Association between air pollution and acute childhood wheezy episodes: prospective observational study

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Abstract

Objective—To examine the association between the air pollutants ozone, sulphur dioxide, and nitrogen dioxide and the incidence of acute childhood wheezy episodes.

Design—Prospective observational study over one year.

Setting—District general hospital.

Subjects—1025 children attending the accident and emergency department with acute wheezy episodes; 4285 children with other conditions as the control group.

Main outcome measures—Daily incidence of acute wheezy episodes.

Results—After seasonal adjustment, day to day variations in daily average concentrations of ozone and sulphur dioxide were found to have significant associations with the incidence of acute wheezy episodes. The strongest association was with ozone, for which a non-linear U shaped relation was seen. In terms of the incidence rate ratio (1 at a mean 24 hour ozone concentration of 40 $\mu\text{g}/\text{m}^3$ (SD=19.1)), children were more likely to attend when the concentration was two standard deviations below the mean (incidence rate ratio=3.01; 95% confidence interval 2.17 to 4.18) or two standard deviations above the mean (1.34; 1.09 to 1.66). Sulphur dioxide had a weaker log-linear relation with incidence (1.12; 1.05 to 1.19 for each standard deviation (14.1) increase in sulphur dioxide concentration). Further adjustment for temperature and wind speed did not significantly alter these associations.

Conclusions—Independent of season, temperature, and wind speed, fluctuations in concentrations of atmospheric ozone and sulphur dioxide are strongly associated with patterns of attendance at accident and emergency departments for acute childhood wheezy episodes. A critical ozone concentration seems to exist in the atmosphere above or below which children are more likely to develop symptoms.

Introduction

In the past 20 years in the United Kingdom both the incidence and the prevalence of childhood asthma have risen dramatically.^{1,2} There is considerable speculation about the reasons for this change, although the phenomenon seems to be worldwide.³ Evidence from lung function studies suggests that the bronchial airways of children have become more hyperresponsive.⁴ This phenomenon and the seasonal fluctuations seen in the incidence of asthma suggest that environmental factors have an important role in the aetiology of the disease. In temperate regions the incidence peaks during the early autumn and in midwinter.⁵ Viral infection is undoubtedly an important aetiological factor in childhood asthma⁶; media interest, however, has focused

on air pollution as another possible reason for the increased incidence. In the United Kingdom concentrations of certain pollutants, such as sulphur dioxide, have fallen since the introduction of the Clean Air Act in 1956.⁷ Increased use of cars, however, has caused an increase in other air pollutants, such as nitrogen dioxide and reactive hydrocarbons. Small particulate air pollution—much of it derived from diesel exhaust—is also under increasing scrutiny.⁸ Under certain atmospheric conditions, such as high temperatures and low wind speed, many of these primary pollutants, under the influence of sunlight, generate an excess of ozone, a gas with powerful oxidant properties.^{7,9}

Several studies have examined the effect of air pollutants by measuring fluctuations in admission and attendance rates at accident and emergency departments.¹⁰⁻¹⁵ These studies show a pattern that suggests that fluctuations in concentrations of air pollutants may trigger asthma attacks in susceptible individuals or may interact in a more complex way to increase airway hyperresponsiveness to virus or allergy triggers. We investigated the association between sulphur dioxide, nitrogen dioxide, and ozone and rates of attendance for wheezy episodes at a local accident and emergency department.

Methods

SUBJECTS

From 1 March 1992 to 28 February 1993 we recorded the attendance of all children (aged 16 years or under) who presented at the accident and emergency department of the Hillingdon Hospital, west London, with symptoms of acute wheezing with or without cough or shortness of breath (n=1025). We gathered the data on a daily basis from the accident and emergency cards completed by the nurse at the triage station. We included as controls all other children presenting to the accident and emergency department during the same time period except those with minor injuries and those with cough not associated with wheeze (n=4285).

AIR POLLUTANT AND METEOROLOGICAL DATA

The London Borough of Hillingdon's environmental protection unit provided the data on pollutants. Ozone, sulphur dioxide, and nitrogen dioxide concentrations were measured by differential absorption spectroscopy, in the light and near ultraviolet wavebands, with a monitor (OPSIS, Sweden) sited on the roof of a hotel near Heathrow airport. The technique is accredited by the United States Environmental Protection Agency and has been evaluated recently in Britain.¹⁶ Provided that monitors are calibrated regularly, the technique is accurate (and reproducible for the pollutants measured in this study). Heathrow airport is 5 km south of the Hillingdon Hospital and at the southern end of the borough of Hillingdon, about

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Fig 1—Map showing location of Hillingdon Hospital, pollution monitor near Heathrow, and major roads in borough

25 km west of central London (fig 1). From measurements made about every eight minutes, we calculated 24 hourly mean values. The Meteorological Office in Bracknell provided meteorological data—from measurements taken at the site near Heathrow airport, including mean 24 hourly temperature (°C) and wind speed (knots).

STATISTICAL ANALYSIS

Data were analysed with the STATA statistical software package.¹⁷ Scatterplots, locally weighted regression scatterplot smoothing averages,¹⁸ and Spearman's rank correlation coefficients were initially used to examine possible associations between daily (case and control) incidence and each of the pollutants (ozone, sulphur dioxide, and nitrogen dioxide) and weather variables. Poisson regression models were then used to explore formally the effect of each pollutant on daily incidence.¹⁹ These models assume that the daily incidence followed a Poisson distribution with mean (daily incidence rate) log-linear in the predictor variables, with initially season as a four level factor (spring, 22 March to 21 June; summer, 22 June to 21 September; autumn, 22 September to 21 December; and winter, 22 December to 21 March). The analysis was repeated, with the addition of temperature and then temperature and wind speed, and then in the case of ozone the weather variables plus the other two pollutants. The weather factors have previously been shown to affect incidence of asthma within seasons.^{10 11}

We used the Poisson distribution because the distribution of the daily incidence was highly skewed. Normal models but with log-link functions, however, gave qualitatively similar results, as did adjustment for autocorrelation by incorporating the previous day's incidence in the model. For each variable *v*, a restricted natural cubic spline function with knots at the 10th, 50th, and 90th centiles was used in the Poisson model to test formally for non-linearity in the relation between *v* and the daily incidence.²⁰ This amounted to adding a completely specified piecewise cubic function *S(v)* to the variable *v* in the model (see appendix). The test for non-linearity is equivalent to testing the significance of *S(v)*. The possibility of a lag effect of each of the pollutants was also explored by comparing rank correlation coefficients of daily incidence with each variable lagged by 1, 2, 3, 4, 5, 6, or 7 days and by

comparing the resulting deviances obtained when each of the lagged variables was fitted separately in a Poisson model. All *P* values quoted are for two tailed tests, and results are significant if *P* < 0.05.

Results

In all, 1025 children attended the accident and emergency department with an acute wheezy episode (median daily incidence 2; interquartile range 1-4), and 4285 children attended for other causes (12; 9-14). Table 1 summarises the data on pollutants and incidence by season. Figure 2 shows the time series in terms of daily incidence of wheezy episodes and pollutant concentrations. Owing to a failure of the monitor, pollutant data are missing for 25-27 July and 26 September to 6 October. Figure 3 shows the

Fig 2—Time series of mean 24 hour concentrations of ozone, sulphur dioxide, and nitrogen dioxide and daily incidence of acute wheezy episodes

Table 1—24 Hour means for air pollutants and incidence of wheezy cases and controls. Values in parentheses are standard deviations

	Spring	Summer	Autumn	Winter	All seasons
Pollutant (µg/m³):					
Ozone	48 (15)	41 (15)	44 (26)	28 (15)	40 (19)
Sulphur dioxide	20 (14)	18 (22)	24 (14)	25 (14)	22 (14)
Nitrogen dioxide	59 (19)	55 (18)	66 (13)	61 (17)	60 (17)
Cases	2.3 (1.9)	2.0 (1.6)	3.8 (2.1)	3.1 (2.2)	2.7 (2.0)
Controls	11.5 (3.5)	11.6 (4.2)	12.2 (3.3)	11.7 (4.0)	11.7 (3.8)

Table 2—Spearman's correlation coefficients for cases and controls with air pollutants and weather variables

	Cases	Controls	Ozone	Sulphur dioxide	Nitrogen dioxide	Temperature
Ozone	-0.25***	-0.02				
Sulphur dioxide	0.17**	0	-0.28***			
Nitrogen dioxide	0.14**	0.06	-0.18**	0.62***		
Temperature	-0.21***	0.02	0.48***	-0.30***	-0.26***	
Wind speed	-0.18**	0.01	0.27***	-0.57***	-0.64***	0.10*

P* < 0.05; *P* < 0.01; ****P* < 0.001 (*P* values are corrected for multiple comparison with Bonferroni method).

scatterplots of cases and controls with respect to each of the three pollutants together with smoothed averages. The figure shows stronger associations with cases than with controls, particularly for ozone. Table 2 shows Spearman's rank correlation coefficients between pollutants and weather variables for cases and controls. Significant negative correlations were found for cases for ozone, temperature, and wind speed. The correlation with ozone is perhaps not the most appropriate summary in view of the non-linear relation between incidence of wheezing and ozone concentration. Significant positive correlations were found with sulphur dioxide and nitrogen dioxide. No significant correlations were found in the controls.

The incidence of acute wheezy episodes differed significantly between seasons ($\chi^2=66.5$, *df*=3, *P* < 0.0001). Overall, the highest incidence occurred during the autumn (table 1), with an incidence rate ratio relative to the summer of 1.89 (95% confidence interval 1.58 to 2.26). For spring and for winter the values relative to the summer were 1.12 (0.92 to 1.36) and 1.56 (1.29 to 1.87) respectively. For controls no significant difference was found between the seasons.

The incidence of acute wheezy episodes was found

Fig 3—Scatterplots for daily incidence of acute wheezy episodes and daily attendance rates of control children by mean 24 hour concentrations of ozone, sulphur dioxide, and nitrogen dioxide; smoothed averages are shown by solid line. The Poisson model (unadjusted) is shown as dotted line for cases in relation to ozone concentrations

to have significant associations with all three air pollutants (table 3). After seasonal adjustment, only the association with ozone and sulphur dioxide remained significant, with ozone the most highly significant. The ozone effect was non-linear; testing for linearity of the effect of ozone on the log incidence rate gave $\chi^2=33.2$, $df=1$, $P<0.0001$. Relative to a mean ozone concentration of $40 \mu\text{g}/\text{m}^3$, an ozone

concentration two standard deviations (38.2) below gave a significantly higher incidence—a threefold increase (incidence rate ratio 3.01 ; 95% confidence interval 2.17 to 4.18). An ozone concentration two standard deviations above the mean was associated with a 34% increase (1.34 ; 1.09 to 1.66). The effect of ozone persisted after further adjustment for temperature and wind speed. Even when ozone was adjusted for the other two pollutants the U shaped relation remained highly significant. The Poisson model (unadjusted) predicting incidence is overlaid on the scatterplot (fig 3).

The association between sulphur dioxide concentrations and log incidence of acute wheezy episodes was significant but with no evidence of non-linearity. After seasonal adjustment for each standard deviation rise of sulphur dioxide the incidence increased by 12% (1.12 ; 1.05 to 1.19). The test for linearity gave $\chi^2=0.06$, $df=1$, $P=0.81$. For nitrogen dioxide a weak positive relation was found before seasonal adjustment (table 3), but after seasonal adjustment no significant association was found. No significant associations were found with controls. Analyses were repeated with logs of 1, 2, 3, 4, 5, 6, and 7 days. No clear lag effect was found.

Discussion

The reliability of diagnoses made in accident and emergency departments in epidemiological studies has been questioned.^{12 21} In children, especially those under the age of 2 years, the clinical distinction between conditions such as atopic and non-atopic asthma, wheezy bronchitis, and bronchiolitis may be impossible to make with certainty. Also at issue is the degree to which attendance rates in accident and emergency departments reflect the prevalence of acute asthma in the community as a whole. At the Hillingdon hospital over 60% of childhood wheezy episodes are self referrals, a figure that remains relatively constant throughout the year.

The application of non-linear Poisson regression models may be the reason that our study showed significant events not previously observed. Some studies have used linear regression models; others have assumed that data conformed to a normal distribution. The models used in our study showed that sulphur dioxide has a positive log-linear association with the incidence of acute wheezy episodes. The most important finding was the U shaped relation between same day atmospheric ozone concentrations and daily incidence. The lowest incidence was seen at a mean

Table 3—Incidence rate ratios (95% confidence interval) of acute wheezy episodes for each standard deviation change of pollutant relative to mean concentration, adjusted for season, weather, and air pollutants

Standard deviation change	Adjustment				
	None	Season	Season plus temperature	Season plus temperature and wind speed	Season plus temperature, wind speed, sulphur dioxide, and nitrogen dioxide
Ozone					
-2	3.60 (2.56 to 5.06)	3.01 (2.00 to 4.53)	3.27 (2.13 to 5.02)	3.01 (1.86 to 4.87)	2.90 (1.88 to 4.48)
-1	1.70 (1.48 to 1.96)	1.59 (1.34 to 1.88)	1.65 (1.38 to 1.97)	1.59 (1.30 to 1.95)	1.56 (1.30 to 1.87)
0	1	1	1	1	1
1	1.08 (0.98 to 1.19)	1.04 (0.94 to 1.14)	1.02 (0.92 to 1.13)	1.01 (0.92 to 1.12)	1.04 (0.94 to 1.15)
2	1.53 (1.19 to 1.96)	1.34 (1.03 to 1.75)	1.31 (1.00 to 1.70)	1.28 (0.97 to 1.68)	1.35 (1.03 to 1.76)
Sulphur dioxide					
1	1.16 (1.10 to 1.23)	1.12 (1.06 to 1.19)	1.12 (1.06 to 1.19)	1.08 (1.00 to 1.16)	NA
Nitrogen dioxide					
1	1.07 (1.01 to 1.14)	1.03 (0.96 to 1.09)	1.02 (0.96 to 1.09)	1.20 (0.85 to 1.09)	NA

NA=not applicable.

ozone concentration of about $40 \mu\text{g}/\text{m}^3$. The effect persisted even after adjustment for season, temperature, and wind speed. We could not find a precedent for this observed effect. The inference is that an optimal protective concentration of atmospheric ozone exists.

Ozone has been used in laboratories in the past as an antiseptic and as a medicinal inhalation in the 19th century.^{22, 23} Its oxidant powers with regard to destruction of airborne pathogens, however, are speculative.²⁴ Evidence has emerged recently that the human alveolar macrophage infected with the respiratory syncytial virus may have an attenuated inflammatory response after exposure to critical concentrations of ozone.²⁵ Alternatively, other unidentified confounding factors—such as infective or allergic factors or even other pollutants—may be operating at low concentrations of ozone. With respect to other pollutants, both sulphur dioxide and nitrogen dioxide concentrations were at their highest at the lowest concentrations of ozone, progressively decreasing with increasing ozone concentrations until approximately $40 \mu\text{g}/\text{m}^3$ (fig 4). A possible explanation for this is that the meteorological circum-

Fig 4—Scatterplots with smoothed averages for relation between mean 24 hour concentrations of ozone and sulphur dioxide and between mean 24 hour concentrations of ozone and nitrogen dioxide

stances which give rise to low ozone concentrations—namely, cold, still, anticyclonic weather—also cause a low boundary layer mixing height (A R MacKenzie, personal communication). The effect of this is twofold: ozone deposition is enhanced, thus ozone concentrations are lowered; and emissions are trapped in a shallower layer of atmosphere, allowing accumulation of sulphur dioxide, nitrogen dioxide, and possibly many other pollutants. The rise in sulphur dioxide and nitrogen dioxide at the highest concentrations of ozone may occur either on hot still days or during times of stratospheric intrusions, such as thunderstorms or low pressure systems.²⁶ The variations in sulphur dioxide and nitrogen dioxide on their own, however, could not explain the U shaped relation between ozone and incidence of asthma. The relation remained strong even after adjustment for sulphur dioxide and nitrogen dioxide.

Previous studies relating ozone concentrations to attendances for asthma at accident and emergency departments have shown conflicting results.^{12, 21, 27} Two studies from summer camps in North America have shown an adverse effect of ozone on the lung function

Key messages

- The incidence of childhood acute wheezy episodes is greatest in the autumn and winter
- This study shows that after seasonal adjustment the incidence was found to be high on days when ozone concentrations were very low or very high
- The log incidence increased in a linear manner with increasing concentrations of sulphur dioxide
- Nitrogen dioxide was not found to have a significant effect on the incidence of wheezy episodes
- The non-linear U shaped relation between ozone and incidence suggests that at low ozone concentrations either other factors are more important in determining wheezy episodes or an optimum protective concentration of ozone exists in the atmosphere

of active normal children.^{28, 29} The second national health and nutrition examination survey, a cross sectional analysis of lung function in over 4000 young people over the age of 6 years, was unable to show an effect of ozone until the highest 20% of exposure to ozone, suggesting a critical threshold of $80 \mu\text{g}/\text{m}^3$ (six hour measurement).³⁰ This is consistent with the strong non-linear relation we found in our study.

Our study has not sought to explain the pathophysiological mechanisms behind the interaction of pollutant and meteorological variables. For ozone these have been well reviewed.³¹ Clearly pollutants may interact with each other and with allergic or infective factors.^{32, 33} We have observed unexpectedly that concentrations of ozone above and below a critical level are associated with an increased incidence. Whether a physiological, chemical, or biometeorological explanation exists for this phenomenon is open to speculation.

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Appendix

Let z denote the ozone concentration measured in units of standard deviations from the mean. The mean ozone concentration was 40.2 and the standard deviation $19.5 \mu\text{g}/\text{m}^3$, so that $z = (\text{ozone} - 40.2)/19.5$. A restricted cubic spline function²⁰ with knots at the 10th, 50th (median), and 90th percentiles of z was used to model the relation between the daily incidence of wheezing episodes and ozone concentration. These percentiles were $P_{10} = -1.19$, $P_{50} = -0.17$, and $P_{90} = -1.58$. The number of episodes during a period of 24 hours is assumed to follow a Poisson distribution with log rate given by:

$$\beta_0 + \beta_1 z + \beta_2 S(z), \text{ where}$$

$$S(z) = 0 \text{ if } z \leq P_{10}$$

$$S(z) = (z - P_{10})^3 \text{ if } P_{10} \leq z \leq P_{50}$$

$$S(z) = (z - P_{10})^3 + \frac{(P_{90} - P_{10})(z - P_{50})^3}{(P_{90} - P_{50})} \text{ if } P_{50} < z \leq P_{90}$$

$$\text{and } S(z) = \frac{(P_{50} - P_{10})(P_{10} - P_{90})(P_{10} + P_{50} + P_{90})}{3(P_{50} - P_{10})(P_{90} - P_{10})} z \text{ if } z > P_{90}$$

The estimates (standard errors) of $\hat{\beta}_0, \hat{\beta}_1, \hat{\beta}_2$ were $0.49 (0.07)$, $-0.75 (0.08)$, and $0.13 (0.02)$ respectively. Relative to the mean value ($z=0$), the log incidence

rate ratio at level z is estimated by $\hat{\lambda} = \hat{\beta}_1 z + \hat{\beta}_2 S(z)$ and the standard error (λ) is estimated from the covariance matrix of $\hat{\beta}_1$ and $\hat{\beta}_2$. A 95% confidence interval for λ is then $\hat{\lambda} \pm SE(\hat{\lambda})\sqrt{\chi^2_{0.95}}$, where $\chi^2_{0.95}$ is the 95th centile of the χ^2 distribution with two degrees of freedom to yield a Scheffe-type simultaneous confidence interval.

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Air pollution and daily mortality in London: 1987-92

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Abstract

Objective—To investigate whether outdoor air pollution levels in London influence daily mortality.

Design—Poisson regression analysis of daily counts of deaths, with adjustment for effects of secular trend, seasonal and other cyclical factors, day of the week, holidays, influenza epidemic, temperature, humidity, and autocorrelation, from April 1987 to March 1992. Pollution variables were particles (black smoke), sulphur dioxide, ozone, and nitrogen dioxide, lagged 0-3 days.

Setting—Greater London.

Outcome measures—Relative risk of death from all causes (excluding accidents), respiratory disease, and cardiovascular disease.

Results—Ozone levels (same day) were associated with a significant increase in all cause, cardiovascular, and respiratory mortality; the effects were greater in the warm season (April to September) and were independent of the effects of other pollutants. In the warm season an increase of the eight hour ozone concentration from the 10th to the 90th centile of the seasonal range (7-36 ppb) was associated with an increase of 3.5% (95% confidence interval 1.7 to 5.3), 3.6% (1.04 to 6.1), and 5.4% (0.4 to 10.7) in all cause, cardiovascular, and respiratory mortality respectively. Black smoke concentrations on the previous day were significantly associated with all cause mortality, and this effect was also greater in the warm season and was independent of the effects of other pollutants. For black smoke an increase from the 10th to 90th centile in the warm season (7-19 $\mu\text{g}/\text{m}^3$) was associated with an increase of 2.5% (0.9 to 4.1) in all cause mortality. Significant but smaller and less consistent effects were also

observed for nitrogen dioxide and sulphur dioxide.

Conclusion—Daily variations in air pollution within the range currently occurring in London may have an adverse effect on daily mortality.

Introduction

The 1952 London smog episode was associated with a twofold to threefold increase in mortality and showed beyond doubt that air pollution episodes could be harmful to health.¹ Subsequent episodes in London were also found to be associated with increased mortality,² but by the late 1960s adverse health effects of outdoor pollution in London were thought to be unlikely.^{3,4} One of the first indications that this might not be the case came from an analysis of London data for 1958-72, which showed that daily mortality was associated with pollution levels on the previous day even after exclusion of episodes and that this effect was present as late as 1972.⁵⁻⁷ No further research into daily mortality and air pollution in London has been done since 1972.

In London, pollution due to the burning of coal has almost completely disappeared, and nowadays the main sources are directly or indirectly attributable to motor vehicles.^{8,9} The measured pollutants that are potentially harmful include particles, nitrogen dioxide, ozone sulphur dioxide, and volatile organic compounds. On most days concentrations of these are well within the World Health Organisation's guidelines, but occasional episodes of high concentrations of ozone in the summer and of nitrogen dioxide and particles in the winter are a cause of concern.¹⁰

Various recent studies, mainly from the United States, have reported associations between daily mor-

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