

The burden of air pollution on years of life lost in Beijing, China, 2004-08: retrospective regression analysis of daily deaths

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STUDY QUESTION

Does air pollution increase years of life lost in Beijing, China?

SUMMARY ANSWER

In terms of years of life lost, which accounts for a person's life expectancy at death, people aged up to 65 years were more affected by air pollutants than those older than 65 years; the risk of death related to air pollutants was higher for people older than 65 years than those aged up to 65 years.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS

Air pollution increases the risk of death, and is a serious problem in Beijing, China. This study also measures years of life lost—a complementary measure for examining the effect of air pollutants on mortality—and the study's findings highlight the need to reduce air pollution in Beijing to protect the health of the population.

Participants and setting

80 515 deaths were recorded by the Beijing Death Classification System during 2004-08, including 48 802 male deaths and 31 713 female deaths. The study was conducted in eight urban districts in Beijing, China.

Design

We did a retrospective regression analysis using a daily time series to examine the effects of air pollutants on years of life lost. We performed sensitivity analyses for associations between air pollution and years of life lost to check the robustness of our models.

Primary outcome(s)

Associations between daily years of life lost and ambient air pollutants (particulate matter with aerodynamic diameter <2.5 µm (PM_{2.5}), PM₁₀, SO₂, and NO₂), after adjusting for long term trends, seasonality, day of the week, and weather conditions. We also examined mortality risk related to air pollutants.

Main results

Mean concentrations of daily PM_{2.5}, PM₁₀, SO₂, and NO₂ were 105.1 µg/m³, 144.6 µg/m³, 48.6 µg/m³, and 64.2 µg/m³ respectively. Years of life lost was higher for men

than women (mean 420.0 years (standard deviation 110.7) v 289.2 (91.8)) and higher for people aged up to 65 years than those older than 65 years (416.4 (127.2) v 292.8 (73.4)). The effects of air pollutants on years of life lost appeared acutely. The associations between air pollutants and years of life lost differed by sex and age groups, according to increased interquartile ranges of pollutants (94 µg/m³ for PM_{2.5}, 106 µg/m³ for PM₁₀, 49 µg/m³ for SO₂, and 30 µg/m³ for NO₂). Effect estimates of PM_{2.5} and PM₁₀ on years of life lost were higher in women than men (11.1 v 4.7 and 9.3 v 6.5 years, respectively). The effect estimates of air pollutants on years of life lost among people aged up to 65 years were significant and about twice those of people older than 65 years. For example, an interquartile range increase in PM_{2.5} was associated with 12.0 years of life lost in people aged up to 65 years and 3.8 years of life lost in those older than 65 years. The risk of death associated with an interquartile range increase in PM_{2.5} was greater for people older than 65 years (2.5% increase of mortality) than those aged up to 65 years (0.7%). All sensitivity analyses confirmed our approaches to be valid.

Bias, confounding, and other reasons for caution

We used ambient pollutant concentrations as surrogates of individual exposure, which could result in measurement error. We did not control for smoking or for prevalence of chronic obstructive pulmonary disease related to the smoking, because this information was unavailable.

Generalisability to other populations

Because data were obtained from only one city, it is difficult to generalise results to other populations.

Study funding/potential competing interests

All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf and declare: support from the National Natural Science Foundation of China and the Australia National Health and Medical Research Council for the submitted work; YG is supported by the Centre for Air Quality and Health Research and Evaluation and the University of Queensland School of Population Health; no other relationships or activities that could appear to have influenced the submitted work.

Association between increase in air pollutants (lag 0-1 day) and years of life lost using single pollutant models, according to subgroups

Pollutant	Years of life lost (mean, 95% CI)			
	Female sex	Male sex	Age ≤65 years	Age >65 years
PM _{2.5}	11.1 (4.7 to 17.5)†	4.7 (-2.9 to 12.3)	12.0 (2.9 to 21)†	3.8 (-0.9 to 8.6)
PM ₁₀	9.3 (3.3 to 15.2)†	6.5 (-0.5 to 13.5)	10.3 (2 to 18.6)†	5.5 (1.1 to 9.9)*
SO ₂	5.6 (-1.9 to 13.1)	10.6 (1.8 to 19.4)*	10.8 (0.3 to 21.3)	5.4 (-0.1 to 10.9)
NO ₂	6.7 (0.3 to 13.1)*	8.4 (0.8 to 16.0)*	10.1 (1.1 to 19.1)*	5.0 (0.3 to 9.8)

*P<0.05.

†P<0.01.

Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE Project

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STUDY QUESTION

Is long term exposure to air pollution associated with incidence of acute coronary events?

SUMMARY ANSWER

There is an association between long term exposure to particulate matter and incidence of coronary events, even for exposure concentrations below the current European air quality limits.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS

The association between long term exposure to air pollution and incidence of coronary events remains controversial. In this large and geographically widespread study there was an association between outdoor residential particulate matter level and incidence of acute coronary events, the association was strong even for exposure levels below the current European limits.

Participants and setting

The study was conducted with data from 11 European cohorts from Finland, Sweden, Denmark, Germany, and Italy participating in the ESCAPE (European Study of Cohorts for Air Pollution Effects) project. Participants who were free from previous coronary events were enrolled from 1997 to 2007.

Design, size, and duration

100 166 participants were followed for an average of 11.5 years. Modelled concentrations at the home address for particulate matter <2.5 µm (PM_{2.5}), 2.5-10 µm (PM_{coarse}), and <10 µm (PM₁₀) in aerodynamic diameter, soot (PM_{2.5} absorbance), nitrogen oxides, and traffic exposure were based on 2008-12 measurements. Identification of first coronary events (myocardial infarction and unstable

angina) was accomplished by interview, inspection of medical records, and death certificates or by record linkage with mortality and hospital discharges registries. We used Cox proportional hazard models to study the incidence of acute coronary events per fixed increments of the pollutants with adjustment for sociodemographic and lifestyle risk factors, and we pooled the results using random effects meta-analysis.

Main results and the role of chance

A total of 5157 participants experienced an incident event. A 5 µg/m³ increase in estimated annual mean PM_{2.5} was associated with a 13% increased risk of coronary events (hazard ratio 1.13, 95% confidence interval 0.98 to 1.30), and a 10 µg/m³ increase in estimated annual mean PM₁₀ was associated with a 12% increased risk of coronary events (1.12, 1.01 to 1.25) with no evidence of heterogeneity across the cohorts. Positive associations were detected below the current annual European limit of 25 µg/m³ for PM_{2.5} (1.18 (1.01 to 1.39) for 5 µg/m³ increase in PM_{2.5}) and below 40 µg/m³ for PM₁₀ (1.12 (1.00 to 1.27) for 10 µg/m³ increase in PM₁₀). We found positive but non-significant associations with other pollutants. Our findings were robust to multiple sensitivity analyses.[t1]

Bias, confounding, and other reasons for caution

We accounted for confounders such as sociodemographic factors (level of education, occupational status, marital status, area based socioeconomic position) and lifestyle risk factors (smoking, BMI, alcohol consumption). In sensitivity analyses, we considered exposure to noise and cholesterol concentration. Misclassification of exposure could be possible as we found higher association between the pollutants and the outcome when we restricted the analysis to studies with better performance of exposure models. For nine out of 11 cohorts the outcome was derived by record linkage procedures with hospital records and some underestimation of the cases could have occurred.

Generalisability to other populations

The cohorts were not a representative sample of the European population, though they included participants with a wide range of latitude, age, exposure levels, and confounders.

Study funding/potential competing interests

The research leading to these results has received funding from the European Community's Seventh Framework Program (FP7/2007-2011) under grant agreement No 211250. A full list of the study authors can be found with the unabridged article on bmj.com

Adjusted association between incidence of coronary events and exposure to PM₁₀ and PM_{2.5} below various threshold values. Figures are pooled hazard ratios (95% CI) for 10 µg/m³ PM₁₀ and 5 µg/m³ PM_{2.5}, I² statistic, and P value for heterogeneity

	No of cohorts included	No of participants included	Hazard ratios* (95% CI)	I ²	P value
PM₁₀ (µg/m³)					
Overall	11	100 166	1.12 (1.01 to 1.25)	0	0.81
<40	9	86 222	1.12 (1.00 to 1.27)	0	0.48
<30	8	77 997	1.12 (0.98 to 1.27)	0	0.94
<20	7	64 363	1.20 (1.01 to 1.41)	0	0.91
PM_{2.5} (µg/m³)					
Overall	11	100 166	1.13 (0.98 to 1.30)	0	0.60
<25	9	87 532	1.18 (1.01 to 1.39)	0	0.56
<20	9	85 216	1.17 (0.91 to 1.50)	30	0.18
<15	7	73 420	1.19 (1.00 to 1.42)	0	0.74

PM=particulate matter.

*Adjusted for age (time variable), year of enrolment, sex, marital status, education, occupation, smoking status, smoking duration, smoking intensity, and socioeconomic area level indicators.

Home energy efficiency and radon related risk of lung cancer: modelling study

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STUDY QUESTION

What are the effects on radon related lung cancer mortality of reducing home ventilation as part of household energy efficiency measures?

SUMMARY ANSWER

Unless specific remediation is used, reducing the ventilation of dwellings will improve energy efficiency only at the expense of population wide adverse impact on indoor radon levels and risk of lung cancer.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS

Radon is the second most important risk factor for lung cancer after smoking. Proposed strategies for reducing greenhouse gas emissions from the housing sector are likely to increase indoor radon levels and associated lung cancer risk substantially.

Participants and setting

Housing stock and population of England.

Design, size, and duration

Using data from the English housing survey, we modelled changes to the distribution of indoor radon levels for the English housing stock resulting from home energy efficiency interventions that entail reduction in uncontrolled ventilation. We estimated the associated impact on lung cancer mortality in the population using life tables, with exposure-response functions derived from the published literature. We also estimated savings in energy for ventilation related space heating and greenhouse gas emissions.

Main results and the role of chance

Increasing the air tightness of dwellings (without compensatory purpose-provided ventilation) was estimated to increase mean indoor radon concentrations in England by 56.6%, from 21.2 becquerels per cubic metre (Bq/m³) to 33.2 Bq/m³. After the lag in lung cancer onset, this would result in an additional annual health burden of 4700 life

years lost and (at peak) 278 deaths. The increases in radon levels for the millions of homes that would contribute most of this additional burden are below the threshold at which radon remediation measures are cost effective. Fitting extraction fans and trickle ventilators to restore ventilation will help offset the additional burden only if the ventilation related energy efficiency gains are lost. Mechanical ventilation systems with heat recovery may lower radon levels and the risk of lung cancer while maintaining the advantage of energy efficiency for the most airtight dwellings but there is potential for significant adverse health impact, especially if such systems fail. A more careful re-evaluation of how retrofitting of dwellings is carried out is needed to ensure that the potential benefits are not compromised by injudicious air tightening.

Bias, confounding, and other reasons for caution

Modelling such a complex system is challenging and depends on a range of assumptions, in particular about the behaviour of people in their homes. While there are uncertainties, our model is almost certainly correct about the general direction of change. We assumed lower future lung cancer risk because of recent decreases in smoking prevalence but examined different assumptions about future smoking prevalence. This showed that substantial future decreases in smoking could help to ameliorate the adverse impact of increased radon levels.

Generalisability to other populations

Similar greenhouse gas mitigation strategies relating to the housing sector are currently being applied or proposed in many other high income countries. The general lessons of this study are likely to also apply to those populations.

Study funding/potential competing interests

This work is part of the project PURGE, a three and a half year project funded by the European Commission's seventh framework programme (grant agreement No 265325).

Radon exposures, health impacts, and changes in ventilation related heating demand for selected home energy efficiency scenarios for English housing stock

Scenarios	Mean radon level (Bq/m ³)	Percentage >200 Bq/m ³	Change in life years lived by population relative to baseline		Change in annual space heating demand for ventilation relative to baseline (TWh)
			0-20 years	0-50 years	
Present (baseline)	21.2	0.6			
Scenario 1 (increased air tightness)	33.2	2.0	-5200	-121 000	-27
Scenario 2 (as for scenario 1+purpose-provided ventilation)	25.5	1.2	-1800	-43 100	-15
Scenario 3 (as for scenario 2+MVHR)	19.6	0.5	4000	21 500	-22
Scenario 4 (as for scenario 3+10% failure in MVHR)	21.8	0.6	-300	-7000	-22

MVHR=mechanical ventilation and heat recovery.
TWh=terawatt hours.

Effect of ageing on cervical or vaginal cancer in Swedish women previously treated for cervical intraepithelial neoplasia grade 3: population based cohort study of long term incidence and mortality

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STUDY QUESTION

How does ageing affect the long term risks of invasive cervical or vaginal cancer among women previously diagnosed and treated for cervical intraepithelial neoplasia grade 3 (CIN3)?

SUMMARY ANSWER

The risk of developing and dying from cervical or vaginal cancer among women previously treated for CIN3 is strongly increased at ages 60 and 70 years, respectively; treatment for CIN3 after age 50 enhances the risk.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS

There are no previous studies on ageing and the risk of contracting or dying from cervical or vaginal cancer after a diagnosis of CIN3. Women previously treated for CIN3 should be followed up in old age.

Participants and setting

All women in Sweden with CIN3, diagnosed and treated during 1958-2008 and followed up for invasive cervical or vaginal cancer, and related mortality.

Design, size, and duration

The cohort comprised 150 883 women with CIN3 (3 148 222 woman years) and was established from Swedish Cancer Registry data. Invasive cancer mortality and cause specific mortality in 1958-2009 were followed up in the Swedish Cancer Registry and the Swedish Cause of Death Register, respectively. We calculated standardised incidence and mortality ratios for cervical and vaginal cancer, using the expected number of cancers and deaths in the general female population in Sweden as the denominator. The ratios were stratified for attained age, treatment

period, and age at treatment for CIN3. We estimated ratios of these variables in multivariable regression models, which were validated by assessing individual data from an open cohort of 6 692 276 Swedish women followed up for CIN3, and the risk of subsequent cervical or vaginal cancer, from 1960 to 2009.

Main results and the role of chance

Women previously diagnosed with CIN3 had an increased risk of acquiring (standardised incidence ratio 2.39, 95% confidence interval 2.26 to 2.53) and dying from (standardised mortality ratio 2.35, 2.11 to 2.61) cervical or vaginal cancer. The risk of contracting invasive cancer accelerated after age 60 years, as did mortality risk after age 70 years. Regression analyses indicated that the increase in risk over time was highly attributable to ageing and that high age at treatment of CIN3 also has a significant effect.

Bias, confounding, and other reasons for caution

Possible selection and observational biases concerning women with CIN3 could have affected the results, presumably in the direction of an underestimation of risks. There is reason to believe that the cohort treated for CIN3 have had a more intense follow up than the controls. The study's long follow-up—more than 45 years—might also favour a selection of women who are otherwise healthier than average. Modelling of several parameters reflecting time is prone to misinterpretation. However, similar results from the study were obtained from a supplementary cohort analysis, based on individual data.

Generalisability to other populations

Since screening programme standards in Sweden have been high for decades and atypical cytology has been managed in accordance with international developments, we expect the results to be highly generalisable in areas with programmes of similar standard. Women treated for CIN3 in areas with less intense follow-up might have higher risks.

Study funding/potential competing interests

All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf and declare: support from the Halland County Scientific Board and the Swedish Cancer Society for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

Incidence of cervical or vaginal cancer per 100 000 person years as a function of attained age

