Air Pollution and Health Effects
Role and Perspectives of Simulation-Based Approaches

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QUALITÀ DELL'ARIA - DATI VALIDATI

Dati Validati - Provincia di Treviso

Bollettino del 30/09/2019 Dati riferiti al 29/09/2019			NO_2			PM10		O_3			SO_2				
			max ora			media giorn.		max ora		max giorn. media mob. 8h	max ora			max gi mob. 8h	
IQA	Ubicazione	Tipo stazione	conc. (µg/m^3)	ora	sup.	conc. (µg/m^3)	sup.	conc. (µg/m^3)	ora	conc. (µg/m^3)	conc. (µg/m^3)	ora	sup.	conc. (mg/m^3)	sup.
-	TV - Strada S. Agnese	TU	32	20	-	20	45				< 3		-	0.1	-
-	Pederobba	BU	28	19	-	22	5							0.2	-
●	Conegliano	BU	45	19	-	20	16	91	14	81					
●	TV - Via Lancieri di Novara	BU	73	21	-	16	42	98	15	84					
●	Mansue	BRU	7	18	-	19	41	101	16	82					

Archivio storico bollettini

Informazioni sull'indice di qualità dell'aria (IQA)

I valori riportati in tabella possono, sporadicamente, subire modifiche a seguito di verifiche eseguite trimestralmente sulle serie più lunghe di dati

Legenda

IQA Indice di qualità dell'aria
- Buona
- Accettabile
- Mediocre
- Scadente
- Pessima
- \- Indice non calcolabile

Applied Mathematical Modelling 65 (2019) 52–71

Contents lists available at ScienceDirect

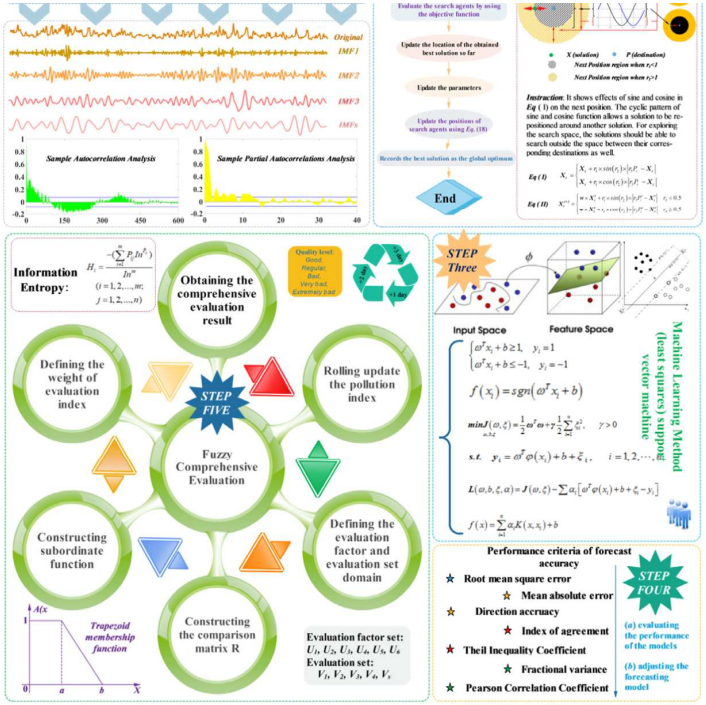
Applied Mathematical Modelling

journal homepage: www.elsevier.com/locate/apm

A dynamic evaluation framework for ambient air pollution monitoring

Ranran Li, Yuqi Dong, Zhijie Zhu*, Chen Li, Hufang Yang

School of Statistics, Dongbei University of Finance and Economics, Dalian 116025, China



Air Pollution and Human Health

Lester B. Lave and Eugene P. Seskin

Air pollution is a problem of growing importance; public interest seems to have risen faster than the level of pollution in recent years. Presidential messages and news stories have reflected the opinion of scientists and civic leaders that pollution must be abated. This concern has manifested itself in tightened local ordinances (and, more importantly, in increased enforcement of existing ordinances), in federal legislation, and in extensive research to find ways of controlling the emission of pollutants from automobiles and smokestacks. Pollutants are natural constituents of the air. Even without man and his technology, plants, animals, and natural activity would cause some pollution. For example, animals vent carbon dioxide, volcanic action produces sulfur oxides, and wind movement insures that there will be suspended particulates; there is no possibility of removing all pollution from the air. Instead, the problem is one of balancing the need of polluters to vent residuals against the damage suffered by society as a result of the increased pollution (*1*). To find an optimum level, we must know the marginal costs and marginal benefits associated with abatement. This article is focused on measuring one aspect of the benefit of pollution abatement.

Polluted air affects the health of human beings and of all animals and plants (*2*). It soils and deteriorates property, impairs various production processes (for example, the widespread use of "clean rooms" is an attempt to reduce contamination from the air), raises the rate of automobile and airline accidents (*3*), and generally makes living things less comfortable and less happy. Some of these effects are quite definite and measurable, but most are ill-defined and difficult to measure, even conceptually. Thus, scientists still disagree on the quantitative effect of pollution on animals, plants, and materials. Some estimates of the cost of the soiling and deterioration of property have been made, but the estimates are only a step beyond guesses (*4*). We conjecture that the major benefit of pollution abatement will be found in a general increase in human happiness or improvement in the "quality of life," rather than in one of the specific, more easily measurable categories. Non
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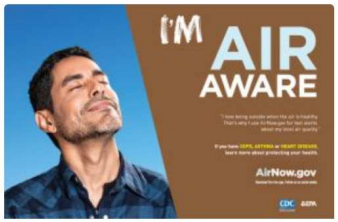
SCIENCE, 1970, vol. 169, pp. 723-733.

I'm Air Aware



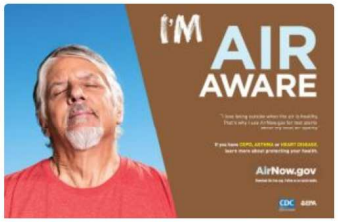
[PDF - 28 MB]

"I love being outside when the air is healthy. That's why I use AirNow.gov for text alerts about my local air quality."

If you have COPD, ASTHMA, or HEART DISEASE, learn more about protecting your health.

AirNow.gov

Download the free app. Follow us on social media.



[PDF - 36 MB]

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EDITORIAL
ENVIRONMENT AND THE LUNG

Improvements in air quality: whose lungs benefit?

Ulrike Gehring[1] and Gerard H. Koppelman[2,3]

being outside when the air is healthy. That's why I use
w.gov for text alerts about my local air quality."

have COPD, ASTHMA, or HEART DISEASE, learn more
protecting your health.

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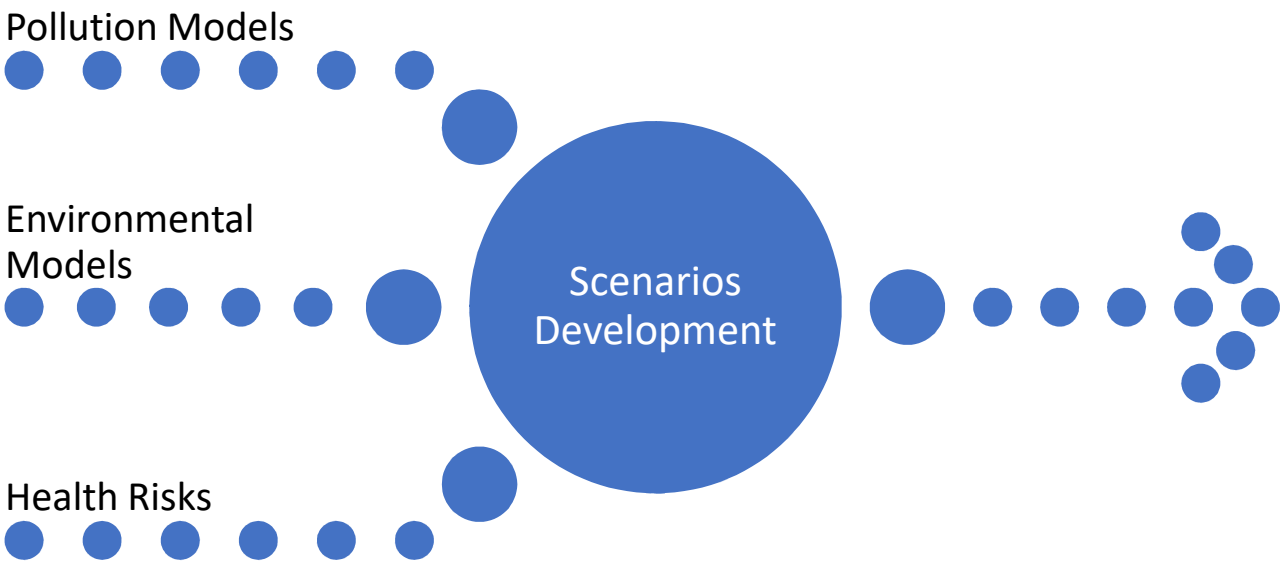
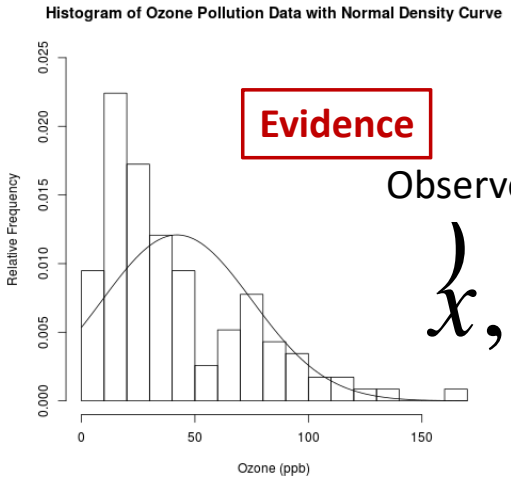


Data Analysis and Estimation
Mathematical Modeling

(Stochastic) simulation

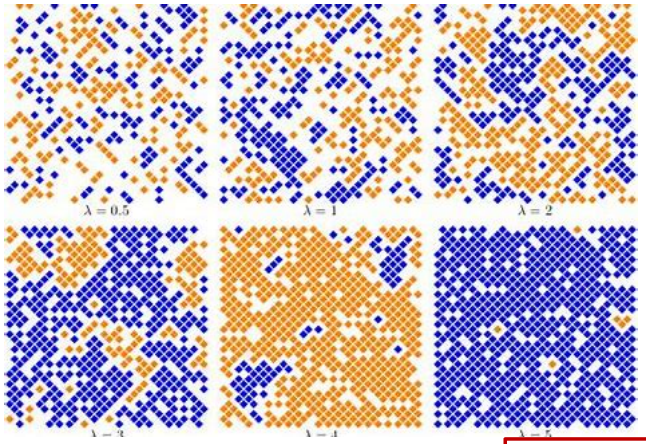
Decision Making Tools



Evidence

Observed Data

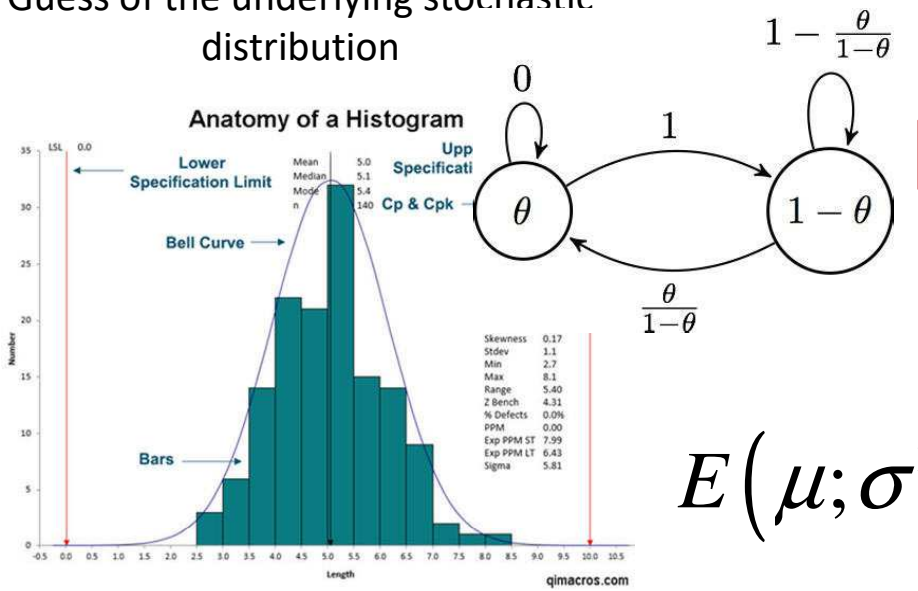
$$x, s^2$$



Monte Carlo Methods

Simulation

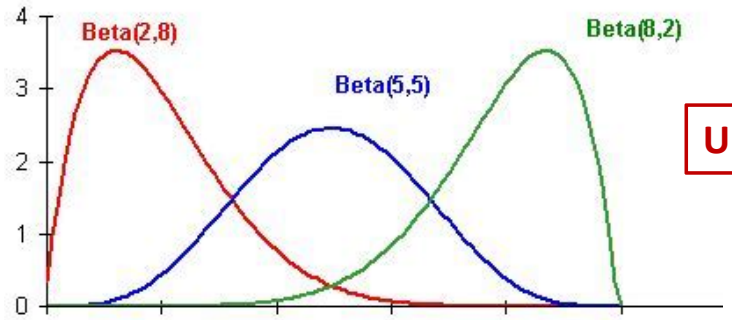
Guess of the underlying stochastic distribution



Model

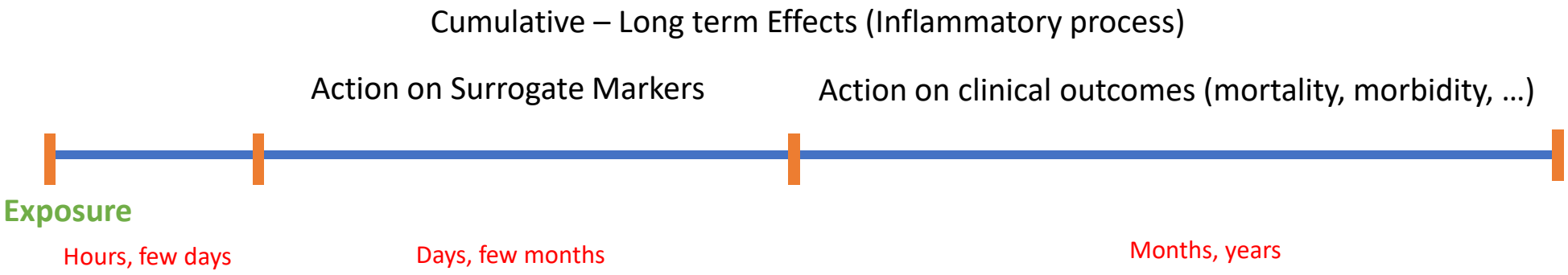
$$E\left(\mu;\sigma^2\right)$$

$$\mu \sim F\left(\mathrm{g}\right)$$



Uncertainty

Macro and Micro-Evidence



Short term

Immediate reaction (Sympathetic nervous channel stimulation)

Surrogate Markers

International Journal of *Environmental Research and Public Health*

MDPI

Article

Effects of Fine Particulate Matter ($PM_{2.5}$) on Systemic Oxidative Stress and Cardiac Function in $ApoE^{-/-}$ Mice

Yiling Pei [1], Rongfang Jiang [1], Yunzeng Zou [2], Yu Wang [3], Suhui Zhang [3], Guanghe Wang [4], Jinzhuo Zhao [1,*] and Weimin Song [1,*]

Table 4. Echocardiography changes in experimental groups after $PM_{2.5}$ exposure.

Groups	EF	FS	LVES*d*	LVED*d*	LVPW*d*	LVAW*d*
C57BL/6 + saline	57.21 ± 12.11	30.38 ± 8.16	3.14 ± 0.73	4.46 ± 0.63	0.69 ± 0.14	0.75 ± 0.12
C57BL/6 + $PM_{2.5}$ (3)	54.04 ± 7.06	28.12 ± 4.73	3.4 ± 0.21	4.73 ± 0.30	0.62 ± 0.07	0.70 ± 0.09
C57 BL/6 + $PM_{2.5}$ (10)	53.74 ± 13.40	28.19 ± 8.96	3.19 ± 0.56	4.43 ± 0.31	0.64 ± 0.12	0.69 ± 0.09
C57 BL/6 + $PM_{2.5}$ (30)	50.21 ± 7.32	25.49 ± 4.69	3.28 ± 0.37	4.4 ± 0.24	0.57 ± 0.09	0.63 ± 0.08
$ApoE^{-/-}$ + saline	67.69 ± 9.07	37.76 ± 7.34	2.57 ± 0.45	4.1 ± 0.28	0.74 ± 0.05	0.87 ± 0.18
$ApoE^{-/-}$ + $PM_{2.5}$ (3)	72.57 ± 6.93 [a,b]	41.49 ± 6.37 [a,b]	2.27 ± 0.43	3.84 ± 0.38	0.70 ± 0.07	0.78 ± 0.06
$ApoE^{-/-}$ + $PM_{2.5}$ (10)	74.76 ± 8.20 [a,b]	43.16 ± 6.75 [a,b]	2.52 ± 0.61	3.89 ± 0.66	0.77 ± 0.07	0.84 ± 0.08
$ApoE^{-/-}$ + $PM_{2.5}$ (30)	67.67 ± 12.25	37.97 ± 9.35	2.51 ± 0.75	3.98 ± 0.65	0.80 ± 0.18 [a]	0.96 ± 0.30 [a]

Table 4 Echocardiography changes in experimental groups after exposed to $PM_{2.5}$. The doses were 0 (control), 3, 10, and 30 mg/kg b.w., respectively. Significant difference ([a] $p < 0.05$) between $PM_{2.5}$ group and control group, significant difference ([b] $p < 0.05$) between C57BL/6 and dose-matched $ApoE^{-/-}$ mice.

In this study, acute exposure to PM2.5 was associated with increased oxidative stress, an imbalance in cardiac autonomic control, and an abnormal ECG type of myocardial ischemia and hypoxia in mice.

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Long-term Air Pollution Exposure and Acceleration of Atherosclerosis and Vascular Inflammation in an Animal Model

Qinghua Sun, MD, PhD
Aixia Wang, BS
Ximei Jin, BS

Context Recent studies have suggested a link between inhaled particulate matter exposure in urban areas and susceptibility to cardiovascular events; however, the precise mechanisms remain to be determined.

Table 1. Weight and Lipid Measurements of Mice Fed Normal vs High-Fat Chow and Exposed to $PM_{2.5}$ vs Filtered Air

	Normal Chow, Mean (SD)			High-Fat Chow, Mean (SD)		
	Filtered Air (n = 8)	$PM_{2.5}$ (n = 8)	*P* Value*	Filtered Air (n = 6)	$PM_{2.5}$ (n = 6)	*P* Value*
Weight, g						
Before exposure	20 (2)	20 (1)	<.001	20 (1)	21 (1)	<.001
After exposure	27 (3)	28 (2)	<.001	34 (7)	33 (5)	<.001
Lipids, mg/dL†						
Cholesterol	850.6 (94.0)	783.4 (88.1)	<.001	1257.8 (82.2)	1314.8 (296.3)	.007
Triglycerides	513.4 (208.8)	506.8 (251.9)	.80	502.2 (170.7)	444.7 (95.5)	.15

Abbreviation: $PM_{2.5}$, concentrated ambient particles of less than 2.5 μm.
SI conversions: To convert cholesterol to mmol/L, multiply by 0.0259; triglycerides to mmol/L, multiply by 0.0113.
*Differences between 2 group observations were compared with *t* test.
†After exposure to either $PM_{2.5}$ or filtered air in mice fed normal or high-fat chow.

In an animal model of apoE−/− mice, exposure to environmentally relevant concentrations of regional northeastern PM2.5 accelerates atherosclerosis.
PM2.5 exposure also attenuates responsiveness to an endothelium-dependent agonist and heightens vasoconstrictor responsiveness.
Additionally, vascular inflammation and protein nitration are prominent aspects of PM2.5-mediated effects on the vasculature.

CURRENT OPINION

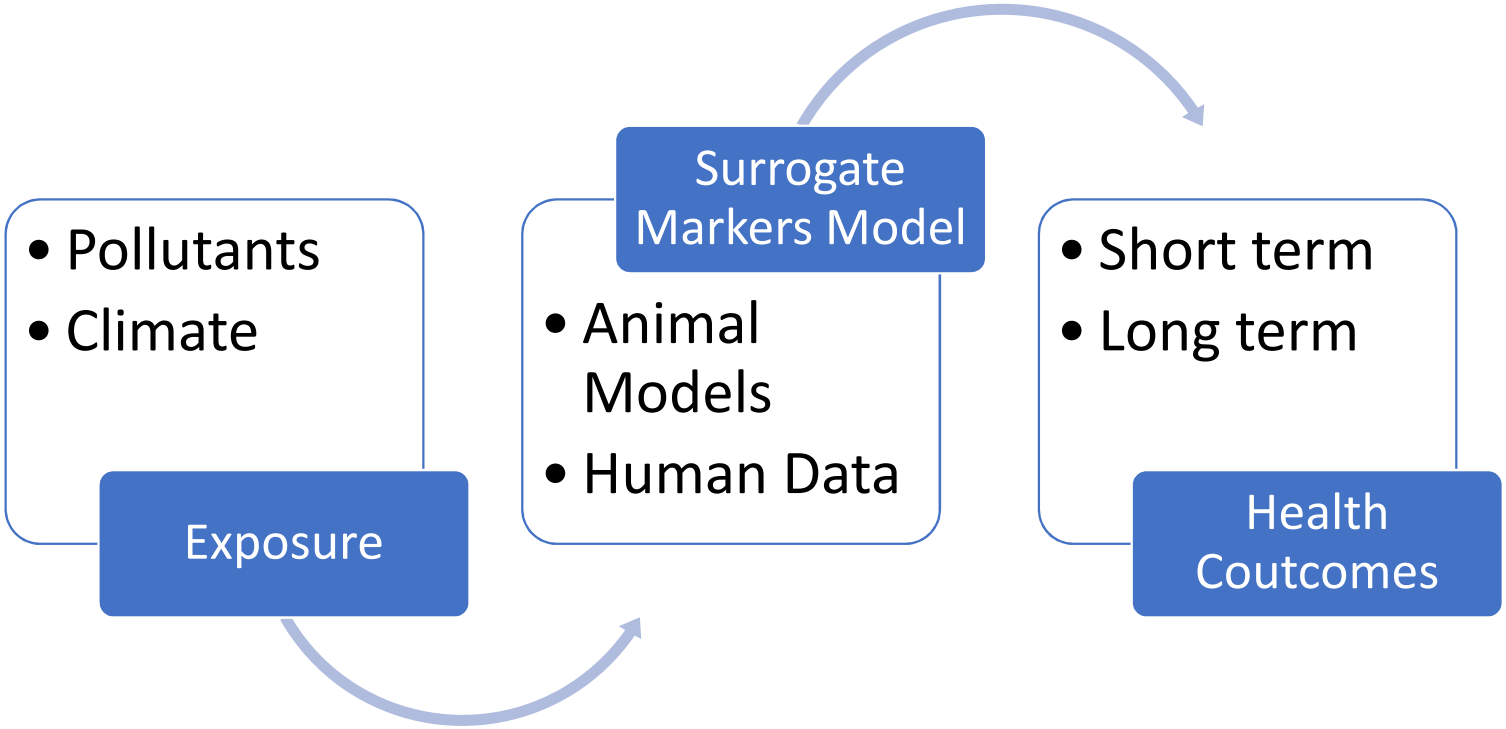
HDL-cholesterol and cardiovascular disease: rethinking our approach

Hasan K. Siddiqi, Daniel Kiss, and Daniel Rader

Purpose of review
A low level of plasma high density lipoprotein cholesterol (HDL-C) is a strong and independent risk factor for atherosclerotic cardiovascular disease (ASCVD). However, several large studies recently revealed that pharmacologic interventions that increase HDL-C concentration have not improved cardiovascular outcomes when added to standard therapy. In addition, specific genetic variants that raise HDL-C levels are not clearly associated with reduced risk of coronary heart disease. These observations have challenged the 'HDL hypothesis' that HDL-C is causally related to ASCVD and that intervention to raise HDL-C will reduce ASCVD events. This article will present the current data on the HDL hypothesis and provide a revised paradigm of considering HDL in the atherosclerotic pathway.

Recent findings
Recent evidence has shed light on the complex nature of HDL-C metabolism and function. There are compelling data that the ability of HDL to promote cholesterol efflux from macrophages, the first step in the 'reverse cholesterol transport' (RCT) pathway, is inversely associated with risk for ASCVD even after controlling for HDL-C. This has led to the 'HDL flux hypothesis' that therapeutic intervention that targets macrophage cholesterol efflux and RCT may reduce risk. Preclinical studies of such interventions show promise and early phase clinical studies, though small, are encouraging.

Summary
The role of HDL-C in modulating atherosclerotic disease is as yet uncertain. However, new findings and therapies targeting HDL-C show early promise and may provide an important intervention in attenuating the burden of ASCVD in the future.


• Pollutants
• Climate
Exposure
Surrogate Markers Model
• Animal Models
• Human Data
• Short term
• Long term
Health Coutcomes

Ecological Outcome studies

Poisson – based models for counts
Aggregate effects on outcomes
Epidemiological Risk Models

$$\log(\mu) = \beta X$$

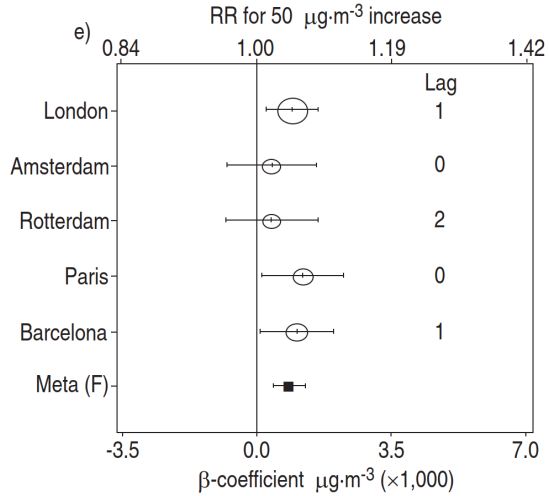


Fig. 1. – Relative risks (RR) and (95% confidence limits) for daily admissions for COPD, for each city, associated with a 50 µg·m^{-3} increase in pollutant. Summary estimate from meta-analysis shown as Meta (F) = fixed effects model or Meta (R) = random effects model. Abscissa show beta-coefficient for log of admissions. The circled area is proportional to the weight attributed to each city in the meta-analysis. a) 24 h sulphur dioxide; b) 24 h black smoke; c) total suspended particulates; d) 24 h nitrogen dioxide; e) 8 h ozone. Lag: effects may be on the same day or lagged up to 3 days (ozone 5 days).

Eur Respir J 1997; 10: 1064–1071
DOI: 10.1183/09031936.97.10051064
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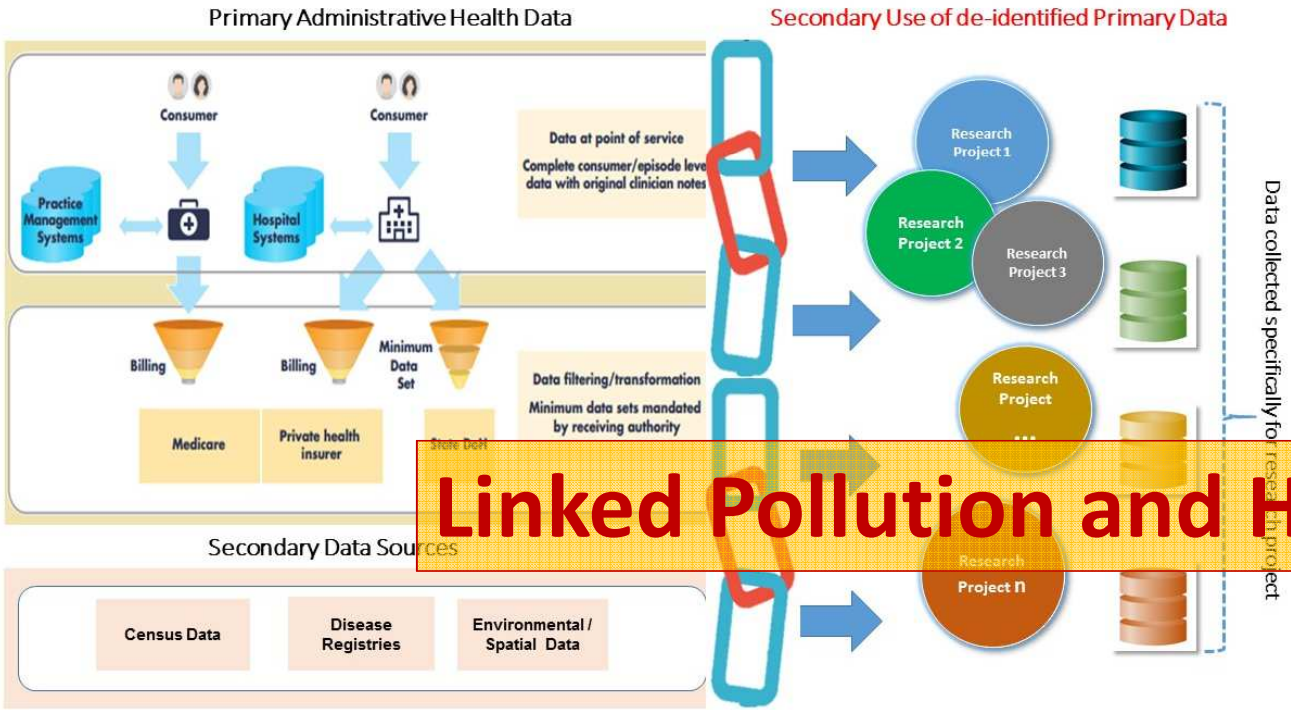
European Respiratory Journal
ISSN 0903 - 1936

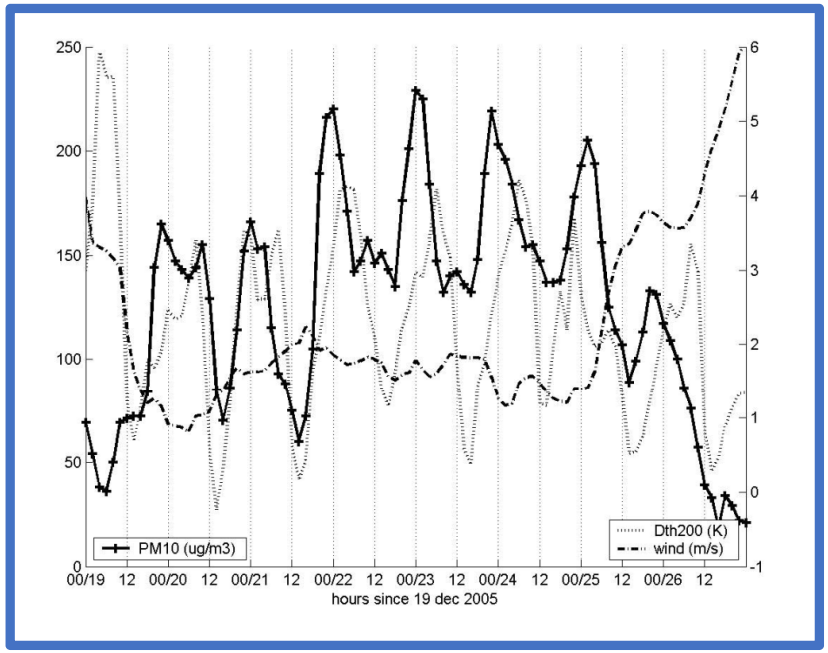
Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project

H.R. Anderson*, C. Spix**, S. Medina***, J.P. Schouten+, J. Castellsague++, G. Rossi+++, D. Zmirou‡, G. Touloumi‡‡, B. Wojtyniak‡‡‡, A. Ponka#, L. Bacharova##, J. Schwartz###, K. Katsouyanni‡‡‡

Details of each city's methods have been reported previously [13–17]. From routine sources, daily counts of emergency hospital admissions for ICD9 490 (unspecified bronchitis), 491 (chronic bronchitis), 492 (emphysema) and 496 (chronic airways obstruction) were obtained. For the purpose of this analysis, these four codes comprise COPD. In Barcelona, data collection was part of a special project. The admissions covered

Individual – Population Based Models





Linked Pollution and Health Care Records

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Association between air pollution and ventricular arrhythmias in high-risk patients (ARIA study): a multicentre longitudinal study

Franco Folino, Gianfranco Buja, Gabriele Zanotto, Elena Marras, Giuseppe Allocca, Diego Vaccari, Gianni Gasparini, Emanuele Bertaglia, Franco Zoppo, Vittorio Calzolari, Rene Nangah Suh, Barbara Ignatiuk, Corrado Lanera, Alessandro Benassi, Dario Gregori, Sabino Iliceto

Summary

Background Although the effects of air pollution on mortality have been clearly shown in many epidemiological and observational studies, the pro-arrhythmic effects remain unknown. We aimed to assess the short-term effects of air pollution on ventricular arrhythmias in a population of high-risk patients with implantable cardioverter-defibrillators (ICDs) or cardiac resynchronisation therapy defibrillators (ICD-CRT).

Lancet Planet Health 2017; 1: e58–64

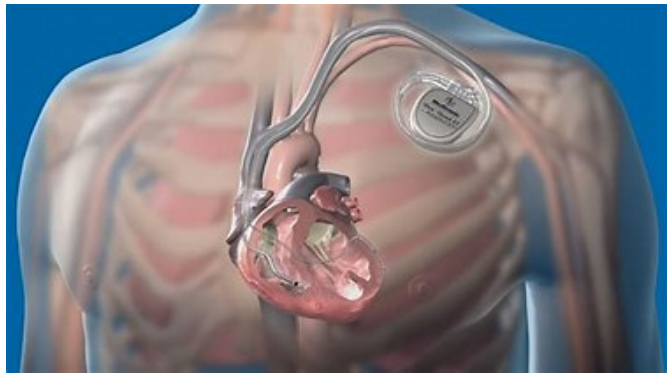
See Comment page e50

Department of Cardiac Thoracic and Vascular Sciences,

Clinical Health Records

ARIA Study Group

Administrative Health Records



281 patients (median age 71 years) across nine centres in the Veneto region of Italy. Episodes of ventricular tachycardia and ventricular fibrillation that were recorded by the diagnostic device were considered.
Concentrations of particulate matter of less than 10 µm (PM10) and less than 2·5 µm (PM2·5) in aerodynamic diameter, carbon monoxide, nitrogen dioxide, sulphur dioxide, and ozone were obtained daily from monitoring stations, and the 24 h median value was considered. Each patient was associated with exposure data from the monitoring station that was closest to their residence.

Estimated impact (2-years) on health

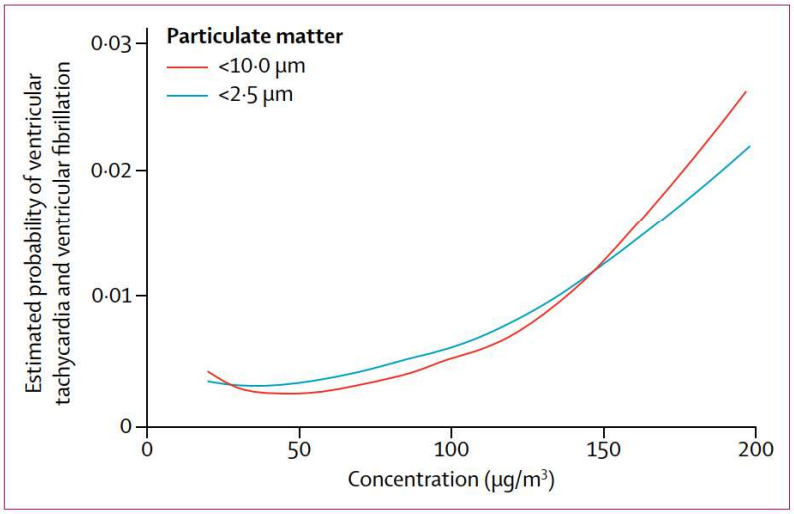


Figure 1: **Estimated probability plot for ventricular tachycardia and ventricular fibrillation episodes at different concentrations of particulate matter of less than 10 µm (PM_{10}) and less than 2·5 µm ($PM_{2·5}$) in aerodynamic diameter**

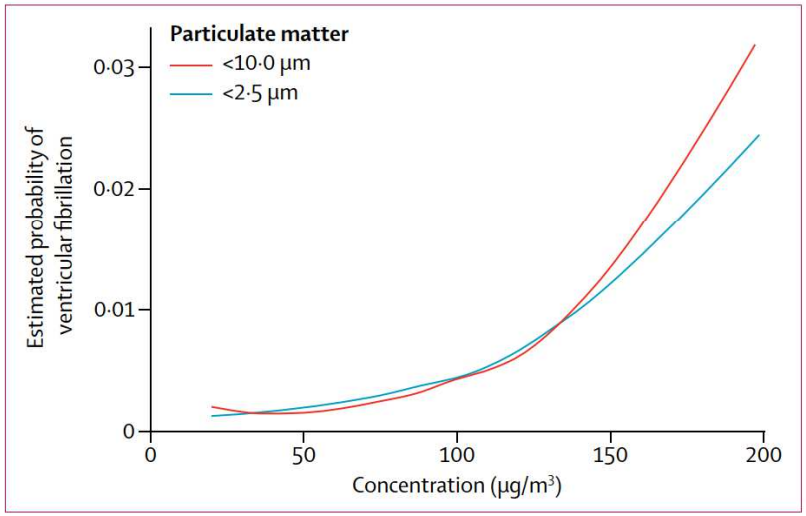
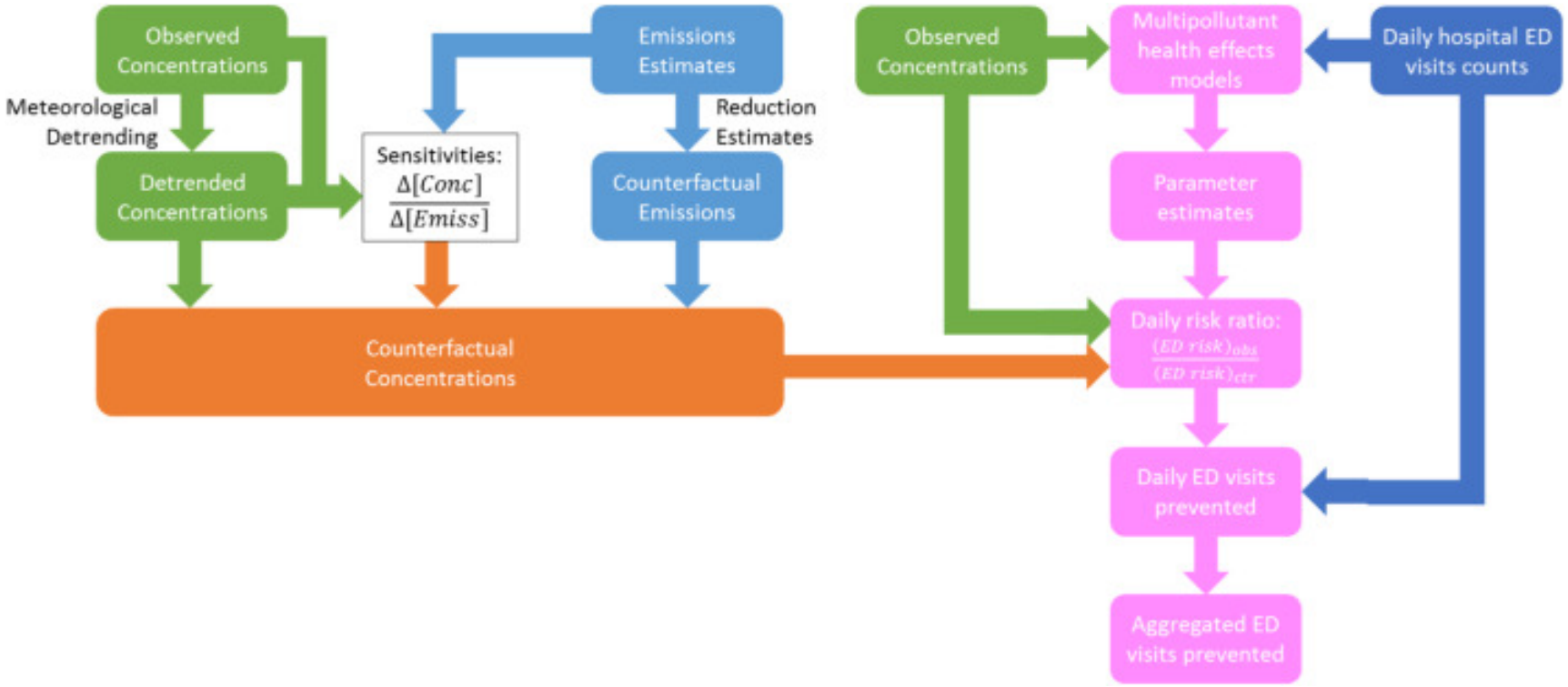


Figure 2: **Estimated probability plot for ventricular fibrillation episodes at different concentrations of particulate matter of less than 10 µm (PM_{10}) and less than 2·5 µm ($PM_{2·5}$) in aerodynamic diameter**

Emergency Room models for short-term effects



Impact of air pollution control policies on cardiorespiratory emergency department visits, Atlanta, GA, 1999–2013

Joseph Y. Abrams[a,*], Mitchel Klein[b], Lucas R.F. Henneman[c], Stefanie E. Sarnat[b], Howard H. Chang[d], Matthew J. Strickland[e], James A. Mulholland[e], Armistead G. Russell[c], Paige E. Tolbert[b]

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The virus causes inflammation of the small airways (bronchioles). The inflammation partially or completely blocks the airways leading to wheezing

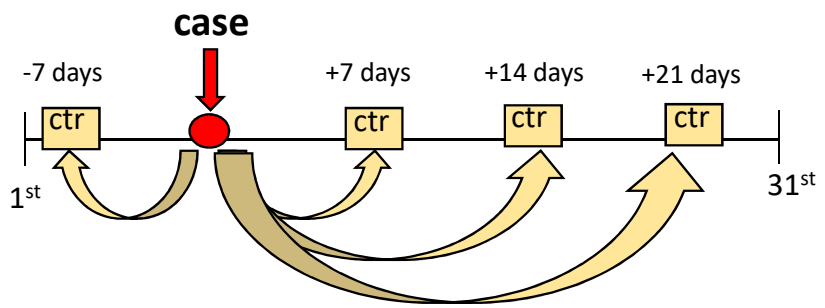
One of the first causes of Pediatric Emergency Room admission in infants

Children aged less than 1 year admited to Pediatric Emergency Room of Padova, Italy, for bronchiolitis between 2007 and 2018

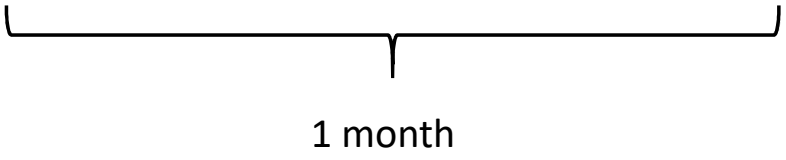
Hospital Admissions (individual data)

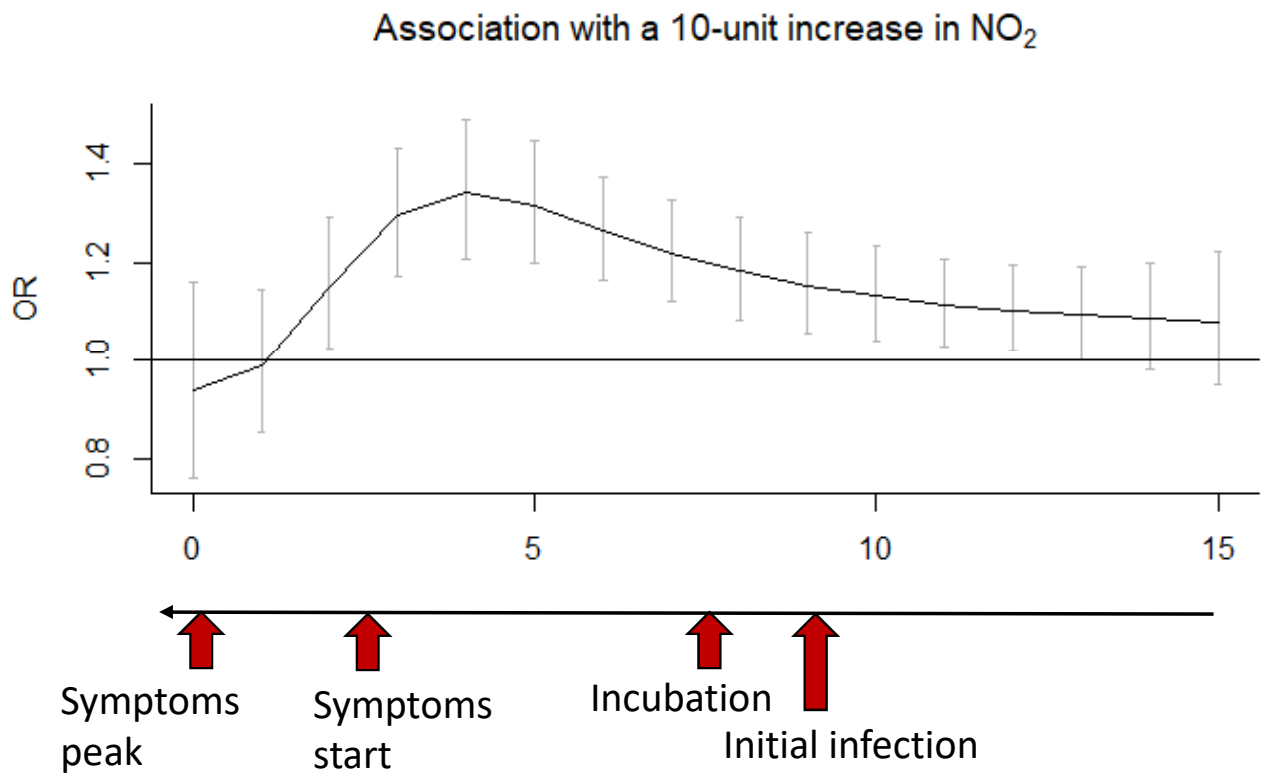
Linked with PMx exposure (ARPAV)

A conditional logistic regression based on a time-stratified case-crossover design was applied on single pollutant models

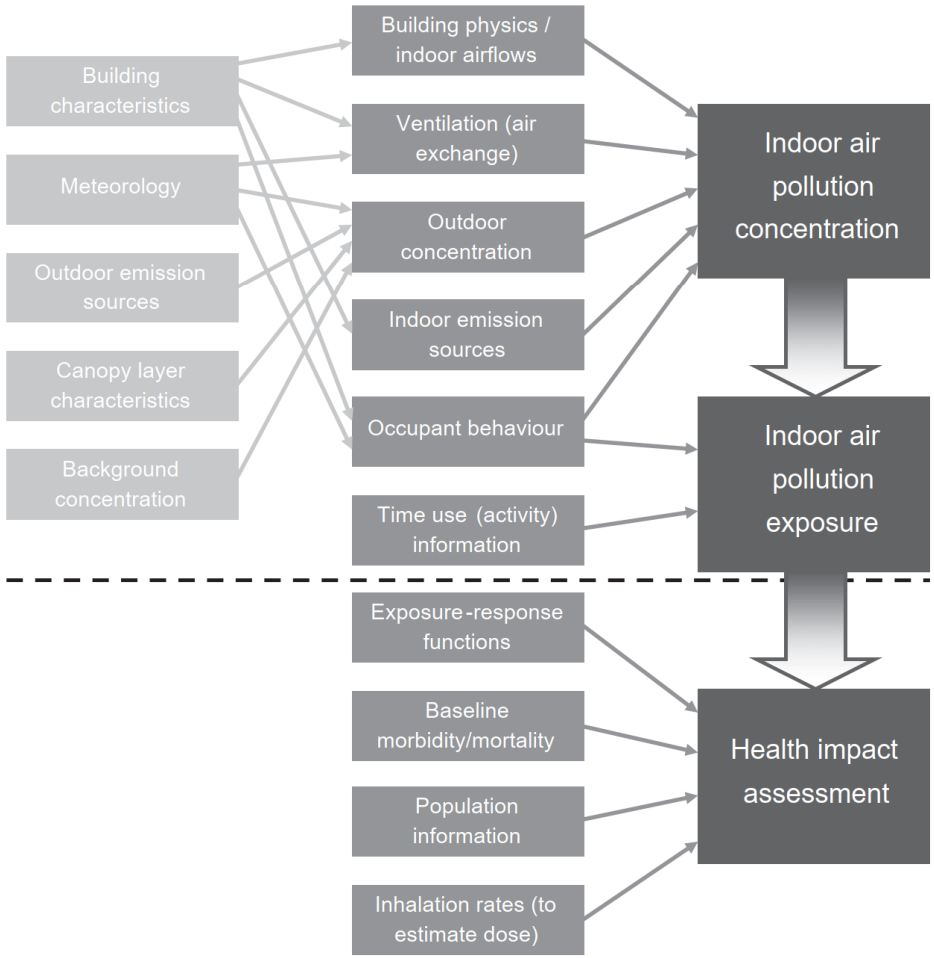


Delayed effect in time evaluated with DLNMs




Association with a 10-unit increase in NO_2
OR
0.8
1.0
1.2
1.4
0
5
10
15
Symptoms peak
Symptoms start
Incubation
Initial infection

A caesura between pollution and health models



Modelling inhalation exposure to combustion-related air pollutants in residential buildings: Application to health impact assessment

James Milner *, Sotiris Vardoulakis, Zaid Chalabi, Paul Wilkinson

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Cochrane Library

Cochrane Database of Systematic Reviews

Interventions to reduce ambient particulate matter air pollution and their effect on health (Review)

Burns J, Boogaard H, Polus S, Pfadenhauer LM, Rohwer AC, van Erp AM, Turley R, Rehfuess E

Many different policies and programmes have been put into place to reduce air pollution; examples include vehicle restrictions to reduce traffic, fuel standards for cars, buses and other motorized transport, industrial regulations to limit pollution from factories, and the replacement of inefficient heating stoves with more efficient, cleaner burning stoves.

We wanted to know whether these measures led to a reduction in the overall number of deaths, and in the number of deaths from cardiovascular and respiratory causes. We also investigated whether the measures led to fewer people going to hospitals for cardiovascular and respiratory problems.

Overall, we observed mixed results across studies. **Many studies observed no clear changes in health or air quality associated with the measures, while others did observe clear improvements. We identified very few studies that reported worsened health or air quality associated with the measures.**

The production of higher quality and more uniform evidence would be helpful in informing decisions.

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Ambio
https://doi.org/10.1007/s13280-019-01164-3

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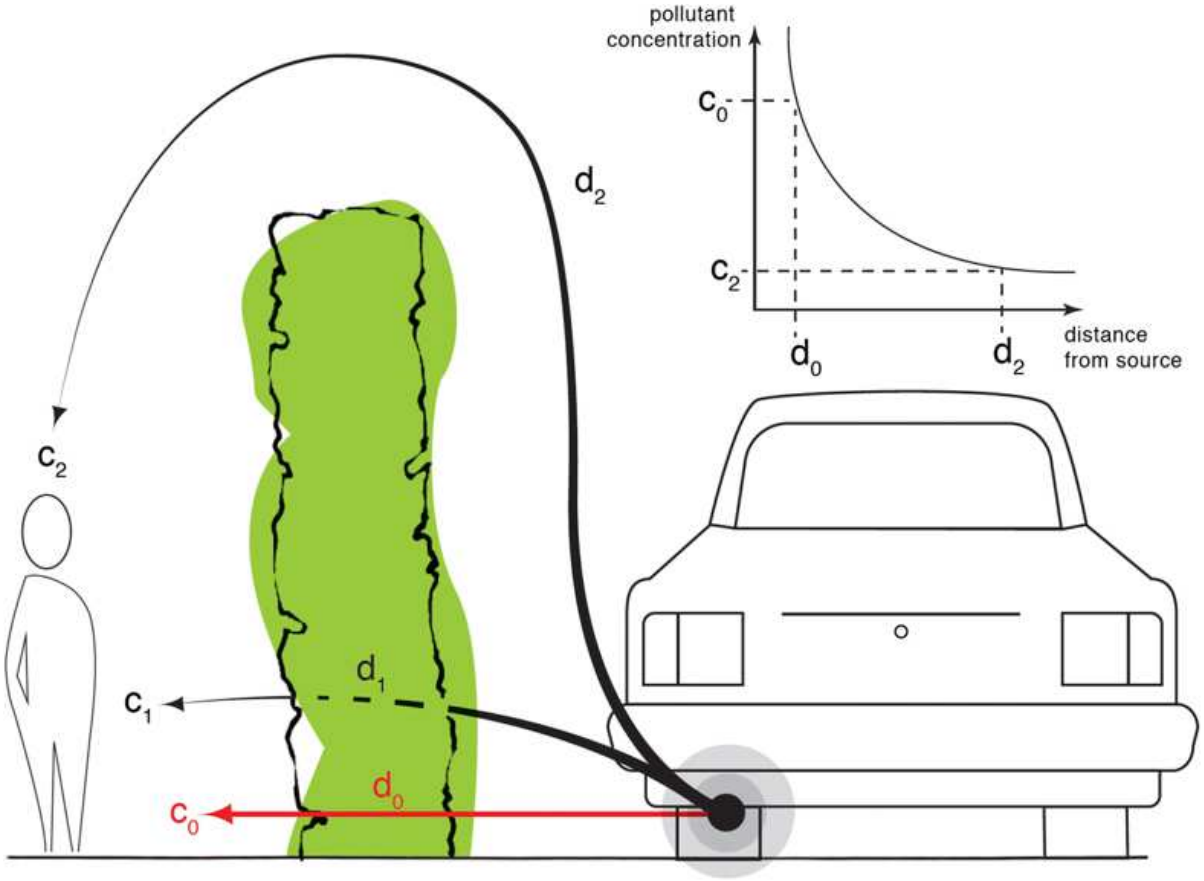
Check for updates

PERSPECTIVE

Using green infrastructure to improve urban air quality (GI4AQ)

C. Nick Hewitt, Kirsti Ashworth, A. Rob MacKenzie

Traslating evidence into scenarios



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General impact-models

$$\log\left(E\left(Y_{kt}\right)\right) = \propto + \beta_1 \text{ pollution }_{1,kt} + \beta_2 \text{ pollution }_{2,kt} +$$
$$\sum_k \lambda_k ZIP_k + \sum_m \lambda_m DOW_{mt} + \sum_n \nu_n \text{hospital}_{nt} +$$
$$g\left(\gamma_1, \ldots, \gamma_N; \text{tim}\, e_t\right) + \sum_o \xi_o \text{IOtemp}_{\text{ot}} +$$
$$\eta_1 \text{ dewpt}_t + \eta_2 \text{ dewpt}_t^2 + \eta_3 \text{ dewpt}_t^3 +$$
$$\delta_1 temp_t + \delta_2 \text{ temp}_t^2 + \delta_3 \text{ tem } p_t^3$$

- Y kt is the count of ED visits for respiratory outcomes in ZIP code k on day t.
- For each pollutant (pollution 1 and pollution 2), daily averages (8-h maximum for O3, 24-h average for all other pollutants) of same-day concentrations were used.
- To control for spatial autocorrelation in the baseline ED visits across the ZIP codes, an indicator variable for the kth ZIP code (ZIP k) was used to represent the areas from which ED counts were spatially aggregated.
- Dummy variables for day of week and holidays (DOW, indexed by m) and for hospital (hospital, indexed by n) were used; the latter accounted for the differing durations of time each hospital was included in the study.
- Long-term trends and seasonality of health outcomes (time) were controlled for with parametric cubic splines with monthly knots (g(γ 1, …, γ N ; x)).
- Meteorology was controlled for using maximum temperature (IO temp) with indicators for each degree Celsius, a cubic function for dew point (dewpt), a cubic function for minimum temperature (temp), and dummy variables for seasons. When simulating the health effect we assume zero lag, but expect results to be generalizable to single day lag

PREVENTING CHRONIC DISEASE
PUBLIC HEALTH RESEARCH, PRACTICE, AND POLICY
Volume 11, E195 NOVEMBER 2014

ORIGINAL RESEARCH

Using Simulation to Compare Established and Emerging Interventions to Reduce Cardiovascular Disease Risk in the United States

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Table 1. Interventions, Target Populations, and Risk Factor Management Costs in the Prevention Impacts Simulation Model (PRISM)

PRISM Intervention Lever	Estimated Target Population 2010, millions	Recipient Population 2010, if Applicable, millions	Unit Cost per Recipient per Year, if Applicable, 2008 $	Initial Lever Setting	Best Plausible Lever Setting[a]
Tighter BP care: non-CVD, post-CVD	78.7[n]	0	88	0	100
Borderline cholesterol care: non-CVD, post-CVD	35.8[o]	0	378	0	100
Tighter cholesterol care: non-CVD, post-CVD	108.6[p]	0	126	0	100
Prediabetes care: non-CVD, post-CVD	73.3[q]	0	850	0	100
Tighter diabetes care: non-CVD, post-CVD	26.4[r]	0	850	0	100
Air established: 3 intervention levers					
Tobacco tax rate	253.4[s]	–	–	34	100
Tobacco marketing restriction index	253.4[s]	–	–	25	100
Fraction of workplaces allowing smoking	253.4[s]	–	–	11	0
Air emerging: 2 intervention levers					
Tobacco counter-marketing index	253.4[s]	–	–	20	100
Average small particulate air pollution (µg/cubic meter PM 2.5)	295.4[t]	–	–	10.9	7.0
Lifestyle established: 3 intervention levers					

Intervention Impacts on Deaths and Costs 2012 Through 2040

Outcome	% Change from Base Case[a] (95% Uncertainty Range)[b]			Base Case[a]
	Established	Emerging	Combined	
Care interventions[c]				
Death rate[d]	−35.6 (−40.6 to −31.0)	−17.2 (−18.2 to −16.3)	−47.7 (−51.9 to −45.7)	5.74
YPLL rate[e]	−34.8 (−39.3 to −30.5)	−15.5 (−16.5 to −14.6)	−45.9 (−49.7 to −44.1)	72.1
Risk management costs[f]	83.3 (76.5 to 89.9)	30.1 (23.8 to 38.2)	128.9 (114.0 to 143.3)	490
Acute and extended care costs[g]	−19.4 (−25.5 to −13.4)	−10.8 (−11.6 to −10.1)	−31.0 (−33.6 to −27.3)	539
Productivity costs[h]	−31.1 (−35.7 to −26.7)	−13.8 (−14.6 to −13.1)	−41.2 (−44.6 to −38.8)	1,769
Combined costs[i]	−8.8 (−13.5 to −4.6)	−5.5 (−6.9 to −4.0)	−9.4 (−14.6 to −6.1)	2,797
Air interventions[c]				
Death rate[d]	−3.5 (−4.2 to −2.8)	−2.3 (−2.8 to −1.9)	−5.6 (−6.3 to −4.8)	5.74
YPLL rate[e]	−3.2 (−3.8 to −2.5)	−2.0 (−2.5 to −1.7)	−5.0 (−5.6 to −4.2)	72.1
Risk management costs[f]	−1.7 (−2.0 to −1.2)	−0.5 (−0.6 to −0.4)	−2.1 (−2.5 to −1.7)	490
Acute and extended care costs[g]	−2.6 (−3.2 to −2.1)	−2.3 (−2.9 to −1.9)	−4.8 (−5.6 to −4.0)	539
Productivity costs[h]	−3.9 (−4.7 to −3.0)	−2.0 (−2.5 to −1.6)	−5.7 (−6.4 to −4.8)	1,769
Combined costs[i]	−3.3 (−4.0 to −2.6)	−1.8 (−2.2 to −1.5)	−4.9 (−5.6 to −4.1)	2,797
Lifestyle interventions[c]				
Death rate[d]	−1.5 (−2.4 to −1.7)	−9.7 (−11.8 to −7.6)	−10.8 (−12.8 to −8.5)	5.74
YPLL rate[e]	−1.5 (−2.4 to −1.7)	−8.6 (−10.4 to −6.7)	−9.8 (−11.5 to −7.7)	72.1
Risk management costs[f]	−1.0 (−1.6 to −0.4)	−7.1 (−8.1 to −5.8)	−7.9 (−8.9 to −6.5)	490
Acute and extended care costs[g]	−0.9 (−1.5 to −0.4)	−7.8 (−10.3 to −5.6)	−8.5 (−10.9 to −6.2)	539
Productivity costs[h]	−1.4 (−2.1 to −0.6)	−7.9 (−9.5 to −6.1)	−8.9 (−10.5 to −6.9)	1,769
Combined costs[i]	−1.2 (−1.9 to −0.5)	−7.7 (−9.4 to −6.0)	−8.6 (−10.2 to −6.7)	2,797
All 3 intervention areas[c]				
Death rate[d]	−37.7 (−42.4 to −33.2)	−25.0 (−26.7 to −23.1)	−51.3 (−54.1 to −48.7)	5.74

Level of Evidence and Frameworks for Simulations

	Model	Effects	Scope
Aggregate Data	Count or population levels	Population average	Public Policy (strategic level)
Individual Data	GLM logit-based Link	Population average	Etiological models
Micro Data	Bayesian endpoints for surrogate	Conditional models	Individual short term impact

Wish list

- Methods
  - Further improve in detailing pollution data (also via algorithms)
  - Define methods for handling correlated outcomes and exposures
- Research
  - Develop algorithms for translational research (from mice to humans, from surrogates to outcomes)
  - Develop validation criteria for simulation of health outcomes impacts of pollutants
- Policy
  - Easier micro-data linkage for surveillance purposes

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THANK YOU!

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