RESEARCH REPORT

Outdoor air pollution and infant mortality: analysis of daily time-series data in 10 English cities

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Background: There is growing concern that moderate levels of outdoor air pollution may be associated with infant mortality, representing substantial loss of life-years. To date, there has been no investigation of the effects of outdoor pollution on infant mortality in the UK.

Methods: Daily time-series data of air pollution and all infant deaths between 1990 and 2000 in 10 major cities of England: Birmingham, Bristol, Leeds, Liverpool, London, Manchester, Middlesbrough, Newcastle, Nottingham and Sheffield, were analysed. City-specific estimates were pooled across cities in a fixed-effects meta-regression to provide a mean estimate.

Results: Few associations were observed between infant deaths and most pollutants studied. The exception was sulphur dioxide (SO₂), of which a 10 µg/m³ increase was associated with a RR of 1.02 (95% CI 1.01 to 1.04) in all infant deaths. The effect was present in both neonatal and postneonatal deaths.

Conclusions: Continuing reductions in SO₂ levels in the UK may yield additional health benefits for infants.

There is now widespread acceptance that short-term increases in ambient air pollution are associated with increased mortality and morbidity, especially in elderly people and those with pre-existing health problems. However, there is now growing concern that there may also be a link with infant mortality and adverse pregnancy outcomes, representing substantial loss of life-years.¹

Some recent reviews on this subject present mixed results and are only in agreement that further research is needed to confirm and clarify any links.^{2–5} Infants may be particularly vulnerable to the adverse effects of air pollution.⁶ The lung is not well developed at birth,³ with 80% of alveoli being formed postnatally.⁷ During the neonatal and post-neonatal periods, therefore, the developing lung is highly susceptible to environmental toxicants.^{7–9}

Associations between particulate matter <10 µg/m³ (PM₁₀) and infant mortality have been observed in time-series studies conducted in cities with notoriously high levels of pollution, such as Mexico City,¹⁰ Seoul¹¹ and Sao Paulo.¹² However, it cannot be assumed that the much lower levels of exposure experienced currently on a daily basis in many Western cities have no harmful effects on susceptible subjects such as infants. Associations between post-neonatal mortality and ambient levels of particulates have been observed in spatial comparisons within the Czech Republic¹³ and the US.^{14 15}

To date, there has been no investigation on the effects of outdoor pollution on infant mortality in the UK. We analyse here time-series data of daily infant mortality counts in 10 major English cities to quantify any associations with short-term changes in air pollution.

METHODS

Data

Data on all-cause infant deaths (death within the first year of life) recorded between 1990 and 2000 were obtained from the Office for National Statistics for the following 10 major cities in England: Birmingham, Bristol, Leeds, Liverpool, London, Manchester, Middlesbrough, Newcastle, Nottingham and Sheffield. For each city, data were collapsed by date of death to generate a time series of daily infant death counts between 1990 and 2000. Further series were created separately for

neonatal deaths (death within first 28 days) and post-neonatal deaths (after day 28 and within the first year).

Daily measures of the following six pollutants were also obtained for the period 1990–2000 from the UK Air Quality Network: carbon monoxide (CO), nitrogen oxide (NO), nitrogen dioxide (NO₂), ozone (O₃), particulate matter (PM₁₀) and sulphur dioxide (SO₂). A minimum of two monitoring sites were available for each city, except for Middlesbrough and Newcastle, where only one site was used. For each pollutant, sites providing <30% of missing data were combined to produce a single series for each city. For each pollutant, correlations between sites were high within each city ($r > 0.74$).

For the same study period, daily maximum and minimum temperature (°C) and daily relative humidity (%) were obtained from the British Atmospheric Data Centre, using one weather station in each city. Daily mean temperature was estimated as the mean of the daily maximum and minimum values. Region-specific reports of laboratory-confirmed influenza A and respiratory syncytial virus activity were also collected from the Health Protection Agency.

Analysis

For each city, daily infant mortality was examined in relation to air pollution using Poisson generalised linear models allowing for overdispersion. Weekly reports of influenza A and respiratory syncytial virus activity were incorporated into each regression model as possible confounding variables, regardless of statistical significance. The non-linear effects of weather were also controlled for using natural cubic splines of mean temperature and relative humidity. In the case of relative humidity, the measure was modelled using the mean of levels on the day of death and the previous 2 days (lags 0–2), and the potential long-term effects of mean temperature were modelled using averaged values of lags 0–7. Three degrees of freedom (df) were used for each of these spline functions. Indicator variables were used to allow for any day-of-week effects.

Cubic smoothing splines of time with equally spaced df were used to control for secular trends (eg, demographic shifts) and any seasonal fluctuations in general birth numbers. Seven df

Abbreviations: df, degrees of freedom; PM₁₀, particulate matter <10 µg/m³

Table 1 Summary statistics for infant mortality and air pollution data between 1990 and 2000

City	Total infant deaths	Neonatal, %	Mean daily pollution concentrations (25th, 75th centiles) ($\mu\text{g}/\text{m}^3$)					
			CO (mg/m^3)	NO	NO ₂	O ₃	PM ₁₀	SO ₂
Birmingham	2354	71.4	0.64 (0.4, 0.8)	24.3 (7, 27)	43.0 (29, 55)	33.5 (20, 45)	21.9 (14, 26)	17.5 (7, 22)
Bristol	644	64.3	1.01 (0.6, 1.2)	47.9 (18, 55)	66.3 (50, 78)	23.8 (12, 33)	23.1 (15, 28)	17.8 (7, 21)
Leeds	2027	62.1	0.73 (0.5, 0.9)	41.5 (18, 49)	48.0 (37, 58)	30.2 (19, 40)	24.0 (15, 28)	17.0 (6, 21)
Liverpool	1050	62.6	0.51 (0.3, 0.6)	37.7 (14, 49)	43.9 (30, 56)	34.8 (21, 47)	23.6 (15, 28)	21.3 (6, 28)
London	9037	66.1	0.77 (0.5, 0.9)	48.0 (22, 57)	66.1 (54, 75)	20.5 (10, 28)	26.3 (18, 31)	21.3 (9, 27)
Manchester	3404	63.2	0.63 (0.4, 0.7)	36.3 (12, 40)	49.0 (35, 58)	25.0 (14, 33)	22.6 (15, 27)	15.1 (9, 18)
Middlesbrough	574	62.9	0.37 (0.2, 0.4)	11.1 (4, 11)	28.3 (18, 37)	42.6 (31, 54)	18.4 (11, 22)	11.4 (4, 15)
Newcastle	986	66.6	0.67 (0.5, 0.8)	33.7 (14, 41)	41.0 (29, 52)	35.6 (23, 47)	21.5 (14, 26)	14.9 (7, 18)
Nottingham	963	67.3	0.62 (0.4, 0.7)	28.5 (11, 32)	45.1 (35, 54)	28.1 (17, 37)	20.7 (14, 24)	16.0 (10, 19)
Sheffield	1249	67.7	0.60 (0.3, 0.7)	60.1 (23, 74)	51.0 (38, 62)	34.5 (23, 45)	23.0 (15, 28)	16.5 (7, 21)

CO, carbon monoxide; NO, nitrogen oxide; NO₂, nitrogen dioxide; O₃, ozone; PM₁₀, particulate matter <10 $\mu\text{g}/\text{m}^3$; SO₂, sulphur dioxide.

per year (roughly equivalent to a 2-month moving average) were used for these smoothing splines. These parameters were constrained to be the same for all cities, although the sensitivity of estimates to the degree of seasonal control was also examined.

To assess the short-term effects of pollution exposure on infant mortality, each pollutant was modelled using the average value of lags 0–2 days before death. Each pollutant was modelled as a linear term and considered separately from other pollutants. Pollutant effects are presented as the relative risk of mortality associated with a 10-unit increase (1 unit for CO) in the pollutant measure. For each pollutant, city-specific estimates were pooled across cities in a fixed-effects meta-regression to provide a mean estimate.

Analyses were repeated separately for neonatal and post-neonatal deaths. All analyses were conducted in STATA version 9.

RESULTS

The average infant mortality rate in the 10 study cities in 2000 was 7.75 per 1000 deaths. The city-specific rates for Birmingham (10.54/1000) and Leeds (10.25/1000) were

considerably higher than the average, and lower in Liverpool (5.36/1000), Bristol (5.46/1000) and Newcastle (5.78/1000). Table 1 provides summary statistics for infant deaths and averaged pollution data for each city between 1990 and 2000. Birmingham had a considerably higher proportion of neonatal deaths than other cities. London and Bristol generally experienced high levels of all pollutants compared with other cities, except for O₃, which tends to be negatively correlated with most of the other pollutants in winter months. In general, SO₂ and, to a lesser extent, PM₁₀ levels decreased over the study period and O₃ levels have risen slightly.

Figure 1 shows the relative risk (RR) of infant death for every 10-unit increase in each pollutant (1 unit for CO). For each city, generally few associations were observed with any of the pollutants. Although Bristol had an increased risk with all pollutants, only in the case of SO₂ was the risk statistically significant at the 5% level. The combined estimates suggested no relationship between pollutants and infant deaths, except in the case of SO₂, for which a 10 $\mu\text{g}/\text{m}^3$ increase was associated with a RR of 1.02 (95% CI 1.01 to 1.04, $p = 0.008$). Restricting analysis to just the summer months (April–September) left the

Note: Middlesbrough SO₂ model did not converge

Figure 1 RR of all infant mortality for 10-unit increase in pollutant (1 unit for carbon monoxide). Pollutant measure as average of lags 0–2 days. CO, carbon monoxide; NO, nitrogen oxide; NO₂, nitrogen dioxide; O₃, ozone; PM₁₀, particulate matter <10 $\mu\text{g}/\text{m}^3$; SO₂, sulphur dioxide.

Table 2 RR (95% CI) for 10-unit increase in pollutant (1 unit for carbon monoxide), for all infant deaths, and by neonatal and post-neonatal deaths. Only mean effects are presented. Pollutant measure was presented as an average of lags 0–2 days.

	CO	NO	NO ₂	O ₃	PM ₁₀	SO ₂
All infant deaths	1.02 (0.96 to 1.09)	1.00 (0.99 to 1.01)	1.00 (0.99 to 1.02)	1.00 (0.98 to 1.03)	0.99 (0.97 to 1.01)	1.02 (1.01 to 1.04)
Neonatal deaths	0.99 (0.92 to 1.07)	1.00 (0.99 to 1.01)	1.00 (0.98 to 1.02)	0.99 (0.96 to 1.02)	0.99 (0.97 to 1.02)	1.02 (1.00 to 1.04)
Post neonatal deaths	1.09 (0.94 to 1.25)	1.00 (0.99 to 1.01)	1.01 (0.98 to 1.04)	0.99 (0.92 to 1.06)	0.99 (0.95 to 1.03)	1.03 (0.98 to 1.08)

CO, carbon monoxide; NO, nitrogen oxide; NO₂, nitrogen dioxide; O₃, ozone; PM₁₀, particulate matter <10 µg/m³; SO₂, sulphur dioxide.

effect estimate for O₃ largely unchanged, but the SO₂ effect was larger in the summer months: 1.03 (1.00 to 1.06). In winter (October–March), the SO₂ effect was 1.01 (0.99 to 1.04).

On repeating the analysis separately for neonatal and post-neonatal deaths, the SO₂ effect was found to remain for both age groupings (table 2). Very few other differences were observed, except a very strong adverse effect of CO in post-neonatal deaths, although CIs were wide due to small numbers.

Effect estimates were largely unchanged when more seasonal control (10 df/year) was used in all models.

Discussion

On the basis of previous evidence, our prior hypothesis was that PM₁₀ may be adversely associated with infant mortality; however, our results suggested a link with SO₂ on both neonatal and post-neonatal mortality. Exposure to SO₂ may irritate the respiratory system, with high concentrations causing constriction of the bronchi and increasing mucous flow, making breathing difficult. Children may be particularly susceptible to such effects. A similar time-series study from Sao Paulo reported a 6% (95% CI 4 to 8) increase in neonatal deaths associated with the interquartile range (9.2 µg/m³) of SO₂.¹² Our estimate of 2% (0 to 4) was roughly for a similar change in SO₂ levels. In a spatial study, Bobak and Leon found associations between infant mortality in the Czech Republic and both total suspended particles and SO₂, which were specific to respiratory mortality in the post-neonatal period.¹³ These results were later reproduced in a case-control study, where an odds ratio (95% CI) of 1.74 (1.01 to 2.98) was estimated for a 50 µg/m³ increase in SO₂ on post-neonatal respiratory mortality.¹ Results of our present study were much smaller, but were on all-cause deaths and only consider, effects of short-term changes in air pollution as opposed to cumulative exposures.

What this paper adds

- An adverse effect of SO₂ exposure was observed on both neonatal and post-neonatal mortality.
- No effects of particulate matter <10 µg/m³ were observed.

Policy implications

- Continuing reductions in SO₂ levels in the UK may yield additional health benefits in infants.

Another spatial comparison from the US by Woodruff *et al*¹⁴ estimated an OR (95% CI) of 1.10 (1.04 to 1.16) of total post-neonatal mortality in the highest tertile of PM₁₀ exposure compared with the lowest tertile. An equivalent comparison from our current study for just PM₁₀ levels in London and post-neonatal deaths gives a RR of 0.94 (0.87 to 1.01), suggesting no contribution from PM₁₀ and no overlap with the Woodruff estimate.

Recent time-series and case-crossover studies have also implicated PM₁₀^{10–16} or NO₂,¹⁷ but no role for SO₂ was observed. Furthermore, no significant effect of CO was observed in these studies—our results suggested that CO may have a strong adverse effect on post-neonatal deaths, although our estimate was imprecise. Other temporal studies have only considered effects of PM₁₀.¹¹

Recent work has demonstrated that correlations between ambient levels and personal exposure of gaseous pollutants such as SO₂ is lower than those for fine particles, and that ambient gaseous pollutant concentrations may be better surrogates of personal PM_{2.5} exposures than they would be as surrogates of personal exposures to the gases themselves.^{18–19} This is most likely to be the case in low-ventilated environments; however, our SO₂ effect was strongest in the summer-time, when ventilation is at its highest in UK homes.

A long time-series was used in the present study—11 years of data from 10 major English cities allowed us to robustly estimate the effects on all-cause mortality. In addition, all effects were insensitive to the different levels of seasonal control, suggesting that our original model choice was satisfactory.

In conclusion, our results suggest an adverse effect of SO₂ exposure on both neonatal and post-neonatal mortality. Continuing reductions in SO₂ levels in the UK may yield additional health benefits for infants.

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SPEAKER'S CORNER.....

Publishing has become a "Russian roulette"

As authors, every now and then we get rather prejudiced, biased or scientifically unfounded—if not fully unethical, because of conflicts of interest—comments from manuscript reviewers. Some journal editors seldom seem to feel accountable for the choice of reviewers.

Today, I seek advice from a colleague and friend, a widely recognised scientist at a top-ranking college in London, UK, about suitable journals to which we could resubmit a given paper. Other than the specific advice, his main reflection is:

Publishing has become a "Russian roulette".

Hopefully, not so in *JECH* and in most other journals. But the debate is worthwhile.^{1 2}

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