

# Training Course on Seamless Prediction of Air Pollution in Africa

## Health Impact Assessments

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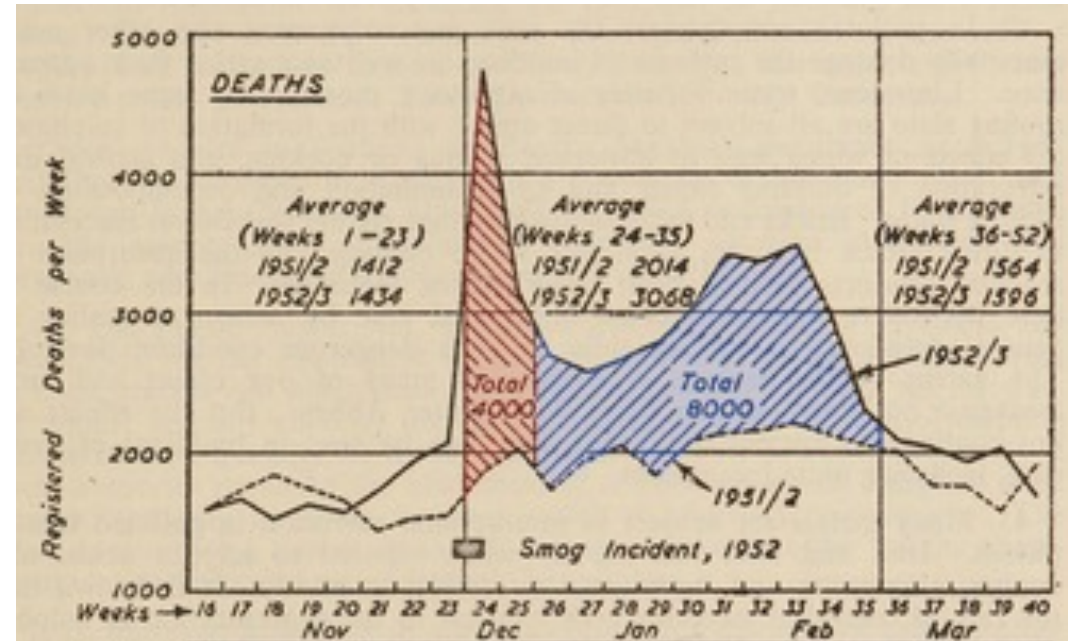
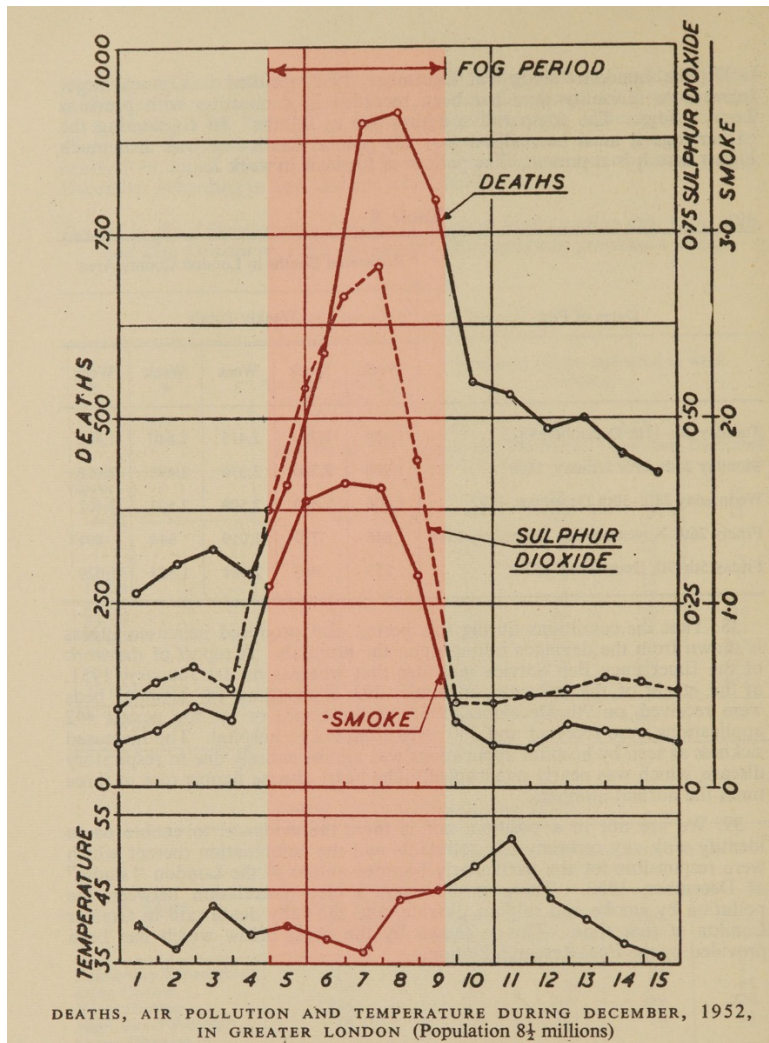
Sophie Gummy (*World Health Organization*)



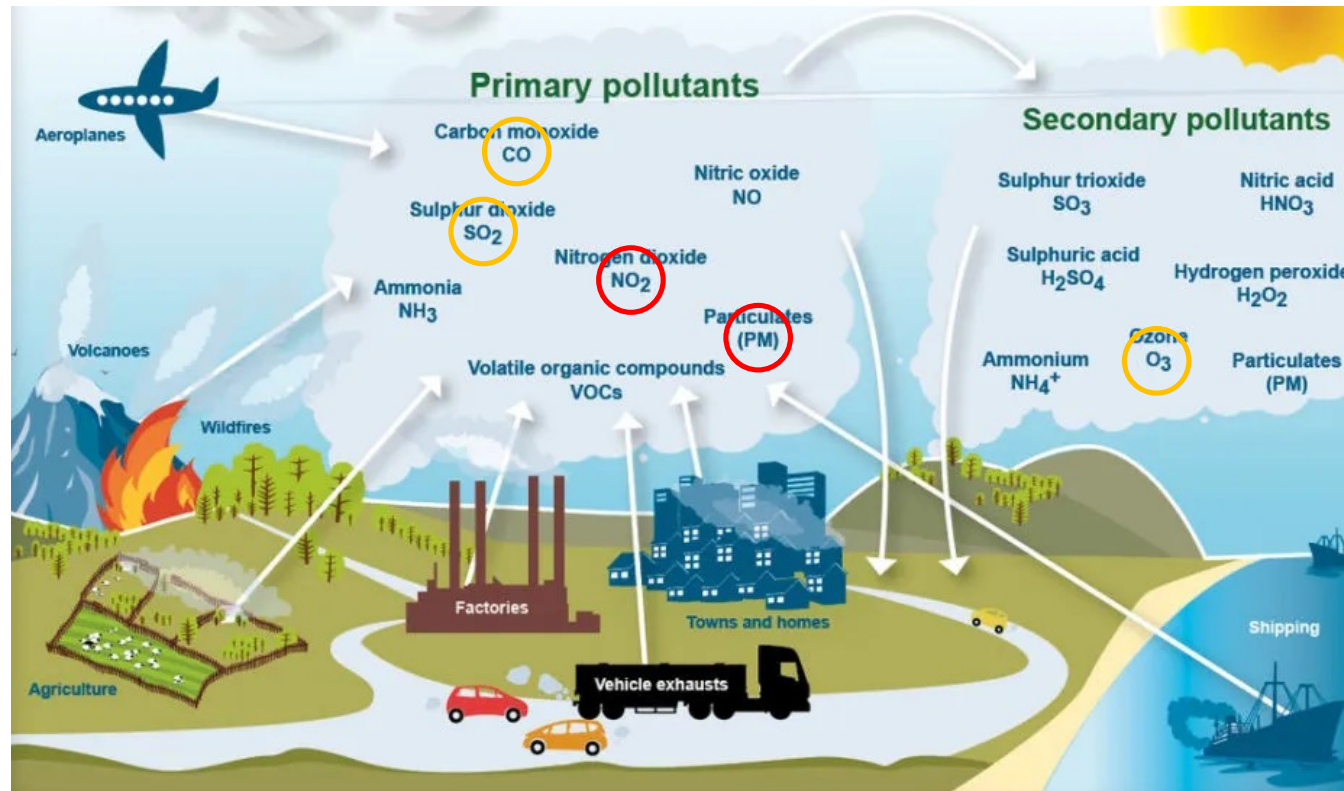
IMPLEMENTED BY EUMETSAT



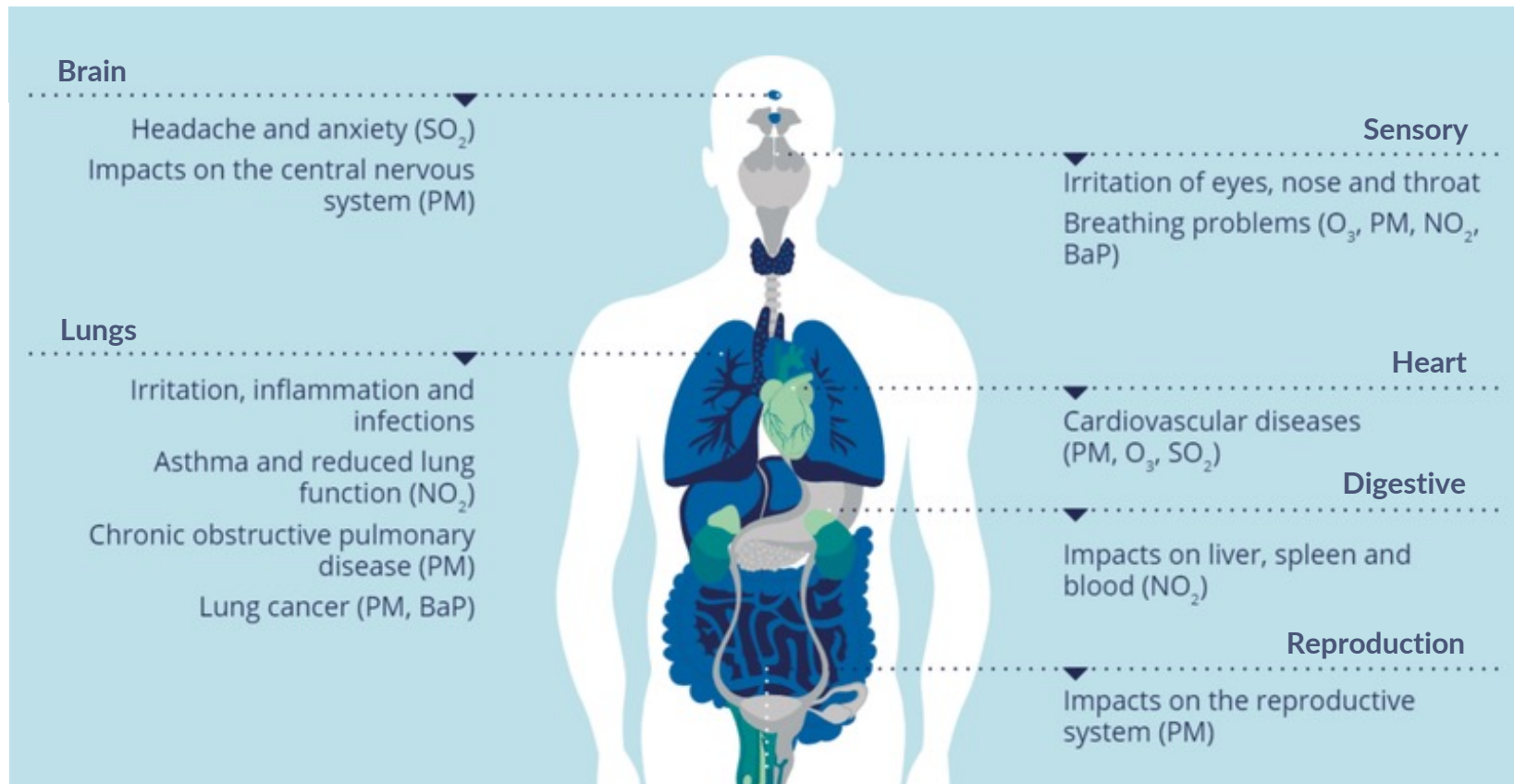
# Deadly smog in London, 1952



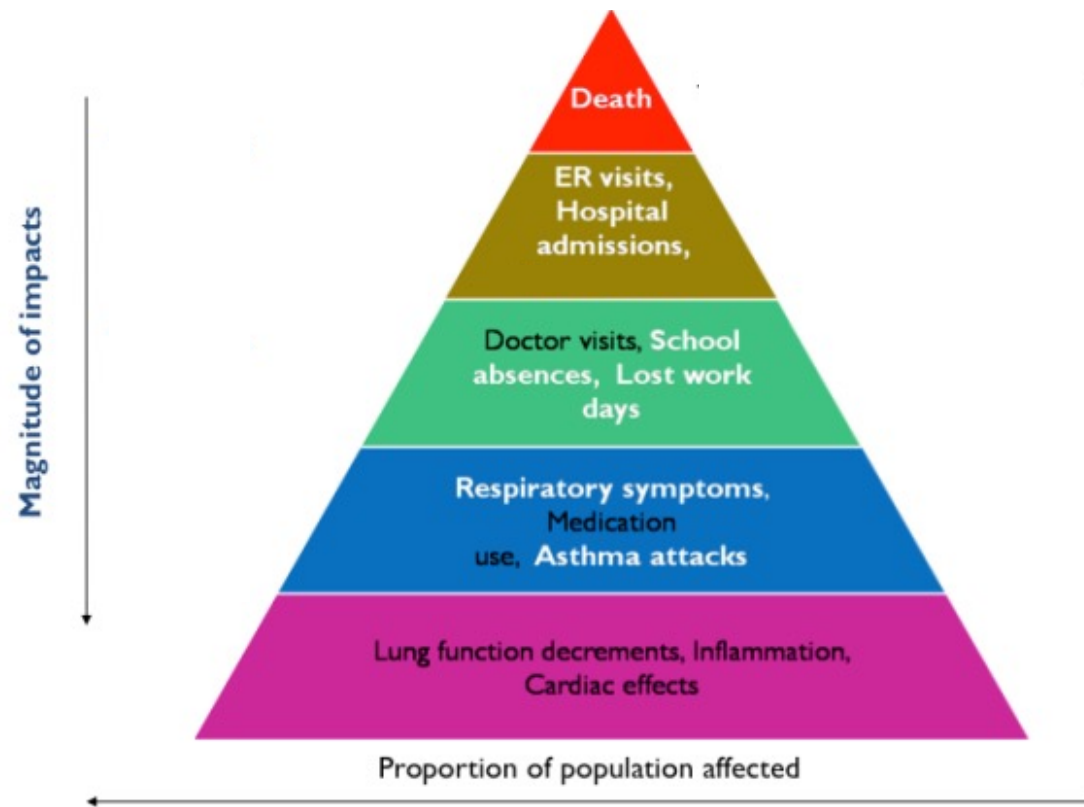
# Major air pollutants



# Health effects of air pollution



# Pyramid of health outcomes



# Regulatory Concentrations

**Table 0.1. Recommended AQG levels and interim targets**

Pollutant	Averaging time	Interim target				AQG level
		1	2	3	4	
<b>PM<sub>2.5</sub>, µg/m<sup>3</sup></b>	Annual	35	25	15	10	5
	24-hour <sup>a</sup>	75	50	37.5	25	15
<b>PM<sub>10</sub>, µg/m<sup>3</sup></b>	Annual	70	50	30	20	15
	24-hour <sup>a</sup>	150	100	75	50	45
<b>O<sub>3</sub>, µg/m<sup>3</sup></b>	Peak season <sup>b</sup>	100	70	–	–	60
	8-hour <sup>a</sup>	160	120	–	–	100
<b>NO<sub>2</sub>, µg/m<sup>3</sup></b>	Annual	40	30	20	–	10
	24-hour <sup>a</sup>	120	50	–	–	25
<b>SO<sub>2</sub>, µg/m<sup>3</sup></b>	24-hour <sup>a</sup>	125	50	–	–	40
<b>CO, mg/m<sup>3</sup></b>	24-hour <sup>a</sup>	7	–	–	–	4

<sup>a</sup> 99th percentile (i.e. 3–4 exceedance days per year).

<sup>b</sup> Average of daily maximum 8-hour mean O<sub>3</sub> concentration in the six consecutive months with the highest six-month running-average O<sub>3</sub> concentration.



# Assessing health effects and impacts

- **Health effects** refers to changes in health status caused by an exposure
  - Short-term effects account for acute impact on health after an immediate exposure (**time-series studies**)
  - Long-term effects involve chronic health effect after a cumulative exposure (**cohort studies**)



- **Health impact assessment** evaluates potential health effects of proposed actions relative to an exposure, to provide advice for decision-making process that will protect health

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**Short-term health effects**

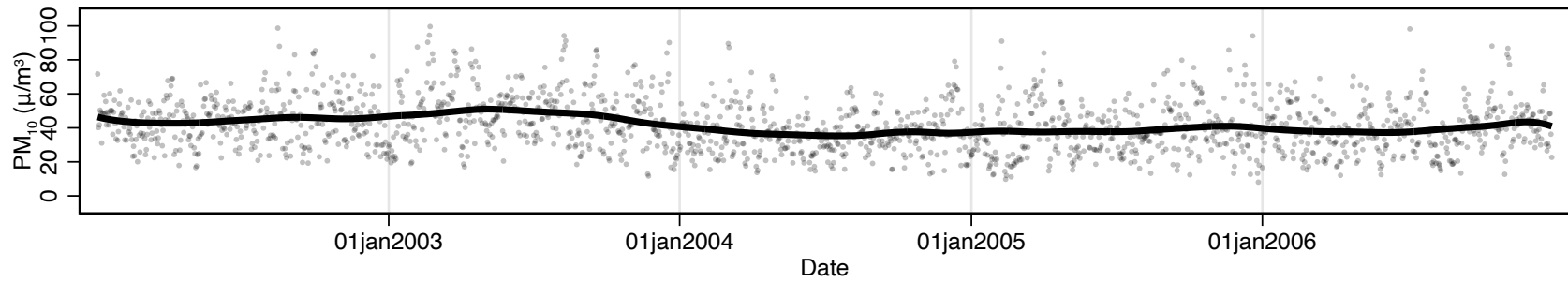
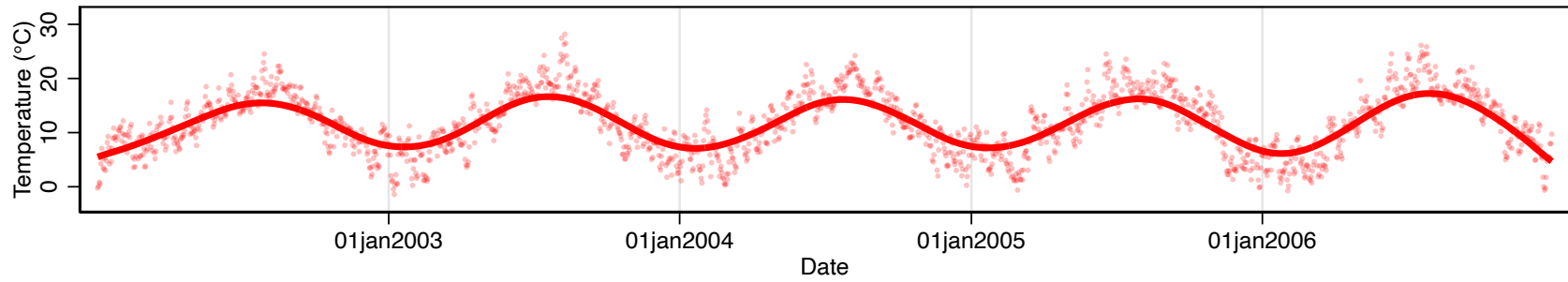
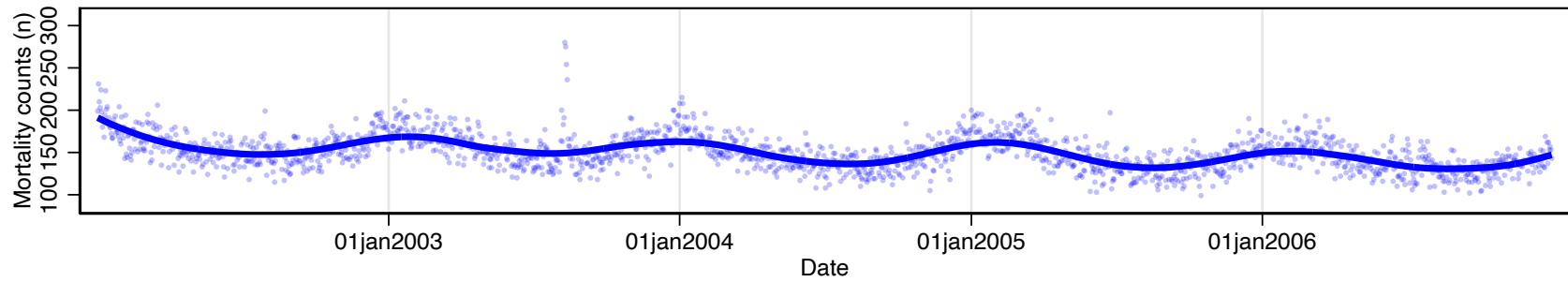
Aurelio Tobías (*Spanish Research Council*)



# Introduction

- **Research question** – *“Is there an association between day-to-day variation in the environmental exposure and daily risk of health outcome”?*
- Health outcomes and environmental exposures are characterized by **similar time-trends**
- Measures of **individual predictors** are usually not available
- Need of a study design relying on **between-day comparison within the same population** and able to control for time-trends to disentangle short-term health effects of air pollution

# London (2002-2006)



# Time-series data

- A time-series is a sequence of **measurements equally spaced** through time
- The **unit of analysis is the day**, not the individual person
- The **health outcome is a count** (e.g., number of deaths)

- First week of time-series data in London (Jan 2002 – Dec 2006)

obs	date	deaths	temp	pm10
1.	01jan2002	199	-0.2	71.7
2.	02jan2002	231	0.1	40.2
3.	03jan2002	210	0.9	41.8
4.	04jan2002	203	0.5	50.4
5.	05jan2002	224	4.2	49.4
6.	06jan2002	198	7.1	31.1
7.	07jan2002	180	5.2	48.6

# Time-series design

- **Strengths**

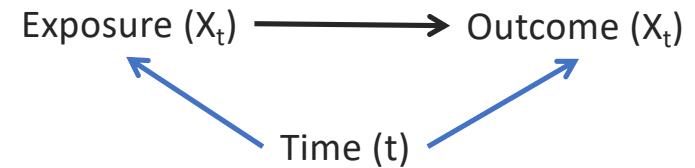
- Use of **administratively collected data**
- Same population is compared with itself, focus is **day-to-day variation**
- Time-invariant or slowly varying individual risk factors **controlled by design** (e.g., sex, age, smoking)

- **Limitations**

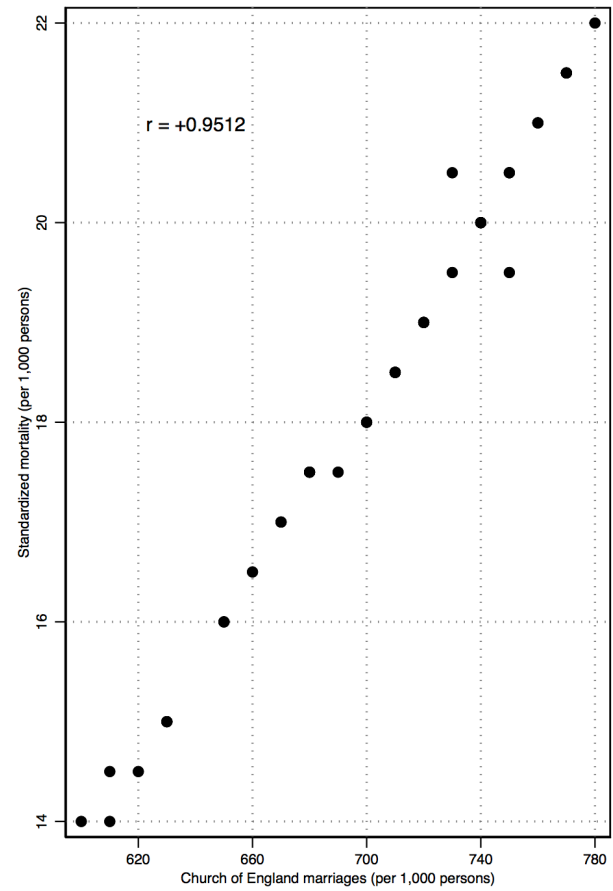
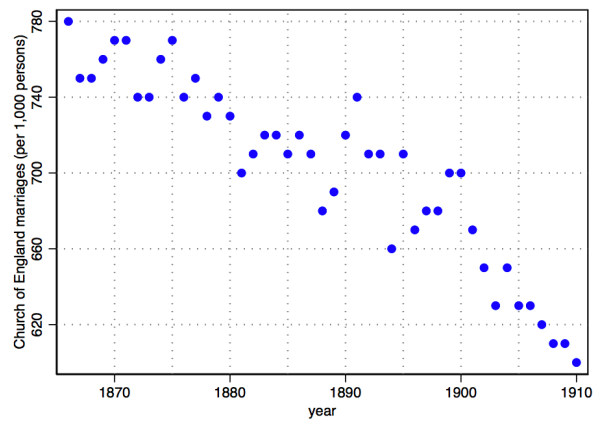
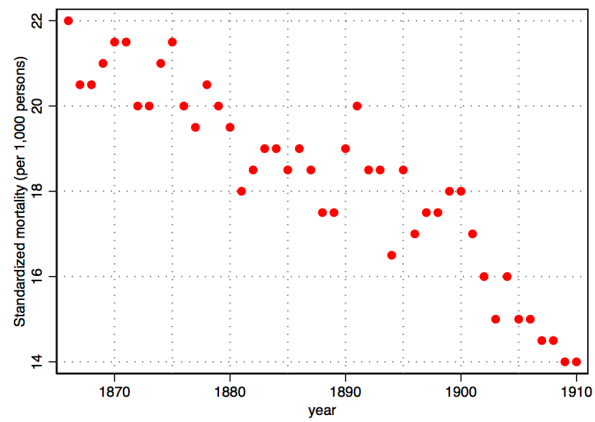
- Ecological design based on aggregated, not individual data
- Not applicable to estimate chronic effects (long-term)
- Sensitive to choices for statistical modelling

# Confounding

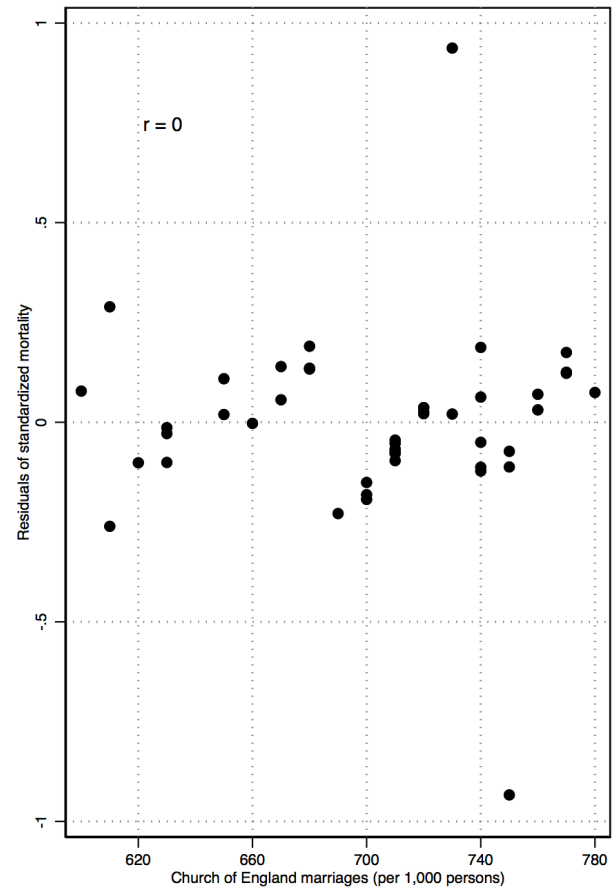
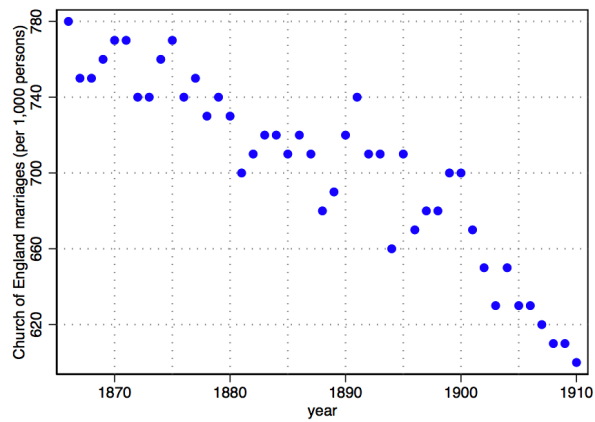
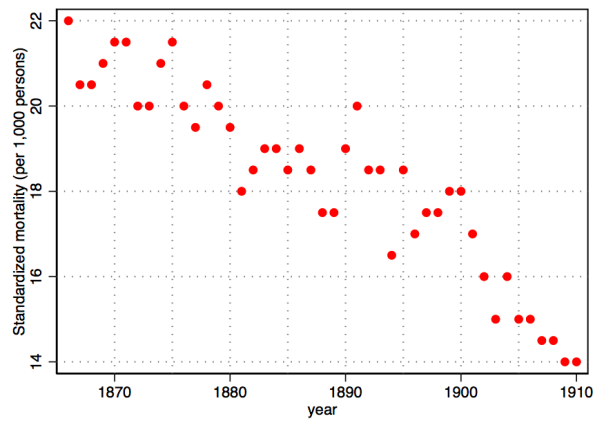
- It must be associated with the exposure (X) being investigated
- It must be independently associated with the outcome (Y) being investigated
- It must not be on the causal pathway between exposure (X) and outcome (Y)



Yule GU. Why do we sometimes get nonsense correlations between time series? J Royal Stat Soc Sci. 1926;89:1-64.

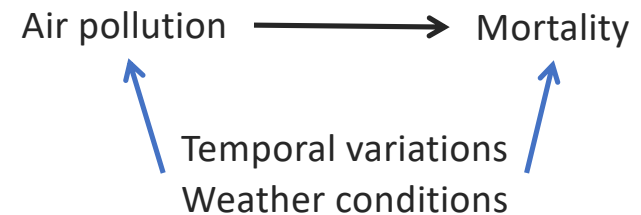


Yule GU. Why do we sometimes get nonsense correlations between time series? J Royal Stat Soc Sci. 1926;89:1-64.



# Confounding

- It must be associated with the exposure (X) being investigated
- It must be independently associated with the outcome (Y) being investigated
- It must not be on the causal pathway between exposure (X) and outcome (Y)





# Modelling framework

- Similar in principle to any regression analysis but with **some specific features**

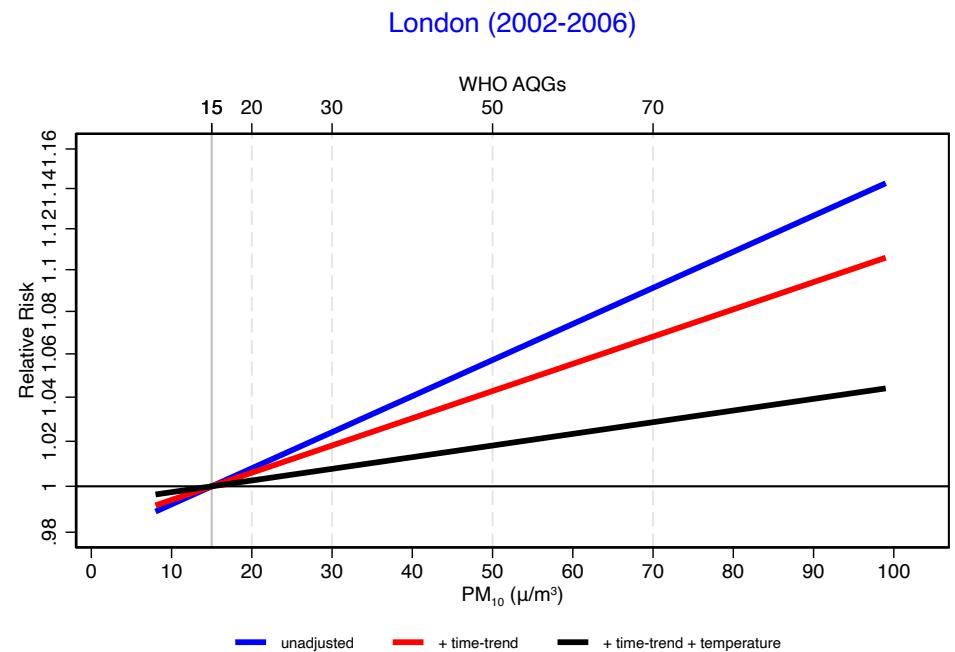
- Poisson regression

$$\log(Y_t) = \beta_0 + \beta_1 PM_{10t} + ns(t) + ns(temp)$$

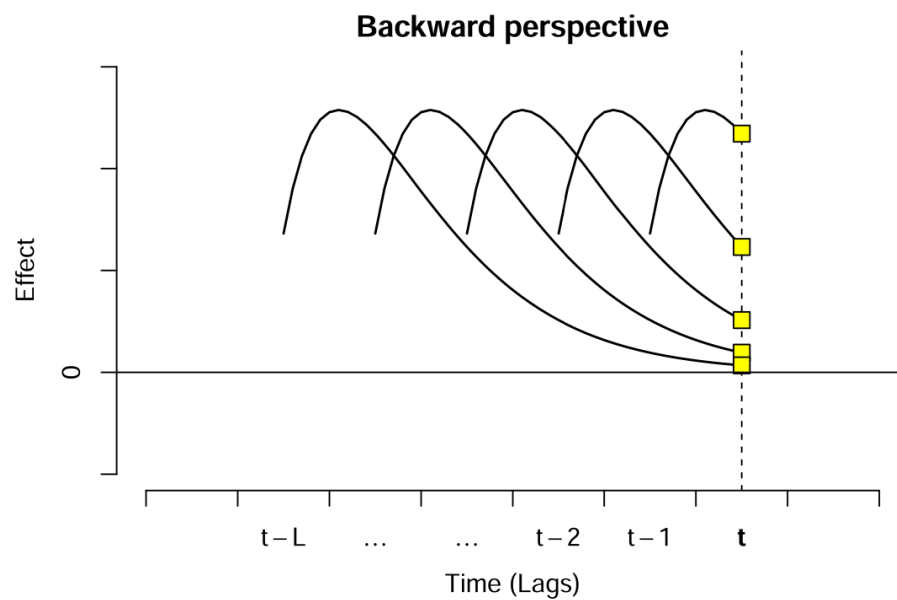
$$Y|x \sim \text{Poisson}(\mu)$$

$$\text{with } E(Y|x) = \mu \text{ and } V(Y|x) = \mu$$

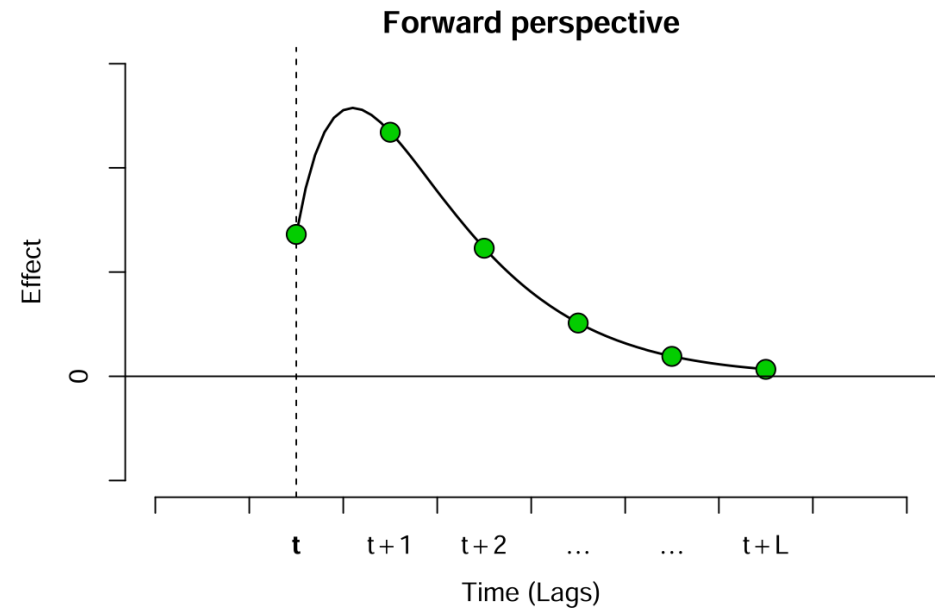
- $\exp(\beta_1)$  is the **relative risk (RR)** for 1 unit increase of the  $PM_{10}$  exposure
- $(RR - 1) \times 100\%$  is the **percentage risk increase**



# Lag effects



- Cumulative effect at time  $t$  for an exposure in the previous  $t-L$  days



- Cumulative effect for an exposure at day  $t$  experienced in the next  $t+L$  days

# Lag variables

- In practice, time-series data is usually analysed with a **forward perspective**
- It requires generating **lagged exposure variables** to be fitted in the time-series regression model
- Poisson regression

$$\log(Y_t) = \beta_0 + \sum \beta_j PM_{10t-j} + ns(t) + ns(temp)$$

$$Y|x \sim \text{Poisson}(\mu)$$

$$\text{with } E(Y|x) = \mu \text{ and } V(Y|x) = \mu$$

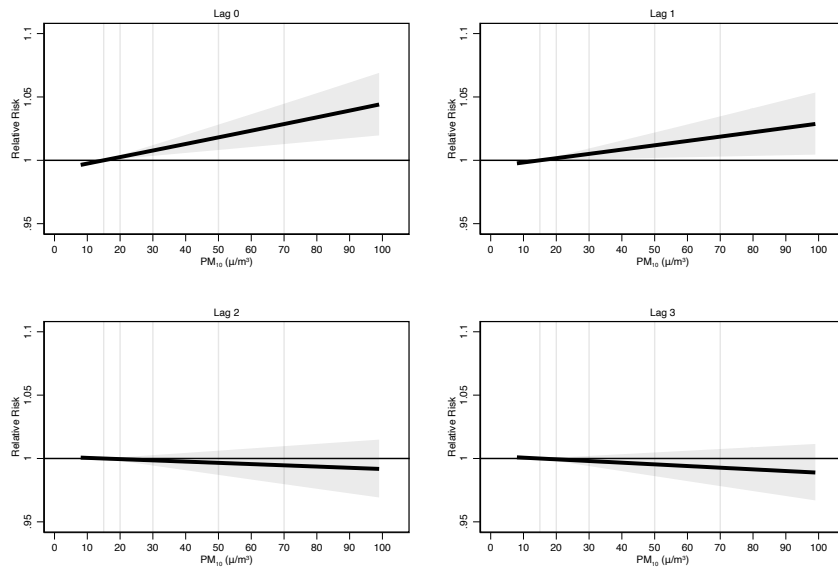
- $\exp(\beta_j)$  is the **relative risk (RR)** for 1 unit increase of the  $PM_{10}$  exposure at *lag j*

- First week of time-series data in London (Jan 2002 – Dec 2006)

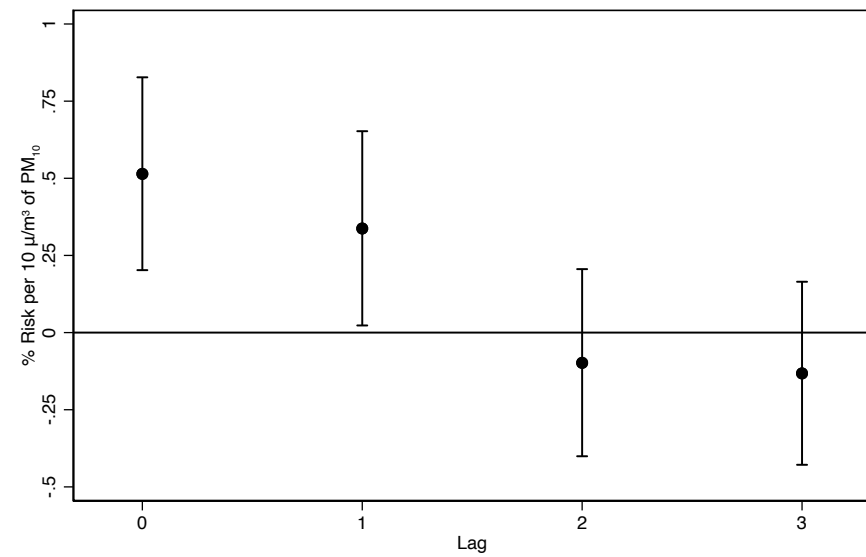
obs	date	deaths	pm10	lag1. pm10	lag2. pm10
1.	01jan2002	199	71.7	.	.
2.	02jan2002	231	40.2	71.7	.
3.	03jan2002	210	41.8	40.2	71.7
4.	04jan2002	203	50.4	41.8	40.2
5.	05jan2002	224	49.4	50.4	41.8
6.	06jan2002	198	31.1	49.4	50.4
7.	07jan2002	180	48.6	31.1	49.4

# Distributed lags

London (2002-2006)



London (2002-2006)



# Multi-location studies

**Table 1. Percentage Change in All-Cause Mortality per 10- $\mu\text{g}$ -per-Cubic-Meter Increase in 2-Day Moving Average Concentrations of Inhalable Particulate Matter (PM<sub>10</sub>) and Fine Particulate Matter (PM<sub>2.5</sub>).\***

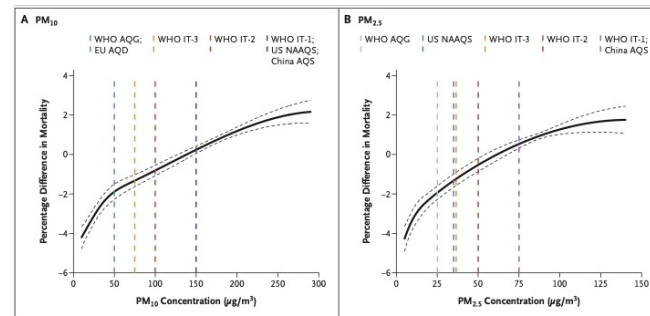
Country or Region	PM <sub>10</sub>		PM <sub>2.5</sub>	
	Cities with Available Data	Pooled Estimate	Cities with Available Data	Pooled Estimate
	no.	% (95% CI)	no.	% (95% CI)
Australia	3	1.32 (0.22 to 2.44)	3	1.42 (-0.12 to 2.99)
Brazil	1	1.22 (0.97 to 1.47)	0	NA
Canada	13	0.76 (0.25 to 1.27)	25	1.70 (1.17 to 2.23)
Chile	4	0.33 (0.14 to 0.53)	4	0.27 (-0.68 to 1.23)
China	272	0.28 (0.22 to 0.34)	272	0.41 (0.32 to 0.50)
Colombia	1	0.03 (-0.34 to 0.39)	0	NA
Czech Republic	1	0.40 (-0.02 to 0.82)	0	NA
Estonia	4	0.46 (-0.69 to 1.63)	3	0.23 (-4.24 to 4.90)
Finland	1	0.07 (-0.51 to 0.65)	1	0.14 (-0.55 to 0.83)
France	18	0.46 (-0.15 to 1.07)	0	NA
Greece	1	0.53 (0.17 to 0.90)	1	2.54 (1.28 to 3.83)
Italy	18	0.65 (0.26 to 1.04)	0	NA
Japan	47	1.05 (0.78 to 1.31)	47	1.42 (1.05 to 1.81)
Mexico	8	0.67 (0.48 to 0.86)	3	1.29 (0.21 to 2.39)
Portugal	2	0.11 (-0.27 to 0.49)	1	0.03 (-1.14 to 1.21)
South Africa	6	0.41 (0.14 to 0.68)	5	0.80 (0.16 to 1.44)
South Korea	7	0.42 (0.27 to 0.58)	0	NA
Spain	45	0.87 (0.60 to 1.15)	19	1.96 (1.18 to 2.75)
Sweden	1	0.20 (-1.03 to 1.44)	1	0.08 (-1.44 to 1.62)
Switzerland	8	0.47 (-0.36 to 1.31)	4	0.79 (-0.96 to 2.58)
Taiwan	3	0.25 (-0.03 to 0.53)	3	0.62 (-0.39 to 1.64)
Thailand	19	0.61 (0.24 to 0.99)	0	NA
United Kingdom	15	0.06 (-0.36 to 0.48)	0	NA
United States	100	0.79 (0.60 to 0.98)	107	1.58 (1.28 to 1.88)
Total	598	0.44 (0.39 to 0.50)	499	0.68 (0.59 to 0.77)

\* Pooled estimates represent the percentage changes in daily all-cause mortality per 10- $\mu\text{g}$ -per-cubic-meter increase in concentrations of particulate matter (PM) with an aerodynamic diameter of 10  $\mu\text{m}$  or less (PM<sub>10</sub>) and PM with an aerodynamic diameter of 2.5  $\mu\text{m}$  or less (PM<sub>2.5</sub>), as determined with the use of trimmed exposure data in which the highest 5% and lowest 5% of PM<sub>10</sub> and PM<sub>2.5</sub> measurements were excluded. NA denotes not available.



## Ambient Particulate Air Pollution and Daily Mortality in 652 Cities

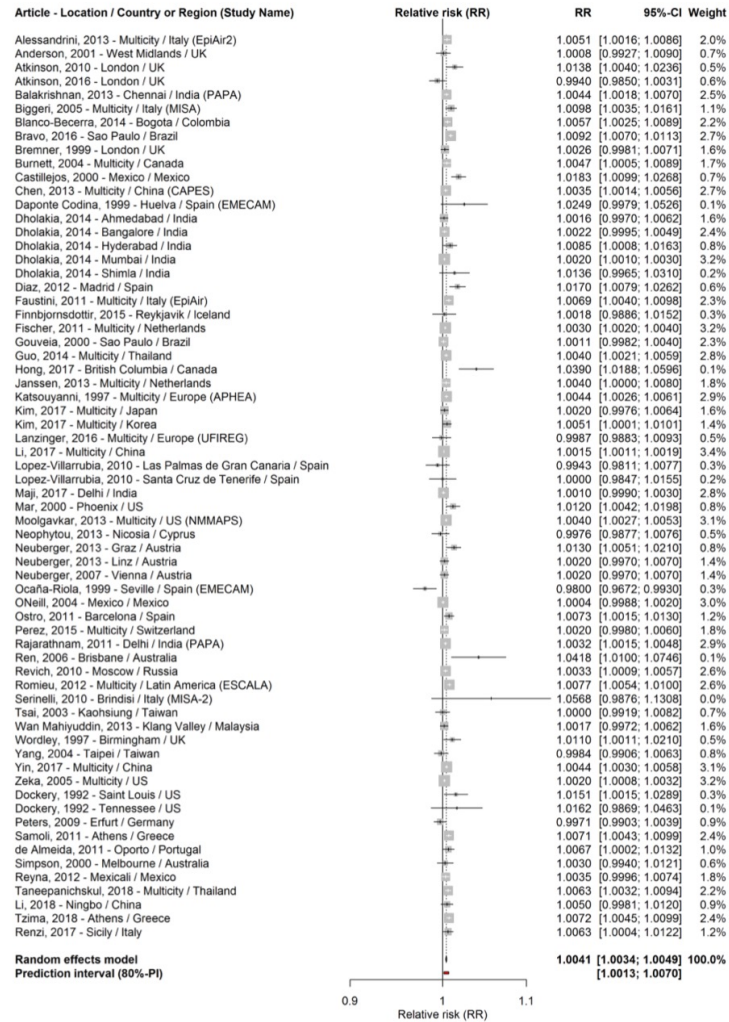
C. Liu, R. Chen, F. Sera, A.M. Vicedo-Cabrera, Y. Guo, S. Tong, M.S.Z.S. Coelho, P.H.N. Saldiva, E. Lavigne, P. Matus, N. Valdes Ortega, S. Osorio Garcia, M. Pascal, M. Stafoggia, M. Scortichini, M. Hashizume, Y. Honda, M. Hurtado-Diaz, J. Cruz, B. Nunes, J.P. Teixeira, H. Kim, A. Tobias, C. Iniguez, B. Forsberg, C. Åström, M.S. Ragetti, Y.-L. Guo, B.-Y. Chen, M.L. Bell, C.Y. Wright, N. Scovronick, R.M. Garland, A. Milojevic, J. Kysely, A. Urban, H. Orru, E. Indermitte, J.J.K. Jaakkola, N.R.I. Rytli, K. Katsouyanni, A. Analitis, A. Zanobetti, J. Schwartz, J. Chen, T. Wu, A. Cohen, A. Gasparriani, and H. Kan



**Figure 3. Pooled Concentration-Response Curves.**

Shown are the pooled concentration-response curves for the associations of 2-day moving average concentrations of PM<sub>10</sub> (Panel A) and PM<sub>2.5</sub> (Panel B) with daily all-cause mortality. The y axis represents the percentage difference from the pooled mean effect (as derived from the entire range of PM concentrations at each location) on mortality. Zero on the y axis represents the pooled mean effect, and the portion of the curve below zero denotes a smaller estimate than the mean effect. The dashed lines represent the air-quality guidelines or standards for 24-hour average concentrations of PM<sub>10</sub> or PM<sub>2.5</sub> according to the World Health Organization Air Quality Guidelines (WHO AQG), WHO Interim Target 1 (IT-1), WHO Interim Target 2 (IT-2), WHO Interim Target 3 (IT-3), European Union Air Quality Directive (EU AQD), U.S. National Ambient Air Quality Standard (NAAQS), and China Air Quality Standard (AQG).

**Figure A.1. Forest plot of 58 studies (66 effect sizes) examining the association between PM<sub>10</sub> and all-cause mortality.**



# Literature review

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Review article

## Short-term exposure to particulate matter (PM<sub>10</sub> and PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>), and ozone (O<sub>3</sub>) and all-cause and cause-specific mortality: Systematic review and meta-analysis

Pablo Orellano<sup>a,\*</sup>, Julieta Reynoso<sup>b</sup>, Nancy Quaranta<sup>c,d</sup>, Ariel Bardach<sup>e</sup>, Agustín Ciapponi<sup>e</sup>

**Table 1**  
Exposures, outcomes and pooled effect sizes.

Pollutant	Outcome	Number of effect sizes	RR (95% CI)	p-value	PI	Egger's test (p-value)
PM <sub>10</sub>	All-cause mortality	66	1.0041 (1.0034–1.0049)	< 0.0001	1.0013–1.0070	< 0.001
PM <sub>10</sub>	Cardiovascular mortality	44	1.0060 (1.0044–1.0077)	< 0.0001	1.0016–1.0105	0.024
PM <sub>10</sub>	Respiratory mortality	41	1.0091 (1.0063–1.0119)	< 0.0001	1.0017–1.0166	0.209
PM <sub>10</sub>	Cerebrovascular mortality	20	1.0044 (1.0022–1.0066)	0.0005	1.0001–1.0087	< 0.001
PM <sub>2.5</sub>	All-cause mortality	29	1.0065 (1.0044–1.0086)	< 0.0001	1.0017–1.0114	0.015
PM <sub>2.5</sub>	Cardiovascular mortality	28	1.0092 (1.0061–1.0123)	< 0.0001	1.0026–1.0158	0.803
PM <sub>2.5</sub>	Respiratory mortality	20	1.0073 (1.0029–1.0116)	0.0023	0.9998–1.0148	0.606
PM <sub>2.5</sub>	Cerebrovascular mortality	7	1.0072 (1.0012–1.0132)	0.0257	0.9953–1.0192	N/A
NO <sub>2</sub> (24-hour average)	All-cause mortality	54	1.0072 (1.0059–1.0085)	< 0.0001	1.0031–1.0113	0.048
NO <sub>2</sub> (1-hour max.)	All-cause mortality	10	1.0024 (0.9995–1.0053)	0.0892	0.9985–1.0064	0.154
O <sub>3</sub>	All-cause mortality	48	1.0043 (1.0034–1.0052)	< 0.0001	1.0013–1.0073	0.001

RR, pooled relative risks; 95% CI, 95% confidence interval; p-value, significance of the association or statistical tests; PI, 80% prediction interval; N/A, not applicable (< 10 studies).

# Summary

- The time-series design is **useful to provide evidence on short-term associations** between air pollution and health outcomes
- Time-series regression is **similar in principle to any regression analysis** but with some specific features
- The design **accounts for temporal variations** (e.g., seasonal changes, day-of-week effects) **and weather conditions** (e.g., temperature) that may influence both air pollution and health outcome
- Time-series studies can **investigate lagged effects**, identifying whether health outcomes occur immediately or with some delay following exposure to air pollution