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Abstract

Study objective – To investigate whether air pollution levels in London have short term effects on hospital admissions for respiratory disease.

Design – Poisson regression analysis of daily counts of hospital admissions, adjusting for effects of trend, seasonal and other cyclical factors, day of the week, holidays, influenza epidemic, temperature, humidity, and autocorrelation. Pollution variables were particulates (black smoke: BS), sulphur dioxide (SO₂), ozone (O₃), and nitrogen dioxide (NO₂), lagged 0–3 days.

Setting and patients – All immediate admissions for respiratory disease (ICD 460–519) to hospitals in London health districts in the five years April 1987 to February 1992 for all ages and the 0–14, 15–64, and 65+ age groups.

Main results – O₃ (lagged one day) was significantly associated with an increase in daily admissions among all age groups, except the 0–14 group, and this effect was stronger in the “warm” season (April–September). In this season, the relative risks of admission associated with an increase in 8 hour O₃ levels of 29 ppb (10th to 90th centile) were 1.0483 (95% CI 1.0246, 1.0726), 1.0294 (0.9930, 1.0672), 1.0751 (1.0354, 1.1163), and 1.0616 (1.0243, 1.1003) for all ages and age groups 0–14, 15–64, and 65+ respectively. Very few significant associations were observed with the other pollutants, though these tended to be positive. Controlling for other pollutants made little difference to the O₃ coefficients. There was evidence of a threshold at about 40–60 ppb O₃ (maximum hourly or maximum 8 hour).

Conclusions – O₃ levels in London have a small but significant effect on hospital admissions for respiratory disease at all ages. The possible role of aerallergen as a confounding factor needs to be examined. Unlike other cities where similar effects have been reported, little or no effect of particulates was observed in London.

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Since the notorious London smog of 1952, which demonstrated beyond doubt that air pollution episodes may have severe health effects,¹ levels of particulates and sulphur dioxide (SO₂) in London have fallen to historically low levels. The main sources of pollution have changed from coal burning to vehicle emissions, and the latter now account for an increasing share of emissions of particulates and oxides of nitrogen.¹² Over the past 20 years, London has experienced episodes of both O₃ and NO₂ pollution; the highest maximum hourly concentrations reached were 212 ppb for O₃ in 1976 and 423 ppb for NO₂ in 1991.

Evidence about the short term effects of air pollution on health in London is currently confined to a number of analyses of daily mortality up to 1972.¹³ These relate to the period of 1972 and black smoke (BS) pollution from coal burning and are consistent in finding that short term health effects were occurring at that time, though not consistent about the relative roles of BS, SO₂ and acid aerosols.⁷

Evidence from other countries, especially the USA, has led to concerned that current levels of particulate pollution in Britain, while low by historical standards, may still be associated with short term health effects,⁸⁹ and this is supported by a recent study of hospital admissions in Birmingham.¹¹ The 1991 episode of NO₂ and particulate air pollution in London was associated with an increase in mortality and hospital admissions consistent with effects predicted from time series studies of particulate pollution from other countries.¹²

There is an urgent need to establish whether current forms of air pollution in London are having adverse short term health effects and in this paper we present the results of an analysis of daily hospital admissions for respiratory disease in relation to the period between 1987–88 and 1991–92. This work is part of the European collaborative APHEA project (air pollution and health, a European approach).¹⁰

Methods

Greater London lies at a latitude of 52° N and enjoys a temperate maritime climate. Average rainfall is 612 mm and average temperatures in January and July are 5-5 and 18-1°C respectively. London occupies a roughly circular area in the Thames basin with a radius of about 45 km and an area of 1600 km². At the 1991 census, the population covered by London health authorities was 7.3 million. The dominant source of pollution is motor vehicles with contributions from power generation and industry in some areas.

Records of emergency admissions for respiratory diagnoses (ICD 460–519) to hospitals...
in the London health districts were obtained from the Hospital Episode System for England for the five year period April 1987–February 1992 (1796 days) (March 1992 was discarded because of an end of year deficit associated with data recording). Diagnosis is recorded at discharge and was available in 73%, 76%, 85%, 88%, and 95% of records for the years 1987/88–1991/92 respectively. Because coding of area of residence was incomplete in earlier years, the district of the hospital of treatment was used to define eligible admissions. Daily counts of respiratory admissions were constructed for all ages, 0–14, 15–64, and 65+ year age groups. Maximum and minimum temperature and relative humidity for 6.00 am and 3.00 pm were obtained from a station in central London (Holborn) and an estimate of daily average levels was obtained by averaging these two values. Details of the air pollution monitoring network may be found elsewhere.\textsuperscript{14} \textsuperscript{15} \textsuperscript{16} O\textsubscript{3}, NO\textsubscript{x} concentrations (8 hour from 9.00 am to 5.00 pm, and maximum 1 hour for each 24 hour period) were measured by the ultraviolet absorption technique at a single urban background monitor situated 5 m above ground level in a back street near Victoria Station in central London. This was the only one available for the whole period under consideration but levels correlated very highly (r>0.95) with monitors subsequently set up in inner London (Bloomsbury) and outer London (Bexley). NO\textsubscript{x} concentrations (daily average and maximum 1 hour) were measured by the chemiluminescence technique at the same monitoring site at Victoria and at another in west London (Earl’s Court) situated at 7 m above ground level in a partly residential area bounded by major roads. The two were highly correlated and their average was used in the analysis. Daily average levels of BS (smoke stain method) and SO\textsubscript{2} (acidimetric bubbler system) were obtained for the four monitors, with data for more than 75% of days. There were: London City (central London), Enfield (north London), Croydon (south London), and Ilford (north east London). Missing BS and SO\textsubscript{2} values were replaced by a regression technique proposed by Buck,\textsuperscript{17} so as to obtain an estimate of the average values across the four stations on each day. Average values for days when more than two stations were missing were considered missing. There was a highly significant correlation between the recordings of the four monitors.

The broad approach adopted by the APHEA collaboration has been described previously.\textsuperscript{15} The statistical analysis followed that proposed by Zeger,\textsuperscript{18} which has been applied to examine the effects of air pollution on mortality\textsuperscript{19}–\textsuperscript{21} and of temperature on sudden infant death syndrome.\textsuperscript{22} Under this approach, the most important assumption is that, conditional on a latent stochastic process, daily counts of hospital admissions follow a log linear model with given mean and variance structures. In particular, we assume the conditional distribution of the daily counts to be Poisson.

The outcome variable was smoothed by controlling for long term trends and seasonal fluctuations which might cause confounding. These and other adjustments were made through multiple linear regression with the outcome variable being log transformed so as to mirror the scale of a log linear model. The long term trend was adjusted for by fitting a second order polynomial for each calendar year. To adjust for seasonal and other cyclical fluctuations, sine and cosine waves were fitted. The periodicity of the cycles employed varied from 20 days to 2 years. These two procedures performed well as judged by standard diagnostic methods such as periodograms and residual plots.

Other calendar features which were related to daily levels of respiratory admissions were day of the week and bank holidays (seven per year). These effects were modelled using six "dummy" variables for day of the week, the baseline being Sunday, and one dummy variable for all bank holidays.

After deseasonalisation, detrending, and calendar effects were removed, the residuals were used to investigate meteorological effects. Cross correlation functions between residuals and temperature and humidity were used to identify at which lag the effect, if any, is strongest. Also, dose response curves, for the main lags, were examined in order to choose the transformations which gave the best model fit.

The potentially confounding effect of the 1989–90 influenza epidemic was corrected for a dummy variable corresponding to a period of six weeks, this being the duration suggested by inspection of hospital admissions for influenza (ICD 487). To do this, an indicator variable was created and then lagged forwards and backwards up to 15 days, until a good model fit was obtained.

The last stage in the development of the basic model was to correct for the remaining autocorrelation of residuals. Multiple regression models with autoregressive errors were fitted to the data in order to decide how many autocorregressive terms were required. The final model was checked and revised as necessary using autocorrelation functions, Durbin-Watson statistics, plots of fitted values and residuals, and periodograms.

A log linear model with autoregressive Poisson errors was then fitted to the data. Parameter estimates obtained at the immediately preceding step (autoregressive multiple linear regression) were used as initial values in a modified version of the IRLS (in iterative re-weighted least squares procedure), for which an SAS routine was available (J Schwartz, personal communication). Before including air pollution indicators in the model, fully adjusted residuals (without correction for autocorrelation) were plotted against different lags of the untransformed air pollutants using modified scatterplots (bubble plots). This procedure provided an approximation for the exposure–response curve, and was used to choose both the lag and transformation, if any, of the air pollutant indicator.

Air pollution levels on the same day and lagged by 1 and 2 days were investigated. Also, the cumulative effect of each air pollutant was
examined, by averaging its levels on the same day (lag 0) and lagged up to 3 days. Then, lagged and transformed air pollutant indicators were fitted separately to avoid problems of interpretation associated with multicollinearity. The effects of each air pollutant indicator were examined separately for “warm” (April–September) and “cool” (October–March) seasons, by creating a further dummy variable. Where a pollutant was found to have a significant effect and where there was a correlation between this pollutant and another, the effect of putting the second pollutant into the model was examined.

Results
Summary statistics for the pollution, meteorological, and hospital admission variables are shown in table 1. Time series of counts of hospital admissions for the four age groups are shown in figure 1. Trend, seasonality, and a peak at the end of 1989 are noticeable features in all series.

The results to be presented all relate to log linear models with autoregressive Poisson errors. The size and significance of effects due to the main confounders are illustrated in table 2 using relative risk estimates and related 95% confidence intervals. The figures in table 2 have been obtained from the full models, excluding air pollution. When air pollution indicators are entered in the model, these estimates, especially those for meteorology, will change according to the specific levels of collinearity. Nevertheless, both the direction and size of these effects remained fairly similar in those models considered.

Meteorological effects are statistically significant (p<0.05) in all groups except that of 15–64 year olds. The influenza epidemic of 1989–90 affected adults and the elderly similarly, but the largest effect was observed in children. These effects are probably underestimated since the true effects could have been partially removed in the process of deseasonalising the time series. The periods selected in the search for these effects, as explained in section 3, were 18/11/89 to 29/12/89, 6/11/89 to 17/12/89, and 28/11/89 to 8/1/90, respectively for the age groups 0–14, 15–64, and 65+. For all ages, it was the same period as for the elderly. Day of week and holiday effects showed similar patterns in all groups, but with less day of the week variation among children than among the elderly, whose admissions tend to be much less frequent on weekends. As expected, Mondays showed the largest positive relative risk in all groups.
whereas bank holidays showed the lowest deficit in admissions.

Figure 2 shows the exposure–response relationship between adjusted residuals for all age admissions and the six air pollution indicators, each lagged as appropriate. For both O₃ indicators, the shape of the curve suggests a non-linear relationship. As most low O₃ days occur in the cool season and are not associated with large adjusted residuals, the model regarding warm season effects should provide a better estimate for the effect of this pollutant. There is an indication of a threshold at 40–50 ppb and 50–60 ppb, for maximum 8 and maximum 1 hour average O₃ concentrations respectively. In comparison, the scatterplots for NO₂ show an inconsistent exposure–response relationship, apart from a slight positive relationship that emerges for the daily average. Similarly, the exposure–response relationship of adjusted residuals and BS is inconclusive. Finally, for SO₂, a weak relationship at levels greater than 60 μg/m³ is revealed. Scatterplots for the other three age groups and the same air pollution indicators were also examined, and found to be similar (not shown).

The effects of the six pollution indicators are shown in tables 3 and 4. For each of the pollution indicators, effects are presented separately for the chosen single day lag and cumulative lag (up to 3 days), each for the warm and cool seasons separately and for the whole year. Results for 1 hour O₃ and 1 hour NO₂ are not shown (available from authors). In each cell in these tables, the lag, relative risk, and 95% confidence interval are presented. Relative risks were calculated for the range resulting from the difference between the 90th and the 10th centiles of the specific air pollutant frequency distribution.

The most consistent association with respiratory admissions was with O₃. The greatest effect was observed among adults aged 15–64, followed by the elderly and, finally, children. As might be expected, the warm season effects of O₃ are much larger than those for the cool season. With few exceptions, the relative risks for 8 hour average O₃ were greater than those for the maximum 1 hour average (not shown). Furthermore, it is notable that all relative risks which are less than unity are in the cool season while, in the warm season, all relative risks are

Table 2  Relative risks and 95% confidence intervals for selected confounding factors

<table>
<thead>
<tr>
<th>Confounder</th>
<th>All ages</th>
<th>0-14 y</th>
<th>15-64 y</th>
<th>65+ y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature*</td>
<td>1.0614 (1.0364,1.087)</td>
<td>1.0698 (1.0309,1.1103)</td>
<td>1.0181 (0.9786,1.0590)</td>
<td>1.0916 (1.0330,1.1515)</td>
</tr>
<tr>
<td>Relative humidity*</td>
<td>1.0247 (1.0115,1.0378)</td>
<td>1.0258 (1.0142,1.0376)</td>
<td>1.0136 (0.9915,1.0362)</td>
<td>1.0282 (1.0080,1.0489)</td>
</tr>
<tr>
<td>Influenza</td>
<td>1.1983 (1.1661,1.2859)</td>
<td>1.3391 (1.1784,1.5217)</td>
<td>1.2400 (1.1348,1.3724)</td>
<td>1.2291 (1.1230,1.3453)</td>
</tr>
<tr>
<td>Monday</td>
<td>1.3152 (1.2981,1.3419)</td>
<td>1.1548 (1.1204,1.1904)</td>
<td>1.2881 (1.2431,1.3347)</td>
<td>1.5440 (1.4944,1.5953)</td>
</tr>
<tr>
<td>Tuesday</td>
<td>1.1953 (1.1755,1.2241)</td>
<td>1.0622 (1.0302,1.0951)</td>
<td>1.1790 (1.1137,1.2177)</td>
<td>1.3932 (1.3481,1.4399)</td>
</tr>
<tr>
<td>Wednesday</td>
<td>1.1699 (1.1463,1.1939)</td>
<td>1.0029 (0.9723,1.0344)</td>
<td>1.1525 (1.1119,1.1947)</td>
<td>1.3871 (1.3481,1.4397)</td>
</tr>
<tr>
<td>Thursday</td>
<td>1.1392 (1.1161,1.1627)</td>
<td>0.9676 (0.9378,0.9983)</td>
<td>1.1402 (1.1099,1.1819)</td>
<td>1.3523 (1.3084,1.3978)</td>
</tr>
<tr>
<td>Friday</td>
<td>1.1632 (1.1398,1.1872)</td>
<td>0.9964 (0.9660,1.0278)</td>
<td>1.0994 (1.0602,1.1401)</td>
<td>1.4274 (1.3815,1.4750)</td>
</tr>
<tr>
<td>Saturday</td>
<td>0.9737 (0.9533,0.9946)</td>
<td>0.9194 (0.8908,0.9489)</td>
<td>0.9065 (0.8727,0.9417)</td>
<td>1.0988 (1.0614,1.1374)</td>
</tr>
<tr>
<td>Holidays</td>
<td>0.8432 (0.8114,0.8762)</td>
<td>0.9419 (0.8847,1.0227)</td>
<td>0.8650 (0.8088,0.9251)</td>
<td>0.7199 (0.6785,0.7638)</td>
</tr>
</tbody>
</table>

* Temperature and relative humidity levels are untransformed and unlagged in all groups. Relative risks for temperature are based on 10°C, and for relative humidity 20 percentage points. For day of the week, Sunday is 1-00.
greater than one, and are statistically significant, with the exception of 8 hour O3 (lag 2) in children.

To explore the exposure–response relationship between hospital admissions and O3 revealed in figure 2, further modelling was carried out. The plot corresponding to the all ages group for the maximum 1 hour O3 suggests that the threshold of the relationship may lie around 40–60 ppb. In order to investigate different threshold hypotheses, separate effects of low and high O3 days were estimated by including an indicator variable for high O3 days. A cut off point at 40 ppb resulted in O3 coefficients for log admissions per ppb equal to 0.00235 (SE 0.00069, p<0.01) for the 124
For BS and SO₂, statistical significance (p<0.05) was found in only three instances, BS lagged 1 day for children over the whole year (negative association), SO₂ lagged 1 day for children in the warm season (positive association), and SO₂ lagged 1 day for adults aged 15–64 in the cool season (positive association). The negative association might be explained by the negative correlation between BS and O₃, a hypothesis investigated later in this section.

Table 5 shows the correlation coefficients between pairs of air pollutant indicators and meteorological variables for the whole year and the warm and cool seasons. Here, some important relationships emerge. For example, there are also strong positive associations between temperature and O₃ in the three periods as well as between any two of NO₂, BS, and SO₂, in the three periods considered. O₃ and NO₂ have a mixed association – that is, positive in the warm season and negative in the cool season, cancelling out in the whole period. Furthermore, O₃ and BS, and O₃ and SO₂, are negatively associated in the cool season as well as for the whole year.

The O₃ indicators were not strongly correlated with the other air pollutant indicators in the whole year, which makes the implementation of models including two pollutants (one being O₃) appropriate. Models for all age admissions comprising confounders, the O₃ 8-hour average, and one other air pollutant are summarised in table 6. All the air pollutants were lagged (single lags only) as in tables 3 and 4. Remarkably, the inclusion of a second air pollutant in the model together with the 8 hour O₃ hardly affects the magnitude and precision of the estimated O₃ effect. However, the estimated effect for the second pollutant is increased over that from the corresponding single pollutant model. One of the assumptions – that the significant effects of these pollutants are similar in both the single pollutant and two pollutant models. Also, it should be noted that the effects of BS in the single pollutant model changes sign from negative to positive when O₃ is included. This is probably explained by negative confounding by O₃.

### Discussion

The most consistent association with respiratory disease hospital admissions was observed with O₃. The strongest effect was observed in the warmer part of the year and there was evidence that effects become evident above 50 ppb for the 8 hour O₃ level and above 50–60 ppb for the maximum 1 hour O₃. While effects were observed with NOₓ, SO₂, and BS in some age groups, these were not as consistent as or of similar size to those of O₃. It should be noted that we experienced more difficulty in modelling the series in children, possibly because this group is more likely to experience frequent respiratory epidemics and because of the influences of school holidays and half term holidays on hospital admission rates.²¹

The effects of O₃ are unlikely to be explained by covariation with other pollutants because the latter did not show the same degree of association with admissions and were not, in

### Table 5

<table>
<thead>
<tr>
<th>Season</th>
<th>Humidity</th>
<th>O₃-8 h</th>
<th>NO₂-24 h</th>
<th>BS-24 h</th>
<th>SO₂-24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Humidity (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All year</td>
<td>-0.344</td>
<td>0.537</td>
<td>-0.071</td>
<td>-0.270</td>
<td>-0.086</td>
</tr>
<tr>
<td>Cool</td>
<td>-0.061</td>
<td>0.281</td>
<td>-0.265</td>
<td>-0.297</td>
<td>-0.260</td>
</tr>
<tr>
<td>Warm</td>
<td>-0.235</td>
<td>0.425</td>
<td>0.136</td>
<td>0.221</td>
<td>0.215</td>
</tr>
<tr>
<td>O₃-8 h</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All year</td>
<td>-0.474</td>
<td>-0.097</td>
<td>0.141</td>
<td>-0.047</td>
<td></td>
</tr>
<tr>
<td>Cool</td>
<td>-0.254</td>
<td>0.035</td>
<td>0.113</td>
<td>0.047</td>
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</tr>
<tr>
<td>Warm</td>
<td>-0.47</td>
<td>-0.288</td>
<td>-0.082</td>
<td>-0.233</td>
<td></td>
</tr>
<tr>
<td>NO₂-24 h</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All year</td>
<td>-0.09</td>
<td>0.009</td>
<td>-0.287</td>
<td>-0.067</td>
<td></td>
</tr>
<tr>
<td>Cool</td>
<td>-0.434</td>
<td>-0.466</td>
<td>-0.088</td>
<td>-0.034</td>
<td></td>
</tr>
<tr>
<td>Warm</td>
<td>-0.29</td>
<td>0.0008</td>
<td>0.199</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BS-24 h</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All year</td>
<td>-0.638</td>
<td>0.447</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cool</td>
<td>-0.766</td>
<td>0.464</td>
<td>0.438</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warm</td>
<td>-0.34</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

### Table 6

<table>
<thead>
<tr>
<th>Model</th>
<th>O₃ coefficient (SE)</th>
<th>2nd pollutant coefficient (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₃-8 h</td>
<td>0.001111**</td>
<td>(0.0003471)</td>
</tr>
<tr>
<td>O₃-8 h + NO₂</td>
<td>0.001109**</td>
<td>(0.0003479)</td>
</tr>
<tr>
<td>BS-24 h</td>
<td>0.0006295*</td>
<td>(0.0002656)</td>
</tr>
<tr>
<td>NO₂-24 h</td>
<td>0.0004184*</td>
<td>(0.0002022)</td>
</tr>
<tr>
<td>BS-24 h</td>
<td>0.0002274</td>
<td>(0.0005356)</td>
</tr>
<tr>
<td>O₃-8 h + BS-24 h</td>
<td>0.0001740</td>
<td>(0.0004000)</td>
</tr>
<tr>
<td>SO₂-24 h</td>
<td>0.0005960</td>
<td>(0.0003328)</td>
</tr>
<tr>
<td>BS-24 h</td>
<td>0.0003170</td>
<td>(0.0002920)</td>
</tr>
</tbody>
</table>

*p<0.05; **p<0.01.
general, strongly correlated with $O_3$. Although NO$_2$ was correlated with $O_3$, inclusion in the model did not affect the scale or standard error of the $O_3$ coefficient to any extent. It is possible that pollens might be confounding the effects of $O_3$ on those respiratory admissions caused by asthma because these are known to be correlated in the south of England. A future paper focussed on asthma admissions will explore this in detail.

An association between $O_3$ and respiratory hospital admissions has been reported from various parts of North America including Onta-
tario, Vancouver, New York State, Detroit, Minneapolis-St Paul, Birmingham, Alabama, Ontario, and Toronto. A meta-analysis of six studies from North America found that there was no significant heterogeneity between the effects observed and that the weighted average relative risk of respiratory admissions for a 50 ppb increase in the hourly maximum $O_3$ level was 1.06. The relative risk observed in London for a 50 ppb increase was of a similar order. It was 1.0368, 1.0698, 1.0632, and 1.0428 for age groups 0–14, 15–64, 65+, and all ages respectively. $O_3$ has been shown to cause toxic inflammation of the airways and small short term reductions in lung function at ambient levels but there is considerable individual variation in response. It is therefore plausible that individuals with pre-existing respiratory disease might be affected just enough to lead to hospital admission, but we have no direct evidence concerning the actual mechanism.

No relationship between BS and hospital admissions was observed in adults. In the studies cited above, particulates (PM$_{10}$ or sulphates) were also found to have effects and these were independent of those of $O_3$. There are also a number of other studies indicating that particulates are associated with admissions for respiratory disease. The levels of BS (and of PM$_{10}$ available from 1992) in London are within the range associated with effects on admissions in some other cities. The London findings are not, therefore, consistent with most other current evidence (mainly based on North America). It should be noted that London covers a large area. It was represented in this study by only four monitors, which showed only moderate correlation between them. Thus, the exposure assessment might have been too im-
precise to detect a real effect. Particulate matter is variable in size and composition and perhaps the type which occurs in London is less toxic.

While $O_3$ levels were obtained from one central city background monitor, it is likely that, being a secondary pollutant, the time series analysis was more representative of regional fluctuations from day to day. This was confirmed by the very high correlations obtained when daily values from the single monitor used were compared with those from another inner London monitor (Bloomsbury) and an outer suburban monitor (Bexley), for which data were available in recent years. Absolute con-
centrations at Victoria were, however, lower than in the suburbs, due to the greater scav-
egging effect of nitric oxide in the centre where emissions are greatest.

The exposure response relationship is important for control policy and prevention. We observed that admissions did not increase until the 8 hour $O_3$ average was above 50 ppb; above this there was a linear relationship. The WHO health guideline for 8 hour average $O_3$ is 50–60 ppb, and the EC health protection threshold is 55 ppb. Recently, the Expert Panel on Air Quality Standards recommended a level of 50 ppb.

It seems that these recommendations fit closely with the observed threshold but it needs to be appreciated that the levels in central London are likely to be lower than those affecting the suburbs. Nevertheless, our findings suggest that there is little or no safety margin in a 50 ppb standard.

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