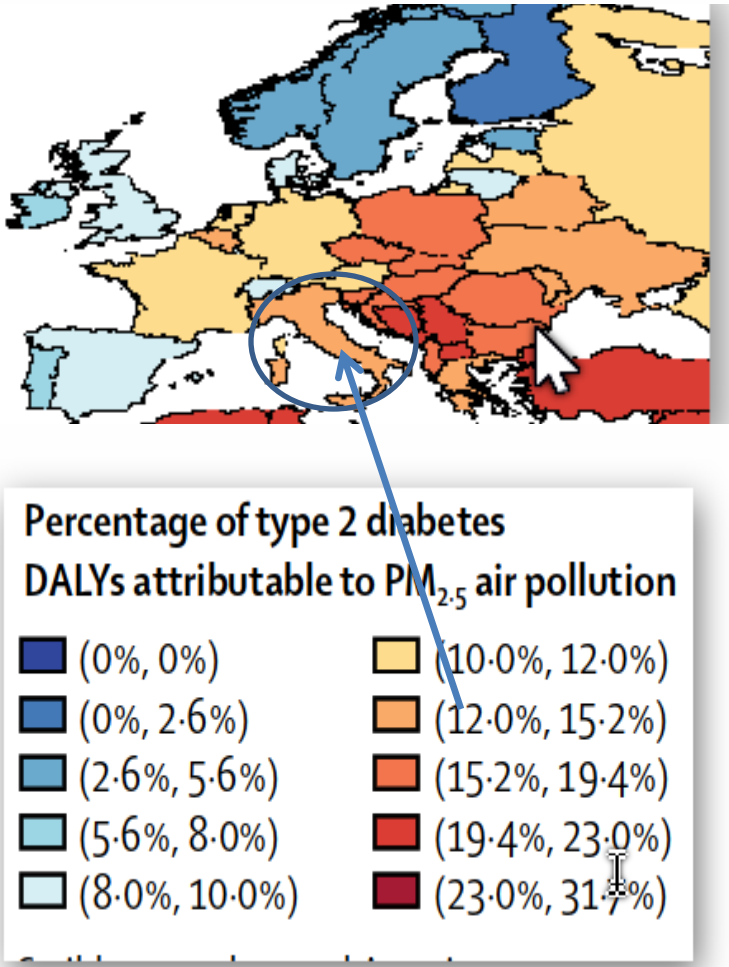
Figure 1: Exposure–response function for $PM_{2·5}$ exposure and type 2 diabetes for an exposure range of 0–100 µg/m³ (A) and 0-500 µg/m³ (B)

Interpretation Air pollution is a major risk factor for diabetes. We estimated that about a fifth of the global burden of type 2 diabetes is attributable $PM_{2·5}$ pollution. Air pollution mitigation therefore might have an essential role in reducing the global disease burden resulting from type 2 diabetes.



Lancet Planet Health 2022;6: e586–600

Estimates, trends, and drivers of the global burden of type 2 diabetes attributable to $PM_{2.5}$ air pollution, 1990–2019: an analysis of data from the Global Burden of Disease Study 2019





Lancet Planet Health 2022;6: e586–600

Associations of $PM_{2.5}$ exposure with diabetes-related mortality for California residents

Richy Zheng[1], Jason G. Su[1]*, Eahsan Shahriary[1], Sadeer Al-Kindi[2], Michael Jerrett[3], Timothy T. Brown[1], Rob McConnell[4] and John Balmes[5]

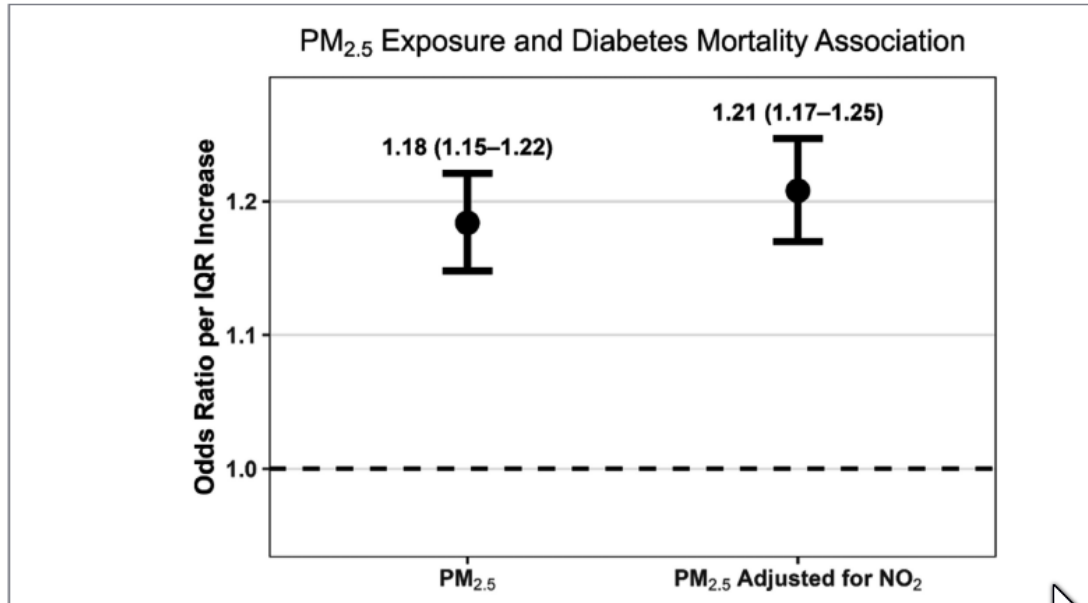


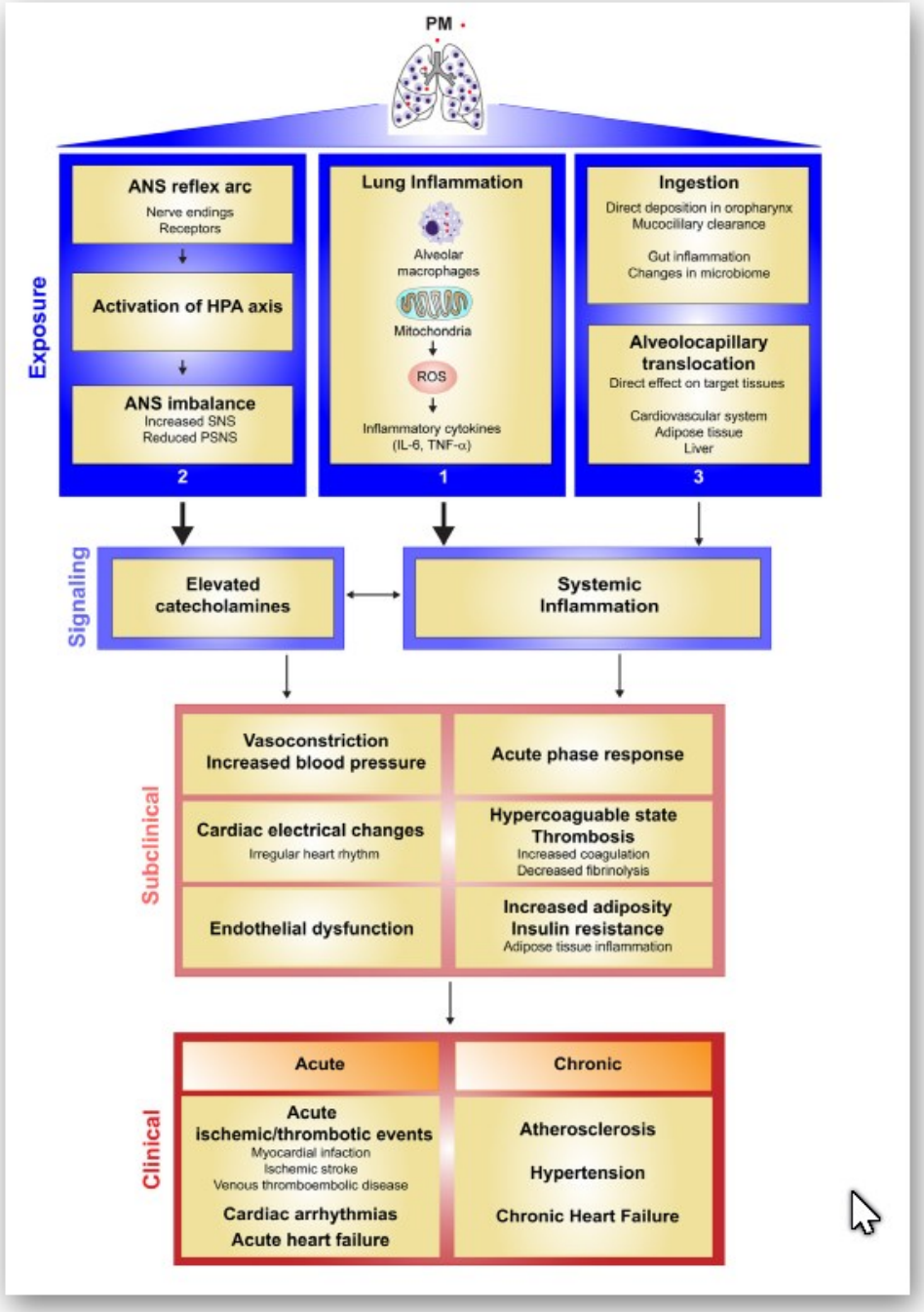
FIGURE 2
Association of $PM_{2.5}$ exposure (standardized with an IQR of 2.65 μg/m³) with diabetes mortality in single- and two-pollutant models.



Front Public Health. 2026 Aug 27;14:1903823.

MECCANISMI BIOLOGICI PATOGENETICI







Front. Endocrinol. 9:680

CONCLUSIONE 1.

- L’evidenza disponibile dimostra un rapporto CAUSALE fra esposizione a PM e patologie cardiovascolari
- L'esposizione a concentrazioni elevate di PM2.5 rappresenta un FATTORE DI RISCHIO CARDIOVASCOLARE MODIFICABILE analogo, in questo, ad ipertensione arteriosa ed iperclesterolemia LDL



CONCLUSIONE 2.

- Data la sua natura, tuttavia, un intervento efficace richiede(rebbe) provvedimenti politici, economici e sociali complessi ed altamente coordinati
- Indispensabile tuttavia l'informazione scientica e la sua divulgazione volta anche al cambiamento di abitudini e comportamenti individuali

