Health effects of an air pollution episode in London, December 1991

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Abstract

Background – In December 1991 London experienced a unique air pollution episode during which concentrations of nitrogen dioxide rose to record levels, associated with moderate increases in black smoke. The aim of this study was to investigate whether this episode was associated with adverse health effects and whether any such effects could be attributed to air pollution.
Methods – The numbers of deaths and hospital admissions occurring in Greater London during the week of the episode were compared with those predicted using data from the week before the episode and from equivalent periods from the previous four years. Relative risks (RR) (episode week versus predicted) for adverse health events were estimated using log linear modelling and these were compared with estimates from control areas which had similar cold weather but without increased air pollution.
Results – In all age groups mortality was increased for all causes (excluding accidents) (relative risk = 1·10) and cardiovascular diseases (1·14); non-significant increases were observed for all respiratory diseases (1·22), obstructive lung diseases (1·23), and respiratory infections (1·23). In the elderly (65 + years) the relative risk of hospital admission was increased for all respiratory diseases (1·19) and for obstructive lung diseases (1·43), and a non-significant increase was observed for ischaemic heart disease (1·04). In children (0–14 years) there was no increase in admissions for all respiratory diseases and only a small non-significant increase for asthma. When compared with control areas the relative risks became non-significant but remained increased.
Conclusions – The air pollution episode was associated with an increase in mortality and morbidity which was unlikely to be explained by the prevailing weather, a coincidental respiratory epidemic, or psychological factors due to publicity. Air pollution is a plausible explanation but the relative roles of nitrogen dioxide and particulates cannot be distinguished.
(*Thorax* 1995;50:1188–1193)

Keywords: air pollution episode, mortality, hospital admissions, particles, nitrogen dioxide.

In December 1991 an anticyclone lay over most of Britain and Western Europe, creating the cold and stagnant air conditions typically associated with long lasting fogs and air pollution episodes in London.[1][2] Emissions from motor vehicles and power sources were trapped by a temperature inversion which prevented the normal circulation and dispersion of pollutants.[3] On Thursday 12 December 1991 nitrogen dioxide (NO_2) levels in inner London exceeded the WHO hourly average guideline of 210 ppb[4] and, early on 13 December, the urban background monitoring site at Bridge Place, Victoria recorded an hourly average level of 423 ppb NO_2. This was the highest hourly average concentration ever recorded at a background site in London since measurements began in 1972. Levels remained high until Sunday 15 December, four days in all. Levels of black smoke during this period increased at the nearby Westminster site to a maximum daily average of 148 µg/m³, well above the monthly mean for that station of 43 µg/m³. Sulphur dioxide (SO_2) levels did not increase to the same extent. London was the only city in Britain to experience a major air pollution episode at that time.

The episode was publicised at the time and there was widespread public and medical concern about its possible health effects with reports from hospital doctors of an increase in hospital attendances, for asthma in particular. Our investigation aimed to determine whether the episode was associated with adverse health effects and, if so, to evaluate the possible role of air pollution. This paper reports the results concerning mortality and hospital admissions. Fuller details may be found in our report to the Department of Health.[5]

Methods

For the purpose of analysis the episode period was defined as the seven day period from Thursday 12 December to Wednesday 18 December ("episode week"). The episode week was compared with the week prior to the episode (5–11 December) ("previous week") and with the corresponding dates (5–11 and 12–18 December) of the preceding four years ("control years"). The study area was all District Health Authorities in Greater London (population 7·2 million). Three control areas were defined: (1) the rest of England (population 40·6 million): (2) the rest of the south east of England (population 10·2 million); and (3) Manchester (population 2·6 million), another large conurbation for which pollution data were available. The episode period and comparison weeks and areas were defined *a priori* and adhered to throughout.

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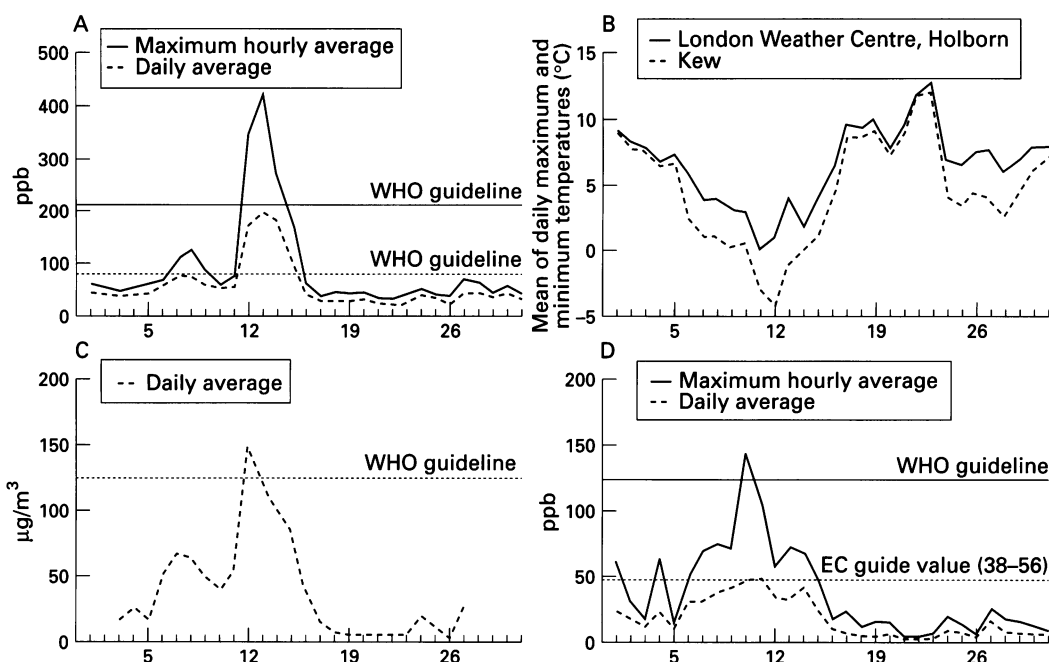Reprint requests to:
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Figure 1   Concentrations of (A) nitrogen dioxide, (C) black smoke, and (D) sulphur dioxide recorded in central London during December 1991. (B) Mean of daily maximum and minimum temperatures in London during December 1991.

Individual mortality records for England were obtained from the Office of Population Censuses and Surveys (OPCS). For a given area, eligible deaths were those of persons resident in that area and whose death was registered in that area. The diagnostic groups selected were: all causes (excluding those due to injury and poisoning, ICD <800); all respiratory (ICD 460–519); all lower respiratory infections (ICD 466, 480–87); obstructive lung diseases (ICD 490–496); all cardiovascular disease (ICD 390–459); and ischaemic heart disease (ICD 410–414). Hospital admission data (first "consultant episode") from all NHS hospitals were obtained from the Hospital Episode System (HES) for England, and categorised by area according to district of treatment. The diagnostic groupings analysed were: all respiratory (ICD 460–519); obstructive lung diseases (ICD 490–496); chronic obstructive pulmonary disease (ICD 490–496 excl 493); asthma (ICD 493); and ischaemic heart disease (ICD 410–414).

All air pollution data were obtained from Warren Spring Laboratory (now the AEA Technology National Environmental Technology Centre). Hourly average NO_2 levels, measured using the chemiluminescence technique, were obtained from three monitors, all in inner London. Hourly SO_2 levels, measured using ultraviolet fluorescence detectors, were obtained from one inner London monitor. In addition, from 11 "sampler" sites throughout greater London daily average data on particulates measured using the smoke stain method (black smoke) and SO_2 measured using the acidimetric bubbler system were obtained. More information on measurement aspects is available elsewhere.[6][7]

For each study area weekly totals of health events were calculated for the weeks beginning 5 and 12 December in the episode year (1991) and the control years 1987–90. These data are available in two tables from the authors. The modelling was performed using the generalised linear interactive modelling package (GLIM).[8] A log linear model was used with a Poisson error to model the number of events in each week for each year. The dependent variable was the number of events in a week. There were three independent variables. Year and week were entered as five-level and two-level factors respectively, and a dichotomous variable was constructed to represent the episode as

Table 1   Levels of air pollution during the episode (12–15 December) in relation to standards and average for the month

	NO₂ (ppb)*		Black smoke (µg/m³)**	SO₂ (ppb)*	
	Max hourly average	Daily average	Daily average	Max hourly average	24 hour average
WHO/EC guideline[4]	209	80	125†	122	38–56
Maximum during episode week‡	423	198	148	72	42
Ratio of maximum to WHO guidelines	2·0	2·5	1·2	0·5	0·9
December average	88	56	43	39	19
Ratio of maximum to December average	4·8	3·5	3·4	1·8	2·2

* Central London urban background monitor (Victoria).
** Central London sampler site (Westminster).
† With over 47 ppb SO_2.
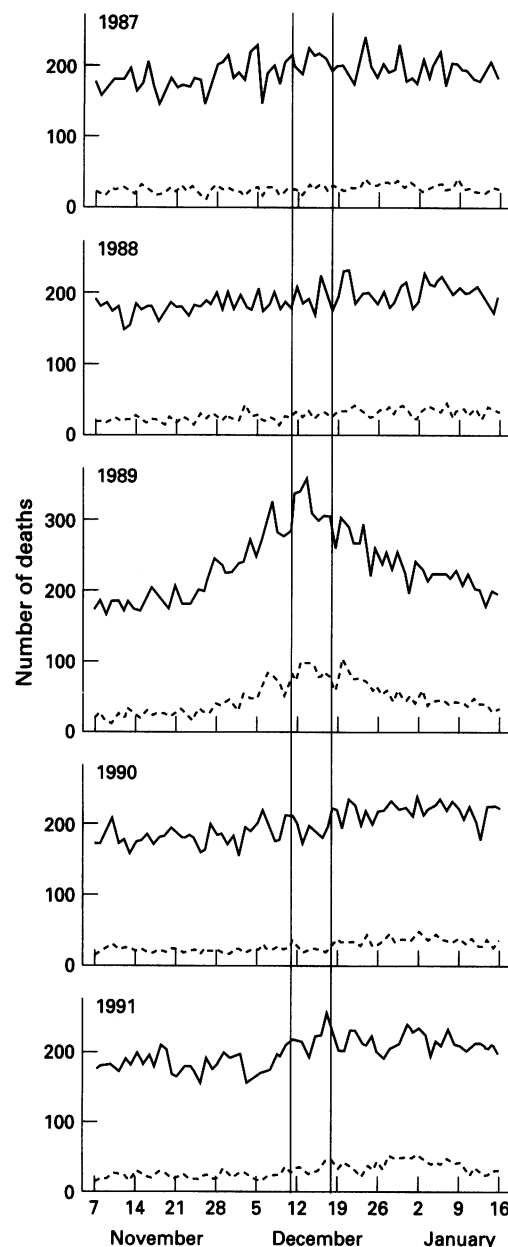‡ 12, 13, and 14 December for black smoke, NO_2, and SO_2, respectively.

Figure 2 Daily mortality for all ages from all causes (excluding accidents) (—) and respiratory causes (----) from 10 November 1991 to 16 January 1992, and the corresponding dates for 1987–90. The episode week (12–18 December) is shown within the vertical lines.
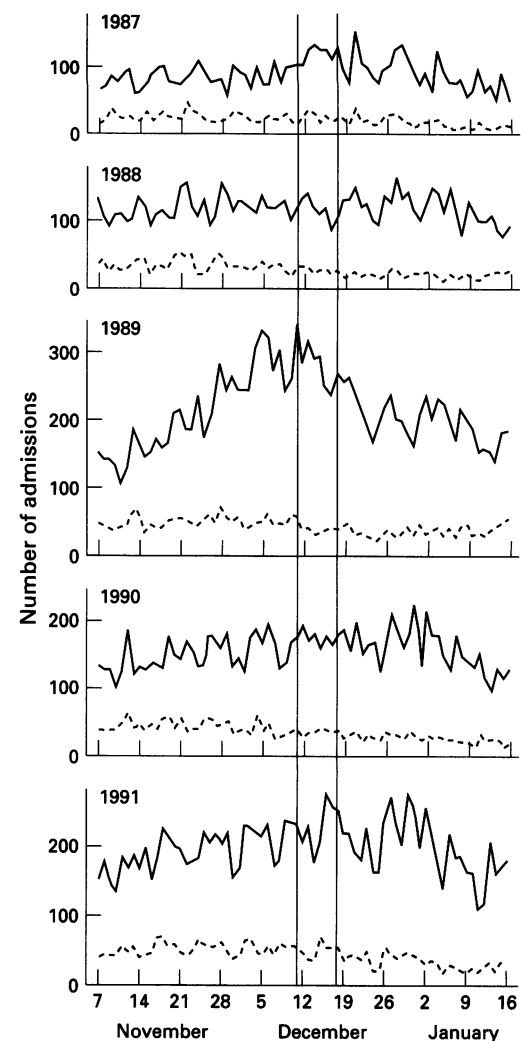

Figure 3 Daily hospital admission for all ages for all respiratory diagnoses (—) and for asthma (---) from 10 November 1991 to 16 January 1992, and the corresponding dates from 1987–90. The episode week (12–18 December) is shown within the vertical lines.

week 2 in year 5. The coefficient for this latter variable ("episode period") represented the logarithm of the relative risk of death (or admission) in the episode week over what would be expected from the number of events in the previous week and previous years. The relative risk of death (or admission) during the episode week was calculated by taking the exponential of this estimate; 95% confidence intervals were similarly calculated using the standard error of the estimate.

The relative excess of deaths or admissions in London during the episode compared with each control area was estimated by log linear Poisson regression models in which counts of health events were modelled as a function of area, year, week, and episode period (as main effects) plus terms representing the statistical interaction of area with year, area with week, and area with episode period. The ex-

ponentiated coefficient for the area/episode interaction term represents the ratio of the relative risks for the episode week in London and the comparison area, which we have called "relative excess risk".

Results

AIR POLLUTION

Concentrations of NO_2, black smoke, and SO_2 in central London for December 1991 are plotted in fig 1. Concentrations of all oxides of nitrogen (NOx) and carbon monoxide (not shown) rose in parallel, reaching peaks of 1500 ppb and 18 ppm, respectively. A parallel increase was seen in black smoke; SO_2 levels also increased but these peaked two days before the highest levels of NO_2 and black smoke.

Table 1 characterises the scale of the episode in normative and relative terms. In terms of WHO guidelines, the highest concentrations were observed for NO_2, followed by black smoke and SO_2. Relative to the December average, however, the increase in black smoke was similar to that of NO_2. In historical terms, NO_2 levels were the highest ever recorded, while levels of black smoke and SO_2 were far

lower than at the time of the 1952 smog. Peak 24 hour concentrations of black smoke recorded at 11 sites varied from 15 μg/m³ (Islington) to 228 μg/m³ (Ilford), with a median of 148 μg/m³ (Westminster), but no clear difference was observed between inner and outer London. None of the control weeks for London or the control areas experienced levels of pollution approaching those observed in the episode.

TEMPERATURE

Temperatures were unseasonably low at the time of the episode (fig 1) and the mean and minimum temperatures were similar in the episode and previous weeks. None of the control weeks in 1987–90 were nearly as cold. There was a considerable temperature gradient within London due to the heat island effect, with inner London (Holborn) being about 4°C warmer than outer London (Kew, Greenwich, Heath-

row). Temperatures in outer London were similar to the rest of the country.

MORTALITY IN LONDON

Daily mortality from all causes (excluding accidents) and all respiratory conditions for the 10 weeks around the episode week for 1991 and the four control years is shown in fig 2. In all years there is an increasing number of deaths during December but the dominant feature is the increase associated with the influenza epidemic in 1989–90. Inspection of the 1991 plot shows a small peak late in the episode week which was the highest number of daily deaths in the entire period. A similar but more pronounced pattern is seen for respiratory mortality. The numbers of deaths in the episode week in London were: all causes (1544); all respiratory (243); respiratory infections (112); obstructive lung diseases (110); cardiovascular (699).

Table 2   Summary of mortality results for London: relative risks (95% confidence intervals) obtained from the log linear analysis† and relative excess risks for London compared with the control areas

Disease/age group	Relative risk in the episode week	Relative excess risk compared with control areas		
		England	South east	Manchester
All causes excl. injury and poisoning				
All ages	1·10 (1·02 to 1·19)*	1·07 (0·98 to 1·17)	1·09 (0·98 to 1·21)	1·13 (0·98 to 1·32)
0–64	1·21 (0·99 to 1·49)	1·15 (0·92 to 1·43)	1·15 (0·87 to 1·51)	1·17 (0·80 to 1·70)
65+	1·08 (0·99 to 1·18)	1·06 (0·96 to 1·16)	1·08 (0·96 to 1·20)	1·13 (0·96 to 1·33)
All respiratory				
All ages	1·22 (0·98 to 1·51)	1·25 (0·99 to 1·57)	1·18 (0·89 to 1·57)	1·84 (1·23 to 2·75)**
65+	1·19 (0·95 to 1·49)	1·23 (0·96 to 1·57)	1·15 (0·86 to 1·55)	1·89 (1·23 to 2·90)**
Respiratory infections				
All ages	1·23 (0·89 to 1·60)	1·26 (0·89 to 1·77)	1·15 (0·76 to 1·75)	1·84 (0·98 to 3·48)
65+	1·24 (0·89 to 1·73)	1·29 (0·90 to 1·85)	1·14 (0·74 to 1·76)	1·97 (1·02 to 3·83)*
Obstructive lung diseases (chronic obstructive pulmonary disease + asthma)				
All ages	1·23 (0·90 to 1·68)	1·22 (0·87 to 1·71)	1·20 (0·79 to 1·83)	1·82 (1·03 to 3·21)*
65+	1·19 (0·86 to 1·65)	1·16 (0·81 to 1·65)	1·15 (0·74 to 1·78)	1·74 (0·94 to 3·24)
Cardiovascular disease				
All ages	1·14 (1·01 to 1·28)*	1·08 (0·95 to 1·23)	1·07 (0·92 to 1·25)	1·15 (0·92 to 1·42)
0–64	1·04 (0·72 to 1·51)	0·95 (0·64 to 1·41)	0·89 (0·55 to 1·45)	1·12 (0·60 to 2·09)
65+	1·15 (1·02 to 1·31)*	1·10 (0·96 to 1·26)	1·10 (0·94 to 1·30)	1·15 (0·92 to 1·45)
Ischaemic heart disease				
All ages	1·18 (1·00 to 1·39)*	1·09 (0·92 to 1·29)	1·10 (0·89 to 1·35)	0·95 (0·84 to 1·07)
0–64	0·99 (0·64 to 1·54)	0·85 (0·53 to 1·35)	0·77 (0·43 to 1·38)	1·07 (0·50 to 2·27)
65+	1·22 (1·03 to 1·45)*	1·14 (0·95 to 1·38)	1·16 (0·93 to 1·45)	1·07 (0·79 to 1·45)

* p<0·05; ** p<0·01.
† Based on the log linear model using deaths in the weeks beginning 5 and 12 December for London only in the years 1987–91.

Table 3   Summary of hospital admissions results for London: relative risks (95% confidence intervals) obtained from the log linear analysis† and relative excess risks for London compared with the control areas

Disease/age group	Relative risk in the episode week	Relative excess risk compared with control areas		
		England	South east	Manchester
All respiratory				
All ages	1·04 (0·96 to 1·13)	0·98 (0·90 to 1·07)	1·06 (0·95 to 1·19)	0·99 (0·87 to 1·13)
0–14	1·01 (0·89 to 1·14)	1·00 (0·87 to 1·14)	1·04 (0·88 to 1·22)	1·11 (0·92 to 1·35)
15–64	0·95 (0·80 to 1·14)	0·87 (0·72 to 1·05)	0·95 (0·74 to 1·22)	0·78 (0·57 to 1·07)
65+	1·19 (1·04 to 1·37)*	1·03 (0·89 to 1·19)	1·15 (0·95 to 1·39)	0·89 (0·70 to 1·13)
Obstructive lung diseases (chronic obstructive pulmonary disease + asthma)				
All ages	1·14 (1·00 to 1·30)*	1·05 (0·91 to 1·21)	1·12 (0·93 to 1·35)	0·99 (0·80 to 1·23)
0–14	1·04 (0·82 to 1·31)	1·07 (0·83 to 1·37)	1·04 (0·76 to 1·43)	1·05 (0·72 to 1·54)
15–64	0·98 (0·77 to 1·25)	0·87 (0·67 to 1·13)	0·99 (0·70 to 1·40)	0·70 (0·47 to 1·05)
65+	1·43 (1·15 to 1·77)**	1·20 (0·95 to 1·51)	1·30 (0·96 to 1·77)	1·20 (0·84 to 1·73)
Chronic obstructive pulmonary disease				
All ages	1·27 (1·03 to 1·55)*	1·09 (0·87 to 1·35)	1·31 (0·98 to 1·74)	1·05 (0·75 to 1·47)
15–64	0·99 (0·61 to 1·60)	0·83 (0·49 to 1·39)	1·25 (0·64 to 2·47)	0·56 (0·26 to 1·21)
65+	1·36 (1·08 to 1·71)**	1·18 (0·92 to 1·52)	1·31 (0·94 to 1·82)	1·26 (0·85 to 1·85)
Asthma				
All ages	1·08 (0·91 to 1·28)	1·03 (0·85 to 1·24)	1·01 (0·79 to 1·29)	0·95 (0·71 to 1·27)
0–14	1·05 (0·83 to 1·33)	1·10 (0·85 to 1·43)	1·02 (0·73 to 1·42)	1·11 (0·75 to 1·66)
15–64	0·98 (0·75 to 1·30)	0·88 (0·65 to 1·20)	0·91 (0·61 to 1·36)	0·75 (0·46 to 1·23)
65+	1·97 (1·10 to 3·53)*	1·30 (0·69 to 2·45)	1·25 (0·54 to 2·90)	0·91 (0·34 to 2·43)
Ischaemic heart disease				
All ages	1·04 (0·90 to 1·21)	0·99 (0·84 to 1·17)	1·05 (0·86 to 1·29)	0·95 (0·74 to 1·22)
15–64	1·03 (0·80 to 1·33)	1·02 (0·78 to 1·34)	1·13 (0·80 to 1·59)	1·28 (0·84 to 1·94)
65+	1·04 (0·87 to 1·26)	0·97 (0·80 to 1·19)	1·01 (0·79 to 1·30)	0·80 (0·59 to 1·10)

* p<0·05; ** p<0·01.
† Based on the log linear model using admissions in the weeks beginning 5 and 12 December for London only in the years 1987–91.

The results of the log linear analysis are shown in table 2. Relative risks were increased for all age/diagnostic categories except for ischaemic heart disease: age 0–64 (0·99). Risks with 95% confidence intervals (95% CI) not including unity were observed for all causes: all ages (1·10), cardiovascular: all ages (1·14), age 65+ (1·15), and ischaemic heart disease: all ages (1·18), age 65+ (1·22). The relative risk for all respiratory: all ages was increased (1·22) and significant at the 10% level (95% CI 0·98 to 1·51). Though not significant, the highest relative risks tended to be for respiratory causes of death.

LONDON VERSUS CONTROL AREAS

The log linear analysis was repeated for each control area, after including the London data, to test the hypothesis that the number of deaths in London during the episode week was significantly different from that predicted on the basis of the control weeks, years, and areas. The results are shown in table 2. Of 36 risk estimates only four were less than unity, and all of these were for cardiovascular disease or ischaemic heart disease. However, no significantly increased risks were observed in the comparison with England or with the south east. In comparison with Manchester significantly increased risks were observed for all respiratory: all ages (1·84), age 65+ (1·89), and obstructive lung diseases: all ages (1·82). These were attributable more to a decline in respiratory mortality in Manchester (RR 0·66) than to an increase in London.[5] Depending on the control area, the estimate of the number of excess deaths in London during the episode from all causes ranged from 101 to 178, for all respiratory deaths from 37 to 109, and for all cardiovascular from 46 to 91.

IMMEDIATE HOSPITAL ADMISSIONS IN LONDON

Daily immediate hospital admissions to London hospitals for all respiratory diagnoses and for asthma for the 10 weeks surrounding the episode week, and the corresponding periods of the previous four years, are shown in fig 3 for all ages. A small peak in all respiratory admissions is visible in the episode week but not for asthma admissions. The numbers of emergency admissions in the episode week in London were: all respiratory diagnoses (1608); obstructive lung diseases (633); chronic obstructive pulmonary disease (280); asthma (353); and ischaemic heart disease (478).

The results of the log linear analysis of the London data are presented in table 3. All the relative risks except for the 15–64 age group were greater than unity and significantly increased relative risks were observed for all respiratory diagnoses: age 65+ (1·19). Significant increases were also observed for obstructive lung diseases: all ages (1·14) and age 65+ (1·43). In the age 65+ age group, risks were significantly increased for both chronic obstructive pulmonary disease (1·36) and asthma (1·97). For all ages the relative risks for respiratory diagnoses (1·04) and for ischaemic

heart disease (1·04) were increased but not significantly.

The relative risks observed in London were compared with those for the other areas and confidence limits were calculated (table 3). Compared with England the risks were not significant or noticeably high. Compared with the south east, more of the risks were increased and two (obstructive lung diseases: age 65+ (1·30) and chronic obstructive pulmonary disease: all ages (1·31)) were significant at the 10% level. Compared with Manchester there was little evidence of major differences and no risks were statistically significant. Depending on the control area, estimates of excess/reduced admissions for all respiratory admissions in London during the episode week ranged from −33 to 91, for obstructive lung diseases from −6 to 77, and for ischaemic heart disease from −25 to 23.

Discussion

Effects of the episode on daily mortality and admissions were not easily discerned by inspecting the time series plots and the evidence for adverse health consequences rests on the results of the log linear analysis. These generally indicate that, during the week of the 1991 air pollution episode, London experienced more deaths and hospital admissions than would have been predicted from data for control weeks, years, and areas. The increased relative risks were most consistent for mortality (all causes, respiratory, and cardiovascular) and for admissions for respiratory disease in older age groups. While only a few tests were statistically significant, it is notable that most of the risks were greater than unity, the value we would expect if there were no effect. Although chance remains one explanation, we feel that the evidence indicates that there may have been a real increase in health events at the time.

It is possible that publicity about the episode and its possible health effects could have influenced hospital admissions and we have no way of excluding this explanation. Where the effects of publicity have been investigated, no effects have been observed[9]; furthermore, this factor is unlikely to have affected mortality which was increased to a greater extent than admissions.

Analysis of a single episode is vulnerable to the coincidental occurrence of an increase in respiratory infections for reasons unrelated to air pollution. Influenza was not known to be present at the time of the episode, and the effects due to the annual increase in respiratory syncytial virus are unlikely since these tend to occur in January. However, many other respiratory viruses could be responsible and it is known that respiratory consultations in general practice were increasing nationally at the time (Dr D Fleming, personal communication). This explanation is less likely to account for the differences between London and the comparison areas, unless any epidemic occurred there at a different time. The occurrence of an influenza epidemic in one of the control years (1989) might have affected the resulting relative

risks, but removal of this year from the analysis made little difference to the estimates of effect.

The excess mortality and admissions in London could have been caused by the prevailing cold weather,[10-13] though it should be noted that central London was warmer than elsewhere. However, while comparisons of London with the rest of the south east and the rest of England resulted in some reduction (and loss of significance) in the relative risks, these tended to remain increased; comparing London with England, the risks for all respiratory deaths (all ages) were very close to significance (1·25, 95% CI 0·99 to 1·57).

Air pollution is a plausible explanation for the increase in health events during the episode. There is good evidence that major air pollution episodes increase mortality and morbidity,[1 14] and time-series studies show associations between daily health events and levels of air pollution which are within current British and international standards. The strongest and most consistent evidence relates to particles.[15 16] The size of health effect observed in this episode was close to that predicted by meta-analysis of particulate studies,[15 16] which suggests that the increase in levels of black smoke which occurred during the episode could explain some of the observed effects.

It is plausible that even a small effect of air pollution on lung function might be capable of causing the admission or death of individuals who are already on the brink of such an event.[17 18] This is consistent with our observation that relative risks were greatest among the elderly.

There is a widespread belief that air pollution episodes, including this one, are associated with an increased incidence of asthma attacks, particularly among children. Adverse effects on lung function in children have been detected during episodes of particulate pollution equivalent to that occurring in London in 1991,[19-21] and recent chamber studies have suggested that NO_2 exposure, at levels encountered during the 1991 episode, may potentiate the bronchoconstrictor response to common aeroallergens.[22 23] The clinical and public health significance of these effects remains uncertain. Our findings do not support contemporaneous reports of an epidemic of paediatric asthma admissions during the episode, and suggest that asthma is not the disease outcome most sensitive to short term air pollution episodes of this type.

We conclude that air pollution is a plausible explanation for the increase in mortality and hospital admissions which occurred in the week of the episode. Although this episode is notable for the historically high level of NO_2, the associated increase in black smoke – though small compared with previous decades – could have contributed at least in part to the increase in mortality and admissions.

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