rate ratio at level z is estimated by $\hat{\lambda} = \hat{\beta}_1 z + \hat{\beta}_2 S(z)$ and the standard error ($\lambda$) is estimated from the covariance matrix of $\hat{\beta}_1$ and $\hat{\beta}_2$. A 95% confidence interval for $\lambda$ is then $\hat{\lambda} \pm SE(\hat{\lambda})\sqrt{\chi^2_2}$, where $\chi^2_2$ is the 95th centile of the $\chi^2$ 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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See p 589, 661, 676


## 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 μg/m³) 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.**



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## 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-

tality and levels of air pollution within the range experienced by London in recent years.[11-13] Currently no such evidence exists for any city in Britain. We report the results of an analysis of air pollution and daily mortality for London during 1987-92. The work forms part of a collaborative European project (Air Pollution and Health, a European Approach (APHEA)).[14]

## Methods

For Greater London during April 1987 to March 1992, we constructed time series of daily counts of all age mortality for all causes of death, excluding accidents—that is, codes <900 according to the international classification of diseases, ninth revision—and for respiratory and cardiovascular causes (codes 460-519 and 390-459 respectively).

We calculated the mean 24 hour temperature and humidity from data from central London (Holborn). Details of the air pollution monitoring methods and network may be found elsewhere.[8,15] We measured ozone concentrations (eight hour average 9 am to 5 pm and maximum one hour average) by the ultraviolet absorption technique at a single background monitor situated in central London near Victoria station. We measured nitrogen dioxide concentrations (daily average and maximum one hour average) by the chemiluminescence technique at the same monitoring site at Victoria and at another urban background site in west London (Earl's Court). We obtained daily average concentrations of black smoke (smoke stain method) and sulphur dioxide (acidimetric bubbler system) from four sites in central, north, northeast, and south London. If only one measuring station provided data for a day, we regarded this day as having no data. If up to two stations did not provide data, we used a regression technique to obtain an estimate of the average values across the four stations on each day.[16] Table 1 shows summary statistics for the various pollutants and meteorological variables used in the analysis.

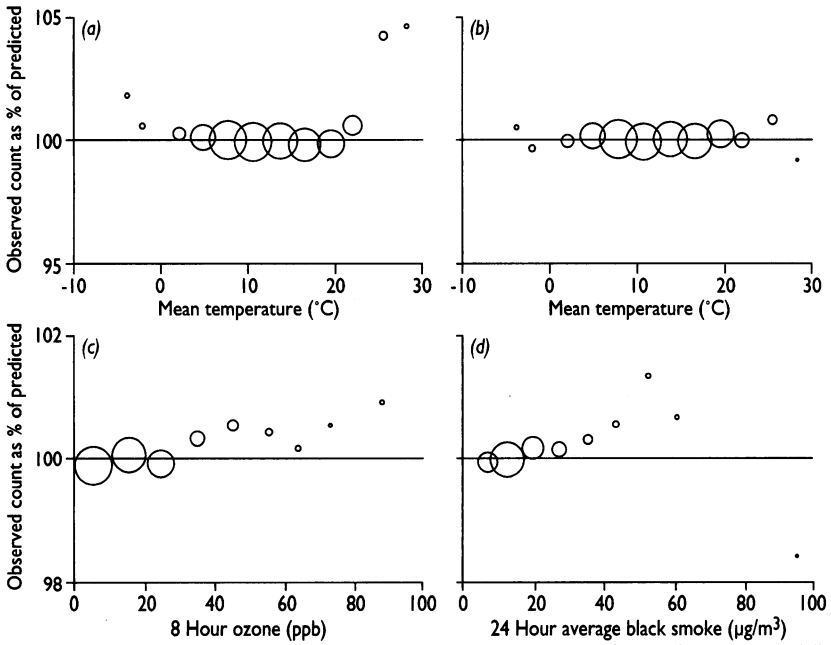


**Fig 1**—*Relation between daily mortality and temperature (a) before adjustment and (b) after three piece linear adjustment for temperature and other confounding factors. Relation between daily mortality and (c) eight hour ozone and (d) 24 hour black smoke, with adjustment for all confounders including temperature. The y axis shows the mean count as percentage of that predicted from the model. The position of the bubble in relation to the x axis corresponds to the mean for that range, and the area of the bubble represents the number of days*

### STATISTICAL ANALYSIS

The analysis followed the collaborative project's approach,[14] which is based on statistical methods developed by Zeger[17] and was applied to the analysis of air pollution and daily health outcomes by Schwartz and other workers.[18-23] In brief, the daily number of deaths was used as the outcome variable in autoregressive log-linear regression models with Poisson errors implemented in SAS.[24] A program to deal with overdispersion in the Poisson model was obtained (Joel Schwartz, personal communication) and is available on request. Before examining the short term associations between air pollution and mortality, we had to correct for confounding by other factors that may also be related to both these variables. Regression models adjusted for time trends and cyclical (such as seasonal) variations with sine and cosine terms. We included dummy variables for each day of the week, bank holidays, and for the period of the 1989-90 influenza epidemic. We checked the effects of these adjustments using standard diagnostic methods, such as periodograms and residual plots (available on request). Adjustment for temperature is very important,[25,26] and after several models were tried—including the use of dummy variables—the U shaped relation between daily temperature and mortality (fig 1a), was most successfully adjusted by fitting three separate linear terms, one for <5°C, one for <20°C, and one for 5-20°C (fig 1b). Single linear terms were used to adjust for relative humidity.

The final step in developing the core model was to allow for any remaining tendency for autocorrelation (the dependence of one day on a previous day(s)); autocorrelation function plots are available on request. Figure 2 shows the resulting fitted values and residuals. This confirms that seasonal cycles have been removed, together with the effects of the influenza epidemic, leaving only short term fluctuations to be

**Table 1**—*Summary data for daily deaths and meteorological and air pollution variables*

| Variable | Mean (SD) | Centile | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Minimum | 5th | 10th | 25th | 50th | 75th | 90th | 95th | Maximum |
| All deaths (No/day) | 175·5 (27·0) | 117 | 139 | 146 | 157 | 172 | 189 | 209 | 221 | 357 |
| Respiratory deaths (No/day) | 22·5 (9·9) | 5 | 11 | 13 | 16 | 21 | 26 | 34 | 39 | 103 |
| Cardiovascular deaths (No/day) | 78·6 (13·7) | 42 | 58 | 62 | 69 | 77 | 87 | 96 | 102 | 143 |
| Mean temperature (°C) | 12·3 (5·2) | −5 | 4 | 6 | 8 | 12 | 16 | 20 | 21 | 29 |
| Relative humidity (%) | 72·1 (10·2) | 40 | 54 | 58 | 65 | 72 | 80 | 86 | 89 | 96 |
| Ozone (ppb): | | | | | | | | | | |
| 8 h average | 15·5 (10·9) | 1 | 2 | 4 | 8 | 14 | 21 | 28 | 36 | 74 |
| Maximum 1 h | 20·6 (13·2) | 0 | 3 | 5 | 11 | 20 | 27 | 36 | 45 | 94 |
| Nitrogen dioxide (ppb): | | | | | | | | | | |
| 24 h average | 37·2 (12·3) | 14 | 22 | 25 | 30 | 36 | 42 | 50 | 58 | 182 |
| Maximum 1 h | 57·2 (23·0) | 21 | 35 | 38 | 44 | 52 | 64 | 81 | 98 | 370 |
| Black smoke (24 h average) (μg/m³) | 14·6 (7·0) | 3 | 7 | 8 | 10 | 13 | 18 | 22 | 26 | 95 |
| Sulphur dioxide (24 h average) (μg/m³) | 32·0 (11·7) | 9 | 16 | 18 | 24 | 31 | 38 | 46 | 52 | 100 |

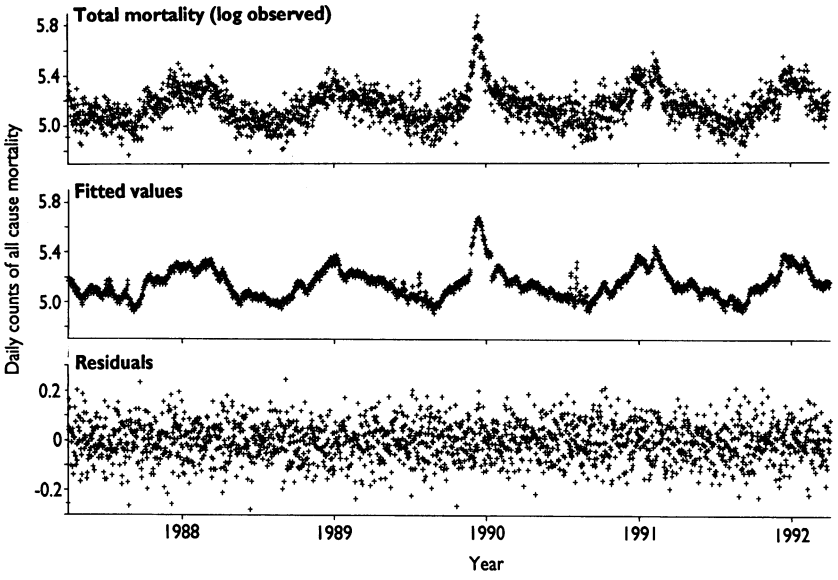


**Fig 2**—*Time series of daily counts of all cause mortality ($log_e$) as observed and after fitting of core model, which includes all confounding factors. The residuals are the difference between fitted and observed values*

**Table 2**—*Percentage increase (95% confidence intervals) in daily all cause, cardiovascular, and respiratory mortality associated with increase in pollutant level from 10th to 90th centile. Results are for whole year and for cool and warm seasons separately using the single day lag associated with the largest effect*†

| | All cause | Cardiovascular | Respiratory |
|---|---|---|---|
| Ozone: | | | |
| 8 h average: | lag 0 | lag 0 | lag 0 |
| All year (3-29) | 2·43 (1·11 to 3·76)*** | 1·44 (−0·45 to 3·36) | 6·03 (2·22 to 9·99)*** |
| Cool season (2-22) | 0·77 (−0·88 to 2·44) | −1·69 (−3·99 to 0·68) | 6·20 (1·67 to 10·94)** |
| Warm season (7-36) | 3·48 (1·73 to 5·26)*** | 3·55 (1·04 to 6·13)** | 5·41 (0·35 to 10·73)* |
| Maximum 1 h: | lag 0 | lag 0 | lag 0 |
| All year (4-35) | 2·59 (1·30 to 3·89)*** | 2·19 (0·34 to 4·08)* | 6·42 (2·63 to 10·35)*** |
| Cool season (2-28) | 0·99 (−0·80 to 2·81) | −1·37 (−3·84 to 1·16) | 6·49 (1·54 to 11·69)** |
| Warm season (11-45) | 3·49 (1·81 to 5·20)*** | 4·37 (1·96 to 6·85)*** | 6·21 (1·29 to 11·38)* |
| Nitrogen dioxide: | | | |
| 24 h average: | lag 1 | lag 0 | lag 1 |
| All year (24-51) | 0·75 (−0·08 to 1·60) | 0·62 (−0·58 to 1·84) | −0·92 (−3·22 to 1·33) |
| Cool season (25-49) | 0·46 (−0·44 to 1·36) | −0·11 (−1·38 to 1·17) | −0·25 (−2·54 to 2·10) |
| Warm season (23-53) | 1·45 (−0·25 to 3·17) | 2·54 (0·18 to 4·96)* | −2·90 (−7·55 to 1·99) |
| Maximum 1 h: | lag 0 | lag 0 | lag 2 |
| All year (37-83) | 0·43 (−0·30 to 1·16) | 0·66 (−0·37 to 1·70) | −1·44 (−3·27 to 0·41) |
| Cool season (38-77) | −0·05 (−0·78 to 0·69) | −0·14 (−1·17 to 0·91) | −0·44 (−2·26 to 1·40) |
| Warm season (35-89) | 1·73 (−0·20 to 3·29)* | 2·96 (0·80 to 5·17)** | −4·54 (−8·52 to −0·38)* |
| Black smoke (24 h average): | lag 1 | lag 1 | lag 1 |
| All year (8-23) | 1·70 (0·82 to 2·58)*** | 0·58 (−0·68 to 1·85) | 0·66 (−1·62 to 2·99) |
| Cool season (9-26) | 1·56 (0·45 to 2·67)** | 0·13 (−1·46 to 1·74) | 0·76 (−2·05 to 3·64) |
| Warm season (7-19) | 2·45 (0·88 to 4·05)** | 1·87 (−0·34 to 4·13) | 0·64 (−3·80 to 5·29) |
| Sulphur dioxide (24 h average): | lag 1 | lag 1 | lag 1 |
| All year (18-46) | 0·95 (−0·01 to 1·91) | 0·22 (−1·35 to 1·81) | 1·68 (−1·31 to 4·76) |
| Cool season (18-49) | 0·69 (−0·64 to 2·03) | 0·22 (−1·83 to 2·32) | 1·84 (−1·98 to 5·82) |
| Warm season (18-43) | 1·52 (0·08 to 2·98)** | 0·44 (−1·68 to 2·61) | 1·84 (−2·57 to 6·46) |

*P<0·05; **P<0·01; ***P<0·001.
†Relative risk may be obtained by dividing % increase by 100 and adding one. The natural logarithm of relative risk divided by number of units of air pollution between 10th and 90th centile will result in original regression coefficient from Poisson model.

analysed in relation to pollution levels. We examined the effects of each pollutant by adding them to the "core" autoregressive log-linear Poisson model described above. We examined untransformed pollutant levels on the same day (lag 0) and lagged by 1 and 2 days. We performed analyses for the whole year and for the "warm" (April to September) and "cool" (October to March) seasons separately.

## Results

Table 2 shows the estimated effects of air pollution for the single day lag that gave the most significant result. Ozone concentrations recorded on the same day (lag 0) showed the most consistent association with daily mortality. For all cause and cardiovascular mortality the effects were significant only in the warm season. The strongest ozone effect was observed for respiratory mortality, with similar sizes of effect in both the warm and the cool seasons. For eight hour ozone concentrations (lag 0) in the warm period, an increase from the 10th to the 90th centile was associated with an increase of 3·5%, 3·6%, and 5·4% in all cause, cardiovascular, and respiratory mortality respectively. The bubble plot of residuals suggests that a threshold may exist at around 50 ppb for eight hour ozone concentrations (fig 1c).

Black smoke (lag 1) was associated with significant effects on all cause mortality for all the year and for the two seasons. Relative risks for cardiovascular and respiratory mortality were all greater than unity but were smaller than those for total mortality and were non-significant. In the warm season, an increase in black smoke from the 10th to 90th centiles was associated with an increase of 2·5% in all cause mortality. The bubble plot (fig 1d) suggests that there is no threshold of effect.

For nitrogen dioxide (1 hour maximum), small but significant positive associations were observed for all cause (lag 1) and cardiovascular (lag 1) mortality, and a negative effect was seen for respiratory mortality (lag 2), all in the warm season. A significant association between sulphur dioxide (lag 1) and all cause mortality was observed in the warm season.

To interpret these results it is necessary to take into account the fact that these pollutants covary in a complex way (table 3). Ozone, for example, during the warm season is positively associated with nitrogen dioxide and sulphur dioxide but in the cool season is negatively associated with these two pollutants and also with black smoke. In the warm season, little or no correlation exists between ozone and black smoke.

Table 4 shows two pollutant models including ozone and black smoke. For the warm season, inclusion of one pollutant made little difference to the effect of the other, which suggests that confounding is most unlikely. For the cool season, the effects of black smoke on all cause mortality were doubled when ozone was included in the model. The effects of both black smoke and ozone remained significant after inclusion of nitrogen dioxide or sulphur dioxide in the model (data not shown).

The effects of nitrogen dioxide on total mortality in the warm season became non-significant when ozone or sulphur dioxide were included in the model but were little affected by inclusion of black smoke (data not shown). The effects of sulphur dioxide on total mortality in the warm season remained significant after inclusion of ozone in the model but not after inclusion of black smoke or nitrogen dioxide.

## Discussion

We found associations between daily mortality in Greater London and various indicators of air pollution. The strongest association was with ozone (lag 0), followed by black smoke (lag 1), and these associations were independent of the effects of other pollutants. Several associations were also observed with nitrogen dioxide and sulphur dioxide, but these were partly explained by their correlation with ozone or black smoke. More associations were observed in the warm than in the cool season.

The key feature of the statistical approach was the removal of any relations that would be caused by longer term or cyclical associations between the pollutant and mortality, influenza epidemics, meteorological variables, or serial correlation. The robustness of the

**Table 3**—*Correlation matrix for air pollution and meteorological indicators, based on whole year and cool and warm seasons*

| | Relative humidity | Ozone (max 8 h) | Ozone (max 1 h) | Nitrogen dioxide (24 h) | Nitrogen dioxide (max 1 h) | Black smoke (24 h) | Sulphur dioxide (24 h) |
|---|---|---|---|---|---|---|---|
| Temperature: | | | | | | | |
| Whole year | −0·340 | 0·529 | 0·541 | −0·068 | −0·005 | −0·262 | −0·075 |
| Cool season | −0·061 | 0·271 | 0·297 | −0·264 | −0·208 | −0·293 | −0·263 |
| Warm season | −0·235 | 0·425 | 0·414 | 0·136 | 0·129 | 0·221 | 0·213 |
| Relative humidity: | | | | | | | |
| Whole year | | −0·472 | −0·484 | −0·096 | −0·113 | 0·140 | −0·048 |
| Cool season | | −0·264 | −0·297 | 0·038 | 0·041 | 0·119 | 0·054 |
| Warm season | | −0·470 | −0·467 | −0·288 | −0·279 | −0·082 | −0·233 |
| Ozone (8 h): | | | | | | | |
| Whole year | | | 0·966 | 0·005 | 0·117 | −0·291 | −0·070 |
| Cool season | | | 0·961 | −0·437 | −0·270 | −0·461 | −0·441 |
| Warm season | | | 0·958 | 0·290 | 0·328 | 0·001 | 0·199 |
| Ozone (max 1 h): | | | | | | | |
| Whole year | | | | 0·063 | 0·169 | −0·290 | −0·098 |
| Cool season | | | | −0·401 | −0·242 | −0·478 | −0·477 |
| Warm season | | | | 0·358 | 0·391 | 0·047 | 0·209 |
| Nitrogen dioxide (24 h): | | | | | | | |
| Whole year | | | | | 0·913 | 0·639 | 0·447 |
| Cool season | | | | | 0·942 | 0·765 | 0·453 |
| Warm season | | | | | 0·875 | 0·464 | 0·439 |
| Nitrogen dioxide (max 1 h): | | | | | | | |
| Whole year | | | | | | 0·542 | 0·390 |
| Cool season | | | | | | 0·716 | 0·416 |
| Warm season | | | | | | 0·309 | 0·364 |
| Black smoke (24 h): | | | | | | | |
| Whole year | | | | | | | 0·445 |
| Cool season | | | | | | | 0·501 |
| Warm season | | | | | | | 0·340 |

**Table 4**—*Effects of ozone and black smoke on daily mortaility before and after inclusion of other pollutant in the model. Percentage increase (95% confidence intervals) for an increase in pollutant from 10th to 90th centile*†

| | All year | Cool | Warm |
|---|---|---|---|
| All cause: | | | |
| Ozone 8 h | 2·43 (1·11 to 3·76)*** | 0·77 (−0·88 to 2·44) | 3·48 (1·73 to 5·26)*** |
| Ozone 8 h plus black smoke | 2·74 (1·42 to 4·06)*** | 1·43 (−0·24 to 3·13) | 3·49 (1·75 to 5·25)*** |
| Black smoke | 1·70 (0·82 to 2·58)*** | 1·56 (0·45 to 2·67)*** | 2·45 (0·88 to 4·05)** |
| Black smoke plus ozone 8 h | 2·80 (1·71 to 3·92)*** | 2·97 (1·54 to 4·41)*** | 2·47 (0·85 to 4·13)** |
| Cardiovascular: | | | |
| Ozone 8 h | 1·44 (−0·45 to 3·36) | −1·69 (−3·99 to 0·68) | 3·55 (1·04 to 6·13)** |
| Ozone 8 h plus black smoke | 1·61 (−0·29 to 3·55) | −1·58 (−3·94 to 0·84) | 3·61 (1·10 to 6·18)** |
| Black smoke | 0·58 (−0·68 to 1·85) | 0·13 (−1·46 to 1·74) | 1·87 (−0·34 to 4·13) |
| Black smoke plus ozone 8 h | 1·18 (−0·38 to 2·75) | 0·24 (−1·83 to 2·35) | 1·84 (−0·45 to 4·19) |
| Respiratory: | | | |
| Ozone 8 h | 6·03 (2·22 to 9·99)*** | 6·20 (1·67 to 0·94)** | 5·41 (0·35 to 10·73)* |
| Ozone 8 h plus black smoke | 6·25 (2·40 to 10·24)** | 6·78 (2·13 to 11·64)** | 5·36 (0·30 to 10·69)* |
| Black smoke | 0·66 (−1·62 to 2·99) | 0·76 (−2·05 to 3·64) | 0·64 (−3·80 to 5·29) |
| Black smoke plus ozone 8 h | 1·66 (−1·23 to 4·63) | 2·21 (−1·53 to 6·10) | 1·43 (−3·35 to 6·33) |

*P<0·05; **P<0·01; ***P<0·001. †See Table 2 for ranges of pollutants.

method has been questioned [27], but other studies have shown that when the method is carried out with due attention to statistical diagnostics, the findings do not differ substantially from those obtained by linear regression or non-parametric methods.[28–31]

The number of sites at which air pollution was continuously monitored during this period was limited, which makes it likely that exposure of the population on a particular day may have been imprecisely measured. Our analysis, however, relied on day to day fluctuations in pollution levels which are more likely to vary in the same direction even if the absolute concentrations are different. Where more than one monitor was used, there was a significant correlation between them for daily concentrations of pollutant. Because ozone is scavenged by nitric oxide emitted by traffic[8] the eight hour ozone concentrations recorded in central London are likely to underestimate those that occur in suburban areas by about 10-15 ppb.

### PARTICLES AND MORTALITY

While it is accepted that major smog episodes such as in London in 1952 have adverse health effects, it is more difficult to believe that the current historically low pollution levels could have even a small effect. Nevertheless, plausible mechanisms have been postulated that might explain a toxic effect on the lung of low concentrations of inhaled particles,[32] and such effects might be sufficient to precipitate the death of persons rendered vulnerable by pre-existing conditions such as advanced cardiorespiratory disease. The relation with all cause mortality (1·1% increase for 10 $\mu g/m^3$ increase in black smoke) was similar to that observed in a number of North American studies in cities where the sources of particles are somewhat different from sources in London.[12,13] However, contrary to most other studies[11] we did not observe relatively greater effects of particles on respiratory and cardiovascular mortality than on all cause mortality.

The British Standard technique for sampling black smoke used throughout Britain measures strongly light absorbing suspended particulate matter, primarily in the size range of 0·1-5 μm. In London, most of this size fraction will derive from diesel vehicle emissions. Other particles of a chemical nature, such as acid aerosols, sulphates, and nitrates, may be an important component of the particle fraction but will be underestimated by this method.[33] The more recently introduced technique of measuring all particles with a mean aerodynamic diameter of $<10$ μm ($PM_{10}$) is regarded as a better measure, and we plan further analyses of more recent data using this indicator.

### OZONE AND MORTALITY

The few studies that have looked at ozone and mortality are less consistent than those for particles.[34] Significant effects have been reported for Los Angeles and New York[35,36] but not for several other cities in the United States.[34] Considerable evidence exists, however, that ozone is associated with daily admissions and emergency room attendances for respiratory disease and with reductions in lung function.[34,37] Our own

## Key messages

- Air pollution in London is now mainly due to emissions from vehicles, and levels are generally well below the World Health Organisation's guidelines
- Evidence from other countries suggests that similar levels of pollution may be associated with short term health effects
- This study suggests that air pollution due to particles and ozone may be associated with increased daily mortality in London
- The evidence is less convincing for nitrogen dioxide and sulphur dioxide
- It would be prudent to assume that these associations are causal and to reduce air pollution levels with the help of appropriate abatement policies

studies of London during the same period show a significant effect of ozone (lag 1 day) on hospital admissions.[38] Good evidence exists that ozone may be toxic to the lung at concentrations encountered in London,[37 39 40] and, as for black smoke, it is plausible that exposure could advance the time of death in an already ill person.

### IMPORTANCE OF FINDINGS

These results, if indicative of a causal relation, suggest that air pollution has a measurable effect on mortality. The extent to which this is mainly due to deaths of individuals who would have died in the very near future is unclear and cannot be addressed by this type of analysis. Perhaps the main public health implication of these results lies in the possibility that much of the population is currently experiencing levels of pollution that are capable of adversely affecting the lung. Recent evidence suggests that such exposure may be associated with long term as well as short term effects on mortality.[41] It would be prudent to assume that current levels of air pollution do have adverse health effects, and this should be taken into account in determining policies for reducing emissions of pollutants, standards for ambient concentrations, and advice to vulnerable groups.

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