

**PM<sub>2.5</sub> AND DAILY HOSPITAL ADMISSIONS  
IN THE  
WEST MIDLANDS CONURBATION, UK  
  
REPORT**

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BEHALF OF THE EXPERT PANEL ON AIR QUALITY  
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## SUMMARY

### **Background**

This report was commissioned by the Department of Health to inform the Expert Panel on Air Quality Standards in its consideration of a standard for PM<sub>2.5</sub>, a measure of fine particles. The main underlying question was whether there was evidence that PM<sub>2.5</sub> could offer any advantages over and above PM<sub>10</sub> for population monitoring of particles. A subsidiary question related to the relative effects of the primary and secondary components of the fine particle fraction.

The main aim of the research described in this report was to investigate whether PM<sub>2.5</sub> was associated with daily emergency hospital admissions for cardio-respiratory diagnoses, and to compare PM<sub>2.5</sub> effects, if any, with those of other measures of particles, namely PM<sub>10</sub>, PM<sub>2.5-10</sub> (the calculated coarse fraction), Black Smoke and sulphate.

### **Methods**

Counts of daily emergency admissions for cardiovascular and respiratory diseases were obtained from the Hospital Episode System for the District Health Authorities comprising the West Midlands Conurbation (population 2.3 million). The period of study was determined by the availability of PM<sub>2.5</sub> data and extended over 823 days between October 1994 and December 1996. Within this period, PM<sub>2.5</sub> data were available for 700 days. Other pollutants examined were PM<sub>10</sub>, PM<sub>2.5-10</sub>, sulphate, Black Smoke, modelled estimates of the primary and secondary fine fractions, sulphur dioxide, ozone, nitrogen dioxide and carbon monoxide. The statistical approach was to apply generalised additive modelling (GAM) to control for seasonal, meteorological calendar and other potential confounders. Based on previous evidence, we chose *a priori* to investigate the pollution effects using the mean of lag 0 and lag 1. However, a comprehensive range of single day and cumulative lags was investigated and these results are also included in the report.

### **Results**

The effects of the various measures of particles differed across diagnostic and age groups and between the cool and warm seasons. Effects ranged from strongly positive to strongly negative, but were mostly small and non-significant.

For cardiovascular admissions there was little evidence of an association with any measure of particles. The percentage change in cardiovascular admissions for a 10<sup>th</sup>-90<sup>th</sup> percentile change in PM<sub>2.5</sub> was -0.5% (95% CLs -2.5,1.3). The largest effects were observed in the elderly and in the warm season. The effects on stroke were negative. There were no clear distinctions between the various measures of PM.

For respiratory admissions, the effects of  $PM_{2.5}$  were mostly positive but small though some were statistically significant. For all respiratory diagnoses, all ages, the estimates (% change (95% CLs) for 10-90<sup>th</sup> percentile change in pollutant) were 1.2% (-0.9,3.4) for all year, 0.6% (-2.5,3.8) for the cool season and 4.2% (0.1,8.5) for the warm season. The results for  $PM_{2.5}$  were similar to those for  $PM_{10}$  which was not surprising because the two measures were highly correlated ( $\rho=0.92$ ). The results for  $PM_{2.5-10}$  tended to be less consistent. Two pollutant models were fitted to find out if the effects of  $PM_{2.5}$  were stable to the inclusion of other measures of particles. The results were consistent with  $PM_{2.5}$  effects being independent of  $PM_{2.5-10}$  and of sulphate, but not of Black Smoke. Conversely, the effect of Black Smoke tended to be enhanced with the inclusion of  $PM_{2.5}$  in the model. On inspection of the lag patterns, there were clear similarities between  $PM_{10}$ ,  $PM_{2.5}$  and Black Smoke. The lag pattern of  $PM_{2.5-10}$  tended to be different from that of  $PM_{2.5}$ . There was a tendency towards larger effects in children in the winter and in the elderly in the summer. The largest and most significant effects were observed for asthma in children, but the pollutant with the largest effect was  $SO_2$  rather than particles.

## Conclusions

It is difficult to come to firm conclusions from the analysis of this rather short  $PM_{2.5}$  time series. However, there is sufficient evidence to conclude the following:

1.  $PM_{2.5}$  is probably associated with small effects on daily admissions for respiratory disease.
2. The effects of  $PM_{2.5}$  are similar to those of  $PM_{10}$  and cannot be separated.
3. Compared with  $PM_{2.5}$ ,  $PM_{2.5-10}$  tended to show smaller and less consistent health effects as well as a different lag pattern.
4. Black Smoke and sulphate, both of which are largely found in the fine fraction, showed effects that were equivalent to those of  $PM_{10}$  and  $PM_{2.5-10}$ .
5.  $PM_{2.5}$  and BS do not appear to have effects independent of each other.
6. Overall the results point to  $PM_{2.5}$  being the more active component of  $PM_{10}$ . However, the results for  $PM_{2.5}$  tended to parallel those for  $PM_{10}$ , and in practice,  $PM_{10}$  would have been sufficient to detect health effects of similar magnitude.

## BACKGROUND

In August 1998 the Department of Health, on behalf of the Expert Panel on Air Quality Standards (EPAQS), commissioned an epidemiological study to investigate the effects on daily mortality and emergency admissions of a measure of fine particles,  $PM_{2.5}$ . Currently in the UK, the measure of ambient particles subject to air quality standards is  $PM_{10}$ , an estimate of the weight of particles of aerodynamic diameter less than  $10\mu m$ . Arguments have been advanced to support the idea that it is the fine particle fraction of  $PM_{10}$ , measured by  $PM_{2.5}$ , that contains the most toxic element. The epidemiological evidence (reviewed for EPAQS in a separate document) relates to only a few studies of mortality and lung function, but there are no studies of effects of fine particles on hospital admissions. Thus there is a paucity of epidemiological evidence from the UK or similar environments elsewhere upon which to base any recommendations for a separate standard for  $PM_{2.5}$ .

The availability of  $PM_{2.5}$  measurements in Birmingham from October 1994 has enabled us to study its effects on daily counts of deaths and emergency hospital admissions. The first report from this study detailed results of the time-series analyses of daily mortality and was submitted to the Department of Health in February 1999. This second report presents the results of a comprehensive time-series analysis of daily emergency hospital admissions for cardiovascular and respiratory diseases. This report presents the methods, main results and conclusions from the large quantity of analyses performed. The Annex contains all the results for all analyses as well as additional descriptive statistics.

The objectives of the analysis can be listed as follows.

1. Is  $PM_{2.5}$  associated with daily hospital admissions?
2. How does any effect compare with that of  $PM_{10}$ ?
3. Is there an independent effect of  $PM_{2.5-10}$ ?
4. Are other measures of fine particles associated with daily hospital admissions? These include Black Smoke, sulphate, and modelled estimates of fine particles of primary and secondary origin.
5. To what extent can any effect of individual particle measures be distinguished from one another?
6. What are the exposure-response relationships?

## METHODS

### HOSPITAL ADMISSIONS DATA

Counts of daily emergency hospital admissions for cardiovascular and respiratory causes were constructed for the period October 1994 to December 1996 (823 days) from Hospital Episode System (HES) files supplied by IBM on behalf of the Office of National Statistics (ONS). Thirteen separate admissions series were investigated: all cardiovascular disease (ICD9: 390-459)(all-ages), cardiac disease (390-429) (all-ages and over 65), ischaemic heart disease (410-413) (ages 0-64 and over 65) stroke (430-438) (ages over 65), all respiratory diseases (460-519) (0-14, 15-64, 65+), asthma (493) (ages 0-14 and 15-64) and chronic obstructive pulmonary disease (490-492, 494-496) (ages over 65). Only admissions of those people resident and admitted in the study area were included. The study area covered Birmingham District Health Authority together with the surrounding District Health Authority areas of Dudley, Sandwell, Solihull, Walsall and Wolverhampton. Together these comprise the West Midlands conurbation with a population of approximately 2.3 million (Source: ONS PP/1 1997 mid-year population estimates).

### ENVIRONMENTAL DATA

Air pollution data for the West Midlands were obtained from AEA Technology.

Particle measures studied were  $PM_{10}$ ,  $PM_{2.5}$ , black smoke (BS), sulphate ( $SO_4$ ) and an estimate of the coarse fraction ( $PM_{2.5-10}$ ) derived by subtracting the  $PM_{2.5}$  from the  $PM_{10}$  measures. In addition, estimates of primary (PFR) and secondary (SFR) particle fractions were studied. These estimates were derived from a statistical model relating  $PM_{10}$ ,  $PM_{2.5}$ , BS and sulphate measurements in the UK between January and March 1996.[1] Also studied were nitrogen dioxide ( $NO_2$ ), ozone ( $O_3$ ), sulphur dioxide ( $SO_2$ ) and carbon monoxide (CO).

Only those pollution monitoring stations providing data for at least 75% of days during the study period were used, a criterion adopted from the APHEA project.[2]  $PM_{10}$  data from three stations (Birmingham Central and East and Hodge Hill) were used to compute daily study-wide average measures. Three stations provided BS measurements (Sedgley, Walsall and Wednesbury), two rurally situated monitors provided  $SO_4$  measurements (Stoke Ferry and High Muffles) and one  $PM_{2.5}$  data (Hodge Hill). 18% of daily measures of  $PM_{2.5}$  were missing, predominantly at the end of the time series. The coarse fraction,  $PM_{2.5-10}$ , was derived from the Hodge Hill data. Study-wide averages were calculated for  $NO_2$  measured at 4 stations (Birmingham Central and East, Hodge Hill and Walsall Alumwell),  $O_3$  measured at two stations (Birmingham Central and East),  $SO_2$  measured at five stations (Birmingham Central and East, Sedgely, Walsall Alumwell and Hodge Hill) and CO measured at two stations (Birmingham Central and East). Daily measures on the same day, and one, two and three days previously as well as cumulative means over two, three and four days for gases and up to 6 days for particles were investigated for each pollutant.

Daily maximum and minimum temperature and relative humidity measured at 6am and 3pm were obtained from the University of Birmingham and the Meteorological Office. The mean of each pair of measurements was calculated and used in the analysis.

## STATISTICAL METHODS

For each time series, defined by disease and age group, a 'core' statistical model describing the temporal pattern in morbidity and the relationship with temperature and humidity was constructed.

The primary concern in developing these models is to adequately remove long-term patterns in the data, which confound the relationship between short-term fluctuations in air pollution and daily counts of hospital admissions. Serial correlation, a feature of such outcome data, needs to be eliminated by including known confounders (e.g. weather) otherwise inferences from our model will be wrong. Specifically, the confidence intervals around our pollutant parameter estimates will be too narrow.

Temporal patterns were modelled using non-parametric 'smoothes' of time. A smoother is a tool for summarising the trend in one variable (in this case daily counts of hospital admissions) as a function of another (here, time), a simple example of which is the moving-average. The relationship between the two variables is determined by the data themselves rather than as a rigid form as in linear regression. The type of smoother used in this study was the cubic B-spline.

By increasing the amount of smoothing performed, more temporal patterns are removed resulting in a closer fit to the data. The inherent trade-off is between the variability and the bias of the resultant fit. Over-controlling for these temporal patterns can induce spurious relationships. One tool that facilitates this assessment is the partial autocorrelation function (PACF), which illustrates the serial correlation structure in the residuals from our model.

Day-of-week patterns were controlled for by six dichotomous dummy variables. Indicators for school holidays and public holidays were also considered and included in the core models if statistically significant. Daily counts of hospital admissions for influenza (487) were used to control for the potential confounding effect of influenza epidemics, where statistically significant.

The strength of relationship with temperature was assessed by plotting the residuals from the seasonally-adjusted model against single-day lags of temperature (24-hour minimum, mean and maximum) for the same day as the admission count and 1 and 2 days prior to the admission count. Cumulative lags (mean of current and previous days, mean of current and previous two days and mean of previous two days) were also plotted. A cubic smoothing spline was used to pick-out graphically the relationship between the residuals and these measures of temperature. Control for temperature was made by selection of the single or cumulative lag showing the strongest relationship. Whether a parametric form or non-

parametric form of control was used depended on the relationship observed. If a simple linear or piece-wise linear representation was satisfactory, this was preferred to a non-parametric (smooth) form. Choice amongst competing models was aided by a standard objective measure (Akaike's Information Criterion). Subsequent to adequate temperature control, relative humidity was assessed and dealt with in a similar manner.

The statistical models fitted were 'generalised additive models' (GAM) which extend ordinary regression models in two main ways. Firstly, the predictor variables can consist of one or more unspecified functions (non-parametric smoothing terms) to address non-linear relationships between outcome and predictor(s), whereas ordinary regression models are linear in their parameters. Secondly, situations where the outcome variable is not Normally distributed, as in daily count data which typically follow a Poisson distribution, are accommodated.[3] Such models of count data are often overdispersed, that is, the variance of the residuals is larger than expected from a Poisson process. This also leads to confidence intervals that are too narrow. Rather than specify a Poisson error distribution we used a more general method, with less assumptions, which estimates the overdispersion and inflates the standard errors accordingly.[4]

Each final core model was re-checked by inspecting plots of the model residuals for any potential outliers or influential points. If present, and if these could be attributed to specific calendar events, further dummy variables were defined as appropriate; otherwise a robust regression procedure was used to reduce the risk of obtaining biased pollutant estimates. On satisfactory completion of the modelling, the partial autocorrelation function was plotted and a decision was made on whether or not autoregressive terms were required in the model to control for any remaining serial correlation.[5]

The pollutants were fitted as linear (i.e. parametric) terms. Due to 18% of PM<sub>2.5</sub> observations being missing, it was necessary to construct separate core models for continuous but shorter series. These series covered a period of 700 days (1/11/94 to 30/9/96). These models were used to assess the PM<sub>2.5</sub> associations as well as the PM<sub>2.5-10</sub> and additional PM<sub>10</sub> associations to allow comparison with results based on the full 823 days of data.

Evidence for effect modification of pollutant by season (cool, October to March, or warm, April to September) was assessed by fitting an interaction term between pollutant and season and noting the P-value obtained for that term.

The exposure-response relationship between a pollutant, expressed on a continuous scale and relative risk of hospital admission was estimated, given the scenario of an increase from the 10<sup>th</sup> percentile of the pollutant to the 90<sup>th</sup> percentile of its range. The potential for a non-linear (threshold) relationship between pollutant and relative risk of death was explored by smoothing the plot of relative risk against pollutant by means of a cubic B-spline applied to the pollutant term in the core model. An F-test for the non-parametric (non-linear) part of the model was used to judge if this relationship could indeed be non-linear.

A limited number of two-pollutant models were fitted to assess the stability of the  $PM_{2.5}$  effect in the presence of several of the other particle measures.

All of the analyses were carried out using S-Plus 4.5 Professional. [6]

## RESULTS

### DESCRIPTIVE STATISTICS

Descriptive statistics for the outcome series (Table 1), pollutants and meteorological variables (Table 2) are shown for the period October 1994 to December 1996. PM<sub>10</sub> measurements are described for both an 823-day and a 700-day series, the latter to correspond with the reduced availability of PM<sub>2.5</sub> and thus PM<sub>2.5-10</sub> data. The summary statistics for both PM<sub>10</sub> series are similar.

Table 3 presents simple (unadjusted) correlation coefficients between PM<sub>2.5</sub> and each outcome, pollutant and meteorological variable for all-year and the cool and warm seasons separately. The table shows that the all-year correlations with each outcome were generally small and positive while the cool season correlations were small and negative and those in the warm season tended to be larger and positive. PM<sub>2.5</sub> was highly correlated with PM<sub>10</sub>,  $\rho=0.92$ , with little difference within the cool and warm seasons. Correlation between PM<sub>2.5</sub> and black smoke was also strong ( $\rho=0.7$ ). Correlations between PM<sub>2.5</sub> and SO<sub>4</sub> and SFR were identical and very similar to those with PFR. The calculated coarse fraction was less well correlated with PM<sub>2.5</sub> ( $\rho=0.52$ ), than with the fine particle measures whether directly measured or estimated from a model. [1]

### REGRESSION ANALYSES

1. We have an *a priori* hypothesis, based on previous studies, that any effects will be observed on the same or next day. We therefore present results for the mean of day 0 and day 1. This approach also helps to avoid the pitfall of over-interpreting a single significant result within the context of many tests of significance. In addition, because so many models were studied, attention is directed more towards those results which are significant at and beyond the 1% level, rather than the conventional 5% level. However, to prevent confusion, we have retained the use of 95% confidence limits when quoting results. The annex contains results of all significance tests for all pollutants and outcomes, and all single day and cumulative lags studied.
2. Tables 4-7 present the results of the regression analyses for all particle measures and gases for each outcome studied. They show the relative risk of admission to hospital as a percentage change in the mean number of daily admissions associated with an increase in the pollutant from the 10th to 90th percentile values of its distribution over the study period. These pollutant ranges are given in the key to the tables. They are reported, together with 95% confidence limits and P values, for all-year and cool and warm seasons, together with a P value for the test of effect modification by season to answer the question "are the cool and warm season results significantly different from each other?"

## PM<sub>2.5</sub> AND OTHER PARTICLE MEASURES

### CARDIOVASCULAR DIAGNOSES

**All cardiovascular disease.** We found no evidence of statistically significant associations between daily counts of all emergency admissions for cardiovascular diseases and PM<sub>2.5</sub>, PM<sub>2.5-10</sub> or PM<sub>10</sub> (Table 4). The results were similar for the other particle measures: black smoke, sulphate and the estimated primary and secondary fine particle fractions. Effect estimates for PM<sub>2.5</sub>, PM<sub>2.5-10</sub> and PM<sub>10</sub> were negative and less than 1% in magnitude. Results for the warm and cool seasons were generally not significantly different though there was a clear tendency, when compared to the all-year results, towards positive associations in the warm season and negative associations in the cool season.

**Cardiac disease.** Admissions for cardiac disease represent the majority of all cardiovascular admissions with more than half of these in the over 65 age group (Table 1). The pattern of associations described for the all-age cardiovascular disease group was also found, not unexpectedly, in the all-age cardiac disease group (Table 4). The warm season effect estimates for PM<sub>2.5</sub> and PM<sub>10</sub> were of a comparable magnitude, 2.4% (95% CLs: -1.6, 6.7) and 3.4% (-0.8, 7.7) respectively, though neither reached statistical significance and were larger than the association with PM<sub>2.5-10</sub>, 0.8% (-4.0, 5.8). As in the all cardiovascular group, associations between cardiac admissions and the SFR and SO<sub>4</sub> particle measures were almost identical. The results for cardiac admissions in the over 65s (Table 4) suggest that this group are particularly sensitive to air pollution levels, although this cannot be formally tested using this study design. All all-year associations between admissions in this sub-group and particles were positive in direction, ranged between 1 and 3% in size but only the effect of BS was evidently different from zero, 3.3% (0.3, 6.4) P=0.03. We found that the warm season effects of fine and coarse particles were the same and were comparable to the estimated effect of PM<sub>10</sub>. These effects were relatively strong, over 4%, but still failed to reach statistical significance. The other particle measures (BS, SO<sub>4</sub> and PFR and SFR) also had similar results to PM<sub>2.5</sub> in this sub-group.

**Ischaemic heart disease.** The results for Ischaemic Heart Disease in the over 65s also showed small, non-significant all-year effects and considerably stronger warm season associations, almost significantly so in the case of PM<sub>2.5</sub> and PM<sub>10</sub>; 6.8% (-0.7, 14.9) and 6.9% (-0.5, 14.8) respectively (Table 5). The coarse fraction estimate, PM<sub>2.5-10</sub>, was 3.5% (-4.6, 12.1), almost half that for PM<sub>2.5</sub>.

**Stroke.** For emergency admissions for stroke in the over 65s there was a tendency for negative, non-significant associations with the particle measures and there were no seasonal differences (Table 5). The most prominent finding was an unexpected strong negative association between the coarse fraction, PM<sub>2.5-10</sub> and stroke admissions was found in the all-year analysis, -8.2% (-13.9, -2.2) P=0.008.

## RESPIRATORY DIAGNOSES

**All respiratory, all ages.** For the all-year, all-age respiratory disease category, we found that all particle measures were positively, though not significantly, associated with an increased risk of admission (Table 6). All associations were small, 1 or 2%, with comparable 95% confidence limits. There was, however, a clear tendency towards larger associations during the warmer months although these seasonal differences also failed to attain statistical significance. There was a clear distinction between the magnitude of the warm season effect estimates for PM<sub>2.5</sub> and PM<sub>10</sub> and that of the coarse fraction estimate; 4.2% (0.1, 8.5) and 2.6% (-1.4, 6.7) compared to -0.7% (-4.9, 3.8) for PM<sub>2.5-10</sub>. The strongest association with respiratory admissions was with Black Smoke.

**All respiratory, by age group.** When the respiratory disease category was sub-divided by age distinct patterns emerged (Table 6). In children, age 0-14 years, all all-year particle effect estimates were positive and all, except SFR and SO<sub>4</sub>, were of comparable magnitude and were mostly significant. For PM<sub>2.5</sub> the percentage change in admissions was 3.4% (-0.1, 7.0), for PM<sub>10</sub>, 4.1% (0.4, 8.0) and for PM<sub>2.5-10</sub> the estimate was 4.4% (-0.3, 9.4). No statistically significant seasonal differences were found nor was there a tendency towards stronger associations during the warm season. In adults, age 15-64, we found no significant associations between respiratory admissions and any measure of particles in either the all-year or seasonal analyses. Associations with PM<sub>2.5</sub> were negative in the all-year and cool season results and positive in the warm season.

The most unusual results from these analyses of hospital admissions were found in the elderly group, age 65 and over, admitted with respiratory disease. We found that all particle measures were negatively associated, though not significantly so, with all-year admissions. However, we found highly significant seasonal differences in the results. During the cool season, all particle measures were negatively associated with admissions and many of these approached statistical significance at the 1% level. These contrasted sharply with the strong positive associations found in the warm season. For PM<sub>2.5</sub> the all-year result was -1.3% (-4.7, 2.2) compared to -5.7% (-10.5, -0.6) in the cool season and 8.7% (2.1, 15.3) in the warm season, P=0.0009 for test of seasonal difference in effect estimates. A very similar result was recorded for PM<sub>10</sub> and a weaker effect for PM<sub>2.5-10</sub>. The SFR, BS and SO<sub>4</sub> measures were also strongly associated with respiratory admissions in the elderly during the warm season.

**Asthma (0-14, 15-64) and COPD (65+).** We also investigated asthma admissions in children and adults and COPD admissions in the elderly (Table 7). In each of these diagnostic sub-groups the mean daily number of admissions were approximately one third of the relevant all respiratory groups (Table 1). We found that the pattern of age-specific associations present in the all respiratory groups were, on the whole, replicated for asthma and COPD, suggesting a lack of specificity by disease classification. However, in the asthma sub-groups we did find that the magnitude of the associations was considerably larger than in the all respiratory groups, although the levels of statistical significance were comparable. There was little difference in the estimated, all-year, effects of PM<sub>2.5</sub>, PM<sub>10</sub> and PM<sub>2.5-10</sub> on children's or adults' asthma, but once again the

seasonal analyses revealed quite different effect estimates during the warm season compared to the cool season. For children, the change in admissions associated with PM<sub>2.5</sub> in the warm season was 14.2% (1.5, 28.4) and with PM<sub>10</sub>, 9.4% (-2.7, 23.0) but for PM<sub>2.5-10</sub> the association was almost half that of PM<sub>2.5</sub> at 5.7% (-7.8, 21.2). It should be emphasised though, that these estimates have poor precision and that the upper confidence limits of each of these three estimates are all above 20%. A similar seasonal pattern of results was found for COPD admissions.

## GASES

There was little evidence for an association between any of the pollutant gases and an increase in admissions for cardiovascular diagnoses. Effect estimates tended to be positive but ranged from -6% to +2.5%. For respiratory disease there were strong associations between SO<sub>2</sub> and admissions for all respiratory complaints and asthma in children. The cumulative mean measure of the current and previous day was associated with an increase in all respiratory admissions of 4.6% (1.4, 7.8) P=0.004. Admissions for asthma increased by 10.9% (4.5, 17.8) P=0.0007 in association with SO<sub>2</sub> with a very large warm season effect of 19.3% (7.9, 31.9) P=0.0006 and P-value for interaction term P=0.05.

We found no evidence of an effect of O<sub>3</sub> on either respiratory or cardiovascular outcomes. NO<sub>2</sub> was positively and significantly associated with admissions for respiratory disease during the warm season; 6.2% (2.2, 10.4) P=0.002.

## RESULTS FOR SINGLE DAY LAGS

So far, we have presented only the results for the cumulative lag 0-1 since this was our *a priori* position. However, it is also informative to compare the distribution of single day lags. Figures 1 and 2 illustrate the pattern of associations between cardiovascular and respiratory outcomes respectively for the various lag measures of each pollutant. The estimates and 95% confidence intervals for lags 0 to 3 together with the result for the mean of lags 0 and 1 (presented in tables 4 to 7) are plotted for PM<sub>10</sub>, PM<sub>2.5</sub>, PM<sub>2.5-10</sub>, Black Smoke and Sulphate.

For cardiovascular disease, Figure 1 shows that the choice of the *a priori* measure for each pollutant of mean of lag 0 and 1 provided a good 'over-all' estimate of the result for a given pollutant in terms of the magnitude of the effect and the level of statistical significance. Figure 1 also enables the relative size of effect for each particle measure to be compared. There was little to choose between the strength of the PM<sub>10</sub> and PM<sub>2.5</sub> associations whilst the calculated coarse fraction component of PM<sub>10</sub> seemed to vary more in terms of direction and magnitude of effect. BS and SO<sub>4</sub> appeared to give larger effects estimates than PM<sub>2.5</sub>, especially during the warm season. These differences were large in percentage terms but were probably of little significance in terms of health effects. For example, during the warm season, the effect estimates for PM<sub>10</sub>, PM<sub>2.5</sub>, BS and SO<sub>4</sub> were -1.2, 0, 2.3 and 1.9% respectively.

Figure 2 shows similar data for all respiratory diseases. In the all year analysis, there was little to choose between the various measures of particles. PM<sub>2.5</sub> and Black Smoke showed similar

patterns of lags with slightly larger relative risks for Black Smoke. Similarly in the cool season, PM<sub>2.5</sub> and Black Smoke had similar distributions of lags, but with changes in relative risks for Black Smoke being a little larger. In the warm season, the Black Smoke had the largest effects closely followed by PM<sub>2.5</sub> and sulphate. A consistent finding was that the pattern of lags for PM<sub>2.5</sub> was the same as observed with PM<sub>10</sub> but very different from that of PM<sub>2.5-10</sub>.

#### EXPOSURE -RESPONSE RELATIONSHIPS

Separate models were built to cover the period from November 1994 to September 1996, within which a largely continuous series of PM<sub>2.5</sub> measurements were available. This enabled exposure-response relationships to be examined. We chose to investigate admissions for cardiovascular diseases, respiratory diseases and asthma in children. We did not find any evidence that the exposure-response of these pollutants was different from linear. That is to say that we did not find any threshold effects. Relevant figures and tables may be found in the annex.

#### TWO-POLLUTANT MODELS

PM<sub>2.5</sub> was correlated with the various other particle measure to a varying degree. The purpose of the two pollutant modelling was to obtain some insight into whether any effects of PM<sub>2.5</sub> might be attributed to another pollutant with which it was associated. The results of the dual pollutant analyses are shown in Table 8. For cardiovascular diagnoses, the PM<sub>2.5</sub> estimate was rather unstable when any of the other particle measures were included in the model. For respiratory disease, the PM<sub>2.5</sub> estimate was not affected by inclusion of either PM<sub>2.5-10</sub> or sulphate, but was strongly affected by inclusion of BS. Because this analysis is dealing with small non-significant estimates with wide confidence intervals, interpretation must be very cautious. However, the results for respiratory admissions are consistent with the following conclusions: 1) that PM<sub>2.5</sub> effects are independent of PM<sub>2.5-10</sub>; and 2) that the effects of PM<sub>2.5</sub> are just as likely to be due to the black smoke component as the sulphate component.

#### DISCUSSION

The results of these analyses may be summarised as follows:

Risks of health effects associated with particles varied across diagnostic groups, age groups and seasons and ranged from strongly positive to negative, but the overall impression is that most effects are small and generally non-significant.

There were few associations between particles and cardiovascular diagnoses. The greatest effects were among the elderly in the warm season. There were negative effects on stroke. There was no clear distinction between any of the measures of particulate matter.

There were more effects on respiratory diseases. Risks were generally positive and some were significant. There was a tendency for the largest effects to be observed among children in the cool season (more noticeably for asthma) and among older adults in the warm season.

The effects of  $PM_{2.5}$  tended to parallel those of  $PM_{10}$  rather than  $PM_{2.5-10}$ . In two pollutant models, the effects of  $PM_{2.5}$  were independent of  $PM_{2.5-10}$ . The pattern of lags was similar for  $PM_{2.5}$  and  $PM_{10}$ .

The effects of Black Smoke were marginally greater than those of  $PM_{2.5}$  but followed a very similar lag distribution. Furthermore, in two pollutant models, the effect of  $PM_{2.5}$  was sensitive to the inclusion of Black Smoke and vice-versa

The effects of sulphate were less closely related to those of  $PM_{2.5}$  than those of Black Smoke.

Before coming to conclusions concerning the effects of  $PM_{2.5}$ , it is important to discuss several methodological aspects of the study, since these might affect the nature and validity of the results.

The statistical approach used GAMs. This is now the method of choice in all major studies of the short-term effects of air pollution including APHEA 2 and the US NMMAPS study. Comparison with the earlier approach which used parametric methods of seasonal control has generally found that the results are similar. Most sensitivity studies have used mortality data and these are more orderly than hospital admissions data. However, as part of our contract to study the effects of air pollution on hospital admissions in London, we have conducted a formal comparison of the two methods. This report is in preparation but it concludes that the results for hospital admissions are also relatively insensitive to whether parametric or semi-parametric methods are used. The statisticians concerned with this work are currently involved with the APHEA collaboration and have had the benefit of close contact with leading exponents of GAM techniques in air pollution epidemiology. We are therefore fairly confident that the method has been applied correctly and that errors such as over-fitting of the models have been avoided.

An important obstacle to addressing the research questions was the limited data available. Most authorities consider it important to have at least three years of data. We had less than two years of data which will have made it difficult to make sufficiently precise estimates of what are probably small effects. To overcome this small signal to noise ratio would require a longer data series in a larger city such as London. However it should be noted that the  $PM_{2.5}$  effect estimates obtained for all of the diagnostic and age group combinations were very similar to the results found for  $PM_{10}$  (averaged over 3 separate sites) on the same 700-day series and indeed on the full 823-day series.

It is also of interest to see how the results of this compare with those of earlier Birmingham studies and of London. This can only be done for  $PM_{10}$  and Black Smoke, but will give some

indication of whether our results are in line with other studies, and the generalisability of the results. Some results for  $PM_{10}$  and Black Smoke observed in this study are compared with those of London 1992-94 in Table 9.[7] Percentage changes in relative risk are given for a 10 unit increment in each pollutant for the cumulative mean measure of the current and the previous day. The results are more similar for all respiratory disease, all ages than all cardiovascular disease, all ages, and most similar for black smoke. One reason why significant results were not observed in Birmingham, while they were in London could be that the Birmingham study was less powerful given the smaller event counts for each outcome.

Some of the conclusions rest on the results of two pollutant models. These are problematic for various reasons, including sometimes high correlations between them, and the strong seasonal patterns, and should be interpreted cautiously. When variables in a statistical model are closely correlated, regression coefficients can become unstable, that is, sensitive to small random fluctuations in the response variable and to the particular variables included in the model. This is reflected in large standard errors, a problem compounded by small numbers of days for analysis. A longer time series might have given more stable results.

## CONCLUSIONS

1. Interpretation of the findings must consider the large number of analyses carried out. Most credence should be given to results that relate to the main hypothesis using the cumulative lag 0+1 days, and to main rather than subgroup analyses.
2. In the main all-year all age analysis, there was no strong or consistent association between  $PM_{2.5}$  and daily admissions for respiratory disease.
3. Various associations emerged when various diagnoses and subgroups were analysed.
4. Where present, the effects of  $PM_{2.5}$  are similar to those of  $PM_{10}$  and cannot be separated.
5. Compared with  $PM_{2.5}$ ,  $PM_{2.5-10}$  tended to show smaller and less consistent health effects as well as a different lag pattern.
6. Black Smoke and sulphate, both of which are largely found in the fine fraction, showed effects that were equivalent to those of  $PM_{10}$  and  $PM_{2.5-10}$ .
7.  $PM_{2.5}$  and BS do not appear to have effects independent of each other.
8. Overall the results suggest point to  $PM_{2.5}$  being the more active component of  $PM_{10}$ . However, the results for  $PM_{2.5}$  tended to parallel those for  $PM_{10}$ , and in practice,  $PM_{10}$  would have been sufficient to detect health effects of similar magnitude.

## REFERENCES

1. Stedman JR. The secondary particle contribution to elevated PM<sub>10</sub> concentrations in the UK. Oxfordshire: AEA Technology, 1998.
2. Katsouyanni K, Schwartz J, Spix C *et al*. Short-term effects of air pollution on health: a European approach using epidemiologic time series data: the APHEA protocol. *J.Epidemiol.Community.Health* 1996; **50** (suppl 1): S12-8.
3. Hastie TJ, Tibshirani RJ. Generalized Additive Models. London: Chapman Hall, 1990.
4. Zeger SL. A regression model for time series of counts, *Biometrika* 1988; **75**: 621-9.
5. Schwartz J, Spix C, Touloumi G *et al*. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J.Epidemiol.Community.Health* 1996; **50** (suppl 1): S3-11.
6. S-Plus 4.5 Professional. Data Analysis Products Division, MathSoft Inc., Seattle, Washington, April 1998.
7. Atkinson RW, Bremner SA, Anderson HR *et al*. Short-term associations between emergency hospital admissions for respiratory and cardiovascular disease and outdoor air pollution in London. *Arch of Envir H* 1999; **54**: 398-411.

**Table 1. Descriptive Statistics for outcome variables**

	season	mean	std.dev	min.	10 <sup>th</sup>	med.	90 <sup>th</sup>	max.	
All Cardiovascular	All-Year	70.8	14.6	33	50	72	89	112	
	All Ages	Cool	73.2	14.7	37	53	74	91	112
		Warm	67.8	14.0	33	48	69	85	103
		All-Year	50.4	10.7	24	36	51	64	87
	Cardiac Disease	Cool	51.8	10.7	27	37	53	66	87
		Warm	48.6	10.6	24	35	48	63	72
		All-Year	33.2	7.9	12	23	33	44	59
	65+	Cool	34.2	7.8	15	24	34	44	59
		Warm	32.0	7.9	12	21	32	43	54
All-Year		9.9	3.4	0	6	10	14	22	
Ischaemic Heart Disease	Cool	10.4	3.5	0	6	10	15	22	
	Warm	9.3	3.2	2	6	9	14	21	
	All-Year	13.8	4.1	3	9	13	19	29	
Ages 0 – 64	Cool	14.3	4.2	3	9	14	20	29	
	Warm	13.2	4.0	3	8	13	19	26	
	All-Year	9.7	3.6	1	5	9	14	24	
Stroke Ages 65+	Cool	10.4	3.6	1	6	10	15	20	
	Warm	8.8	3.3	2	5	8	13	24	
	All-Year	66.2	22.5	18	42	63	99	152	
All Respiratory	Cool	75.9	23.6	36	49	70	109	152	
	Warm	54.1	13.3	18	37	54	71	87	
	All-Year	26.9	12.7	3	13	24	44	81	
	0 – 14	Cool	32.3	13.2	8	18	29	53	81
		Warm	20.3	8.3	3	10	20	31	47
		All-Year	16.1	5.3	1	10	16	23	38
	15 – 64	Cool	17.4	5.4	3	11	17	25	38
		Warm	14.5	4.7	1	9	14	21	29
		All-Year	23.1	9.2	6	13	22	35	63
65+	Cool	26.1	10.2	6	16	24	41	63	
	Warm	19.3	5.8	6	12	19	27	35	
	All-Year	7.7	4.3	0	3	7	13	34	
Asthma	Cool	8.1	3.6	0	4	8	13	20	
	Warm	7.2	5.1	0	2	6	14	34	
	All-Year	5.0	2.6	0	2	5	9	14	
15 – 64	Cool	5.5	2.7	0	3	5	9	14	
	Warm	4.4	2.3	0	2	4	8	12	
	All-Year	8.1	4.0	0	4	7	14	29	
COPD Ages 65+	Cool	8.8	4.6	1	4	8	15	29	
	Warm	7.2	3.0	0	3	7	11	17	

**Table 2. Descriptive Statistics for environmental variables**

		<b>N</b>	<b>mean</b>	<b>Std.</b>	<b>min.</b>	<b>10<sup>th</sup></b>	<b>median</b>	<b>90th</b>	<b>max.</b>
<b><i>Pollutants</i></b>									
<b><i>Measured Particles</i></b>									
PM <sub>10</sub> (µg/m <sup>3</sup> ) 24hr	All Year	823	23.3	12.9	4.0	11.4	20.0	38.3	102.3
	Cool	457	23.5	14.6	4.0	10.6	19.3	40.6	102.3
	Warm	366	22.9	10.4	5.4	12.5	20.9	36.4	71.7
PM <sub>10</sub> * (µg/m <sup>3</sup> ) 24hr	All Year	700	23.6	12.9	4.0	11.8	20.3	38.4	102.3
	Cool	334	24.4	15.1	4.0	11.2	20.0	42.9	102.3
	Warm	366	22.9	10.4	5.4	12.5	20.9	36.4	71.7
PM <sub>2.5</sub> (µg/m <sup>3</sup> ) 24hr	All Year	675	14.5	9.7	2.1	6.0	11.7	25.8	82.8
	Cool	324	16.1	11.8	2.1	5.9	12.8	29.2	82.8
	Warm	351	13.1	7.0	3.0	6.1	11.0	21.8	47.2
PM <sub>2.5-10</sub> (µg/m <sup>3</sup> ) 24hr	All Year	669	9.0	4.6	1.0	4.1	8.0	15.2	35.4
	Cool	318	7.4	4.2	1.0	3.5	7.4	13.5	28.1
	Warm	351	9.8	4.8	2.0	4.6	8.7	15.6	35.4
BS (µg/m <sup>3</sup> ) 24hr	All Year	823	13.2	9.0	1.8	5.1	10.9	23.6	71.9
	Cool	457	15.6	10.7	1.8	5.1	12.6	29.1	71.9
	Warm	366	10.3	4.7	1.9	4.9	9.9	16.0	31.5
SO <sub>4</sub> (µg/m <sup>3</sup> ) 24hr	All Year	814	3.7	3.1	0.5	1.3	2.7	7.7	22.6
	Cool	455	3.9	3.5	0.5	1.3	2.7	8.4	22.6
	Warm	359	3.5	2.4	0.7	1.4	2.7	6.9	13.2
<b><i>Modelled Particles</i></b>									
Primary (µg/m <sup>3</sup> ) 24hr	All Year	804	7.0	6.5	0.6	2.1	5.3	13.7	63.5
	Cool	451	8.5	7.8	0.6	2.4	6.2	17.1	63.5
	Warm	353	5.0	3.3	0.6	1.8	4.1	8.4	25.9
Secondary (µg/m <sup>3</sup> ) 24hr	All Year	814	8.9	7.4	1.2	3.2	6.5	18.5	54.4
	Cool	455	9.3	8.4	1.2	3.0	6.5	20.2	54.4
	Warm	359	8.4	5.8	1.7	3.4	6.5	16.6	31.9
<b><i>Gases</i></b>									
NO <sub>2</sub> (ppb) 1 hr	All Year	823	37.2	15.1	10.7	22.9	34.9	51.7	176.1
	Cool	457	37.8	15.9	10.7	24.9	35.9	49.5	176.1
	Warm	366	36.5	14.0	12.2	21.3	34.4	52.9	111.5
O <sub>3</sub> (ppb) 8 hr	All Year	823	24.0	13.8	0.4	6.7	24	37.5	89.9
	Cool	457	17.8	9.8	0.4	4.5	17.8	30.6	38.9
	Warm	366	31.6	14.3	8.7	17.9	28.3	50.4	89.9
SO <sub>2</sub> (ppb) 24 hr	All Year	823	7.2	4.7	1.9	3.3	5.8	12.3	59.8
	Cool	457	7.4	5.3	2.0	3.3	5.9	12.6	59.8
	Warm	366	6.9	3.9	1.9	3.3	5.7	11.9	25.2
CO (ppm) 8 hr	All Year	821	0.8	0.7	0.2	0.3	0.6	1.3	10.0
	Cool	457	0.9	0.9	0.2	0.4	0.7	1.7	10.0
	Warm	364	0.6	0.3	0.2	0.3	0.5	0.8	2.5
<b><i>Meteorological (Daily)</i></b>									
Temperature (°C)	All Year	823	9.3	5.2	-3.4	2.4	9.3	16.1	22.6
	Cool	457	6.4	4.0	-3.4	0.7	6.7	11.5	14.8
	Warm	366	13.0	4.1	2.6	6.7	13.3	18.1	22.6
Humidity (%)	All Year	823	78.7	10.0	51	65.5	79.5	91.5	99.0
	Cool	457	83.1	8.2	53.5	72.0	83.5	93.6	99.0
	Warm	366	73.1	9.2	51.0	62.5	72.0	85.5	97.5

**Table 3. Correlations between PM<sub>2.5</sub> and the outcome series, other pollutants and meteorological variables: all-year and by season (based on 700-day series)**

<b>Variable</b>	<b>All-Year</b>	<b>Cool Season</b>	<b>Warm Season</b>
All Cardiovascular All Ages	0.04	-0.05	0.11
Cardiac Disease All Ages	0.04	-0.06	0.12
65+	0.07	0.00	0.14
Ischaemic Heart Disease Ages 0 – 64	-0.03	-0.10	-0.01
Ages 65+	0.05	-0.04	0.11
Stroke Ages 65+	0.03	-0.04	0.05
All Respiratory All Ages	0.10	-0.03	0.14
0 – 14	0.12	0.05	0.04
15 – 64	0.02	-0.08	0.10
65+	0.05	-0.09	0.19
Asthma 0 – 14	0.05	0.07	0.02
15 – 64	-0.05	-0.15	0.05
COPD Ages 65+	0.04	-0.05	0.14
PM <sub>10</sub>	0.92	0.93	0.93
SO <sub>4</sub>	0.62	0.60	0.69
Black Smoke	0.70	0.75	0.53
PM <sub>2.5-10</sub>	0.52	0.61	0.55
PFR	0.61	0.61	0.57
SFR	0.62	0.60	0.69
NO <sub>2</sub>	0.60	0.53	0.75
O <sub>3</sub>	-0.11	-0.46	0.40
SO <sub>2</sub>	0.51	0.49	0.54
CO	0.55	0.55	0.56
Temperature	-0.15	-0.11	-0.02
Relative Humidity	0.08	0.06	-0.09

## Key to tables 4 to 7

Figures represent the percentage change in the mean number of daily admissions associated with an increase in pollutant, averaged over the same and previous days, from the 10<sup>th</sup> to 90<sup>th</sup> percentile values. The relative risk is obtained by dividing by 100 and adding 1. The daily pollutant averages, their units of measurement, their 10<sup>th</sup> to 90<sup>th</sup> percentile ranges for the whole study period and for the cool and warm seasons respectively are given below.

PM <sub>10</sub> , µg/m <sup>3</sup> , 24 hr, 24.4, 28.2, 21.6	PM <sub>2.5-10</sub> , µg/m <sup>3</sup> , 24 hr, 11.3, 9.5, 12.1	NO <sub>2</sub> , ppb, 1 hr, 25.5, 22.3, 28
PM <sub>10</sub> <sup>*</sup> , µg/m <sup>3</sup> , 24 hr, 26.6, 31.7, 24.1	SO <sub>4</sub> , 24 hr, µg/m <sup>3</sup> , 5.8, 6.3, 5.1	O <sub>3</sub> , ppb, 8 hr, 28.6, 24.1, 30.3
PM <sub>2.5</sub> , µg/m <sup>3</sup> , 24 hr, 17.7, 22.7, 15.3	PFR, 24 hr, µg/m <sup>3</sup> , 10.6, 13.1, 5.9	SO <sub>2</sub> , ppb, 24 hr, 8.5, 9.3, 7
BS, 24 hr, µg/m <sup>3</sup> , 16.7, 19.7, 10.0	SFR, 24 hr, µg/m <sup>3</sup> , 13.9, 15.2, 12.2	CO, ppm, 8 hr, 1.0, 1.1, 0.4

Cool Season - October to March, Warm season - April to September

PM<sub>10</sub><sup>\*</sup> - 700-day sub-series, PFR - Primary particle fraction, SFR - Secondary particle fraction,

BS - Black Smoke

**Table 4 Summary of results for all cardiovascular diseases and cardiac disease**

<i>Diagnosis</i>	<i>Pollutant</i>	% Change in <i>admiss. (95% CI) P-Value</i>		% Change in <i>admiss. (95% CI) P-Value</i>		% Change in <i>admiss. (95% CI) P-Value</i>		<i>(Season)</i> <i>P-Value</i>
		All Year		Cool Season		Warm Season		
All Cardiovascular All Ages	PM <sub>10</sub>	-0.6 (-2.5, 1.3)	0.5	-1.3 (-3.9, 1.2)	0.3	0.8 (-2.5, 4.2)	0.7	0.4
	PM <sub>10</sub> *	-0.8 (-3.0, 1.3)	0.4	-2.2 (-5.3, 1.0)	0.2	1.0 (-2.4, 4.5)	0.6	0.2
	PM <sub>2.5</sub>	-0.5 (-2.6, 1.6)	0.6	-1.9 (-5.0, 1.2)	0.2	1.7 (-1.9, 5.3)	0.4	0.2
	PM <sub>2.5-10</sub>	-0.7 (-3.7, 2.3)	0.2	-2.4 (-5.8, 1.1)	0.2	-0.5 (-4.2, 3.3)	0.8	0.4
	PFR	1.2 (-0.5, 3.0)	0.2	1.2 (-1.1, 3.5)	0.3	2.0 (-0.8, 4.9)	0.2	0.3
	SFR	0.3 (-1.5, 2.1)	0.8	-1.0 (-3.3, 1.3)	0.4	3.4 ( 0.2, 6.8)	0.04	0.03
	BS	1.0 (-1.0, 3.1)	0.3	0.9 (-1.7, 3.5)	0.5	2.6 (-1.0, 6.3)	0.2	0.3
	SO <sub>4</sub>	0.3 (-1.5, 2.1)	0.7	-1.0 (-3.3, 1.3)	0.4	3.4 ( 0.2, 6.8)	0.04	0.03
	NO <sub>2</sub>	0.3 (-1.4, 2.1)	0.7	0.1 (-1.8, 2.0)	0.9	0.8 (-2.4, 4.0)	0.6	0.8
	O <sub>3</sub>	0.1 (-2.7, 3.0)	0.9	1.0 (-2.7, 4.7)	0.6	-0.5 (-4.1, 3.2)	0.8	0.5
	SO <sub>2</sub>	-0.4 (-2.2, 1.5)	0.7	-0.3 (-2.6, 2.2)	0.8	-0.7 (-3.6, 2.4)	0.7	0.8
	CO	0.4 (-1.0, 1.7)	0.6	0.5 (-1.2, 2.1)	0.6	0.1 (-2.5, 2.7)	0.96	0.9
	Cardiac Disease All Ages	PM <sub>10</sub>	0.3 (-1.8, 2.4)	0.8	-0.7 (-3.5, 2.2)	0.7	2.3 (-1.3, 6.1)	0.2
PM <sub>10</sub> *		-0.6 (-3.1, 2.0)	0.7	-2.6 (-6.3, 1.2)	0.2	2.4 (-1.6, 6.7)	0.2	0.09
PM <sub>2.5</sub>		-0.4 (-2.8, 2.2)	0.8	-2.4 (-6.0, 1.3)	0.2	3.4 (-0.8, 7.7)	0.1	0.05
PM <sub>2.5-10</sub>		-0.9 (-4.3, 2.7)	0.6	-2.7 (-7.8, 2.7)	0.3	0.8 (-4.0, 5.8)	0.8	0.4
PFR		-0.7 (-3.7, 2.3)	0.1	-2.1 (-6.1, 2.1)	0.2	0.7 (-3.7, 5.3)	0.1	0.3
SFR		0.8 (-1.2, 2.9)	0.4	-0.5 (-3.1, 2.0)	0.7	4.3 ( 0.6, 8.1)	0.02	0.03
BS		1.7 (-0.6, 3.9)	0.2	1.7 (-1.1, 4.6)	0.2	3.2 (-0.7, 7.3)	0.1	0.3
SO <sub>4</sub>		0.9 (-1.2, 2.9)	0.4	-0.5 (-3.0, 2.1)	0.7	4.3 ( 0.6, 8.1)	0.02	0.03
NO <sub>2</sub>		1.1 (-0.8, 3.0)	0.3	0.6 (-1.4, 2.7)	0.6	2.1 (-1.4, 5.7)	0.2	0.5
O <sub>3</sub>		1.6 (-1.5, 4.9)	0.3	-0.5 (-4.6, 3.7)	0.8	2.8 (-1.4, 7.1)	0.2	0.3
SO <sub>2</sub>		0.7 (-1.3, 2.8)	0.5	0.5 (-2.2, 3.2)	0.7	1.2 (-2.2, 4.6)	0.5	0.7
CO		0.9 (-0.6, 2.4)	0.2	1.2 (-0.6, 3.0)	0.2	0.3 (-2.4, 3.1)	0.8	0.9
Cardiac Disease Ages 65+		PM <sub>10</sub>	2.0 (-0.8, 4.9)	0.2	1.2 (-2.6, 5.0)	0.6	4.2 (-0.6, 9.2)	0.09
	PM <sub>10</sub> *	1.5 (-1.5, 4.5)	0.3	-0.3 (-4.6, 4.3)	0.9	4.6 (-0.2, 9.5)	0.06	0.1
	PM <sub>2.5</sub>	1.0 (-1.9, 3.9)	0.5	-0.4 (-4.7, 4.0)	0.9	4.0 (-0.9, 9.1)	0.1	0.1
	PM <sub>2.5-10</sub>	3.0 (-0.6, 6.6)	0.1	1.1 (-3.7, 6.1)	0.7	4.7 (-0.5, 10.2)	0.08	0.4
	PFR	2.3 (-0.3, 4.9)	0.08	2.4 (-0.9, 5.9)	0.2	3.4 (-0.7, 7.7)	0.1	0.3
	SFR	1.4 (-1.2, 4.1)	0.3	0.9 (-2.4, 4.2)	0.6	2.8 (-1.8, 7.7)	0.2	0.4
	BS	3.3 ( 0.3, 6.4)	0.03	3.6 (-0.2, 7.5)	0.06	4.6 (-0.6, 10.1)	0.08	0.3
	SO <sub>4</sub>	1.5 (-1.2, 4.1)	0.3	0.9 (-2.4, 4.2)	0.6	2.9 (-1.8, 7.8)	0.2	0.4
	NO <sub>2</sub>	1.8 (-0.7, 4.3)	0.2	1.8 (-0.9, 4.6)	0.2	1.3 (-3.2, 6.0)	0.6	0.7
	O <sub>3</sub>	-2.0 (-5.9, 2.0)	0.3	-3.9 (-8.7, 1.3)	0.1	-1.2 (-6.1, 4.0)	0.7	0.3
	SO <sub>2</sub>	2.8 ( 0.0, 5.6)	0.05	2.6 (-0.9, 6.3)	0.2	3.1 (-1.3, 7.7)	0.2	0.7
	CO	1.8 (-0.2, 3.8)	0.07	2.2 (-0.2, 4.6)	0.07	0.5 (-3.1, 4.3)	0.8	0.9

**Table 5 Summary of results for ischaemic heart disease and stroke in the over 65 age group**

<i>Diagnosis</i>	<i>Pollutant</i>	<i>% Change in admiss. (95% CI) P-Value</i>		<i>% Change in admiss. (95% CI) P-Value</i>		<i>% Change in admiss. (95% CI) P-Value</i>		<i>(Seasonal P-Value)</i>	
		<i>All Year</i>		<i>Cool Season</i>		<i>Warm Season</i>			
IHD Ages 65+	PM <sub>10</sub>	2.1 ( -2.0, 6.3)	0.3	0.3 ( -5.1, 5.9)	0.9	6.4 ( -0.7, 14.1)	0.08	0.1	
	PM <sub>10</sub> *	0.6 ( -3.8, 5.2)	0.8	-3.2 ( -9.5, 3.4)	0.3	6.9 ( -0.5, 14.8)	0.07	0.04	
	PM <sub>2.5</sub>	-0.3 ( -4.5, 4.2)	0.9	-4.1 ( -10.2, 2.4)	0.2	6.8 ( -0.7, 14.9)	0.08	0.03	
	PM <sub>2.5-10</sub>	1.1 ( -4.1, 6.6)	0.7	-0.8 ( -7.8, 6.7)	0.8	3.5 ( -4.6, 12.1)	0.4	0.5	
	PFR	1.3 ( -2.4, 5.1)	0.5	0.0 ( -4.9, 5.1)	0.99	4.6 ( -1.5, 10.9)	0.1	0.2	
	SFR	2.1 ( -1.8, 6.1)	0.3	1.0 ( -3.8, 6.1)	0.7	4.9 ( -2.0, 12.2)	0.2	0.3	
	BS	2.0 ( -2.3, 6.4)	0.4	1.4 ( -4.0, 7.0)	0.6	4.1 ( -3.4, 12.2)	0.3	0.4	
	SO <sub>4</sub>	2.1 ( -1.8, 6.1)	0.3	1.1 ( -3.8, 6.1)	0.7	4.9 ( -2.0, 12.3)	0.2	0.3	
	NO <sub>2</sub>	2.3 ( -1.4, 6.2)	0.2	1.2 ( -2.8, 5.4)	0.6	4.0 ( -2.7, 11.2)	0.2	0.6	
	O <sub>3</sub>	0.8 ( -4.7, 6.7)	0.8	-4.8 ( -11.6, 2.5)	0.2	6.5 ( -1.3, 14.8)	0.1	0.04	
	SO <sub>2</sub>	1.5 ( -2.5, 5.6)	0.5	2.2 ( -2.9, 7.5)	0.4	0.4 ( -5.9, 7.0)	0.9	0.7	
	CO	1.9 ( -1.0, 4.9)	0.2	1.7 ( -1.8, 5.3)	0.4	3.7 ( -1.8, 9.4)	0.2	0.3	
	Stroke Ages 65+	PM <sub>10</sub>	-3.3 ( -7.9, 1.4)	0.2	-3.7 ( -9.6, 2.7)	0.3	-3.9 ( -11.8, 4.6)	0.4	0.8
		PM <sub>10</sub> *	-3.0 ( -8.1, 2.4)	0.3	-4.1 ( -11.3, 3.7)	0.3	-3.5 ( -11.5, 5.3)	0.4	0.9
PM <sub>2.5</sub>		-1.6 ( -6.6, 3.6)	0.5	-4.2 ( -11.3, 3.4)	0.3	-0.4 ( -8.9, 9.0)	0.9	0.6	
PM <sub>2.5-10</sub>		-8.2 ( -13.9, -2.2)	0.008	-4.6 ( -12.5, 4.0)	0.3	-10.4 ( -18.6, -1.4)	0.03	0.4	
PFR		-1.3 ( -5.6, 3.2)	0.6	-3.8 ( -9.4, 2.1)	0.2	0.1 ( -7.0, 7.8)	0.98	0.6	
SFR		2.0 ( -2.5, 6.7)	0.4	1.4 ( -4.0, 7.2)	0.6	3.4 ( -4.8, 12.3)	0.4	0.6	
BS		-2.7 ( -7.6, 2.4)	0.3	-5.5 ( -11.5, 0.8)	0.08	0.3 ( -8.6, 9.9)	0.96	0.5	
SO <sub>4</sub>		2.0 ( -2.5, 6.7)	0.4	1.5 ( -4.0, 7.2)	0.6	3.4 ( -4.9, 12.4)	0.4	0.7	
NO <sub>2</sub>		-0.8 ( -5.0, 3.6)	0.7	-1.6 ( -6.1, 3.2)	0.5	0.9 ( -6.9, 9.3)	0.8	0.6	
O <sub>3</sub>		-0.5 ( -6.4, 5.8)	0.9	7.5 ( -1.1, 16.8)	0.09	-1.8 ( -10.0, 7.1)	0.7	0.1	
SO <sub>2</sub>		-5.1 ( -9.6, -0.4)	0.03	-5.3 ( -11.0, 0.7)	0.08	-3.8 ( -11.2, 4.2)	0.3	0.9	
CO		-1.6 ( -5.0, 1.9)	0.4	-2.3 ( -6.3, 1.9)	0.3	-5.1 ( -11.5, 1.8)	0.1	0.2	

**Table 6 Summary of results for all respiratory diseases**

Diagnosis	Pollutant	% Change in admiss. (95% CI) P-Value		% Change in admiss. (95% CI) P-Value		% Change in admiss. (95% CI) P-Value		(Season)
								P-Value
All Respiratory All Ages		All Year		Cool Season		Warm Season		
	PM <sub>10</sub>	1.5 ( -0.7 , 3.6)	0.2	1.0 ( -1.8, 3.8)	0.5	3.8 ( -0.3, 8.0)	0.07	0.2
	PM <sub>10</sub> <sup>*</sup>	1.4 ( -0.9, 3.8)	0.2	1.3 ( -1.9, 4.7)	0.4	2.6 ( -1.4, 6.7)	0.2	0.5
	PM <sub>2.5</sub>	1.2 ( -0.9, 3.4)	0.3	0.6 ( -2.5, 3.8)	0.7	4.2 ( 0.1, 8.5)	0.04	0.1
	PM <sub>2.5-10</sub>	0.2 ( -2.5, 3.1)	0.9	1.3 ( -2.4, 5.0)	0.5	-0.7 ( -4.9, 3.8)	0.8	0.5
	PFR	1.6 ( -0.2, 3.5)	0.08	1.9 ( -0.5, 4.4)	0.1	3.8 ( 0.4, 7.3)	0.03	0.1
	SFR	0.8 ( -1.3, 2.9)	0.5	-0.4 ( -3.0, 2.2)	0.7	3.8 ( -0.3, 8.0)	0.07	0.08
	BS	2.1 ( -0.1, 4.2)	0.06	2.3 ( -0.3, 5.1)	0.09	5.2 ( 0.9, 9.7)	0.02	0.08
	SO <sub>4</sub>	0.8 ( -1.3, 2.9)	0.4	-0.4 ( -2.9, 2.2)	0.8	3.9 ( -0.2, 8.1)	0.06	0.08
	NO <sub>2</sub>	1.7 ( -0.2, 3.7)	0.09	0.5 ( -1.5, 2.5)	0.6	6.2 ( 2.2, 10.4)	0.002	0.02
	O <sub>3</sub>	-2.4 ( -5.3, 0.6)	0.1	-4.5 ( -8.0, -0.8)	0.02	-0.6 ( -4.9, 3.9)	0.8	0.1
	SO <sub>2</sub>	1.3 ( -0.7, 3.4)	0.2	1.3 ( -1.2, 3.8)	0.3	2.6 ( -1.1, 6.4)	0.2	0.5
	CO	0.3 ( -1.1, 1.7)	0.7	0.5 ( -1.2, 2.3)	0.6	1.1 ( -2.0, 4.3)	0.5	0.6
All Respiratory Ages 0-14	PM <sub>10</sub>	3.9 ( 0.6, 7.4)	0.02	4.5 ( 0.2, 8.9)	0.04	4.2 ( -2.5, 11.4)	0.2	0.8
	PM <sub>10</sub> <sup>*</sup>	4.1 ( 0.4, 8.0)	0.03	5.3 ( 0.1, 10.8)	0.05	3.8 ( -2.9, 11.1)	0.3	0.99
	PM <sub>2.5</sub>	3.4 ( -0.1, 7.0)	0.05	4.1 ( -0.9, 9.4)	0.1	5.0 ( -2.0, 12.5)	0.2	0.6
	PM <sub>2.5-10</sub>	4.4 ( -0.3, 9.4)	0.07	5.8 ( -0.2, 12.0)	0.06	3.3 ( -4.4, 11.6)	0.4	0.5
	PFR	2.9 ( 0.1, 5.7)	0.04	3.1 ( -0.5, 6.9)	0.09	5.8 ( 0.3, 11.7)	0.04	0.1
	SFR	1.5 ( -1.7, 4.8)	0.4	1.7 ( -2.3, 5.8)	0.4	0.2 ( -6.3, 7.0)	0.97	0.7
	BS	3.9 ( 0.7, 7.3)	0.02	4.4 ( 0.3, 8.5)	0.03	7.4 ( 0.4, 14.9)	0.04	0.2
	SO <sub>4</sub>	1.5 ( -1.7, 4.9)	0.4	1.7 ( -2.2, 5.9)	0.4	0.2 ( -6.3, 7.1)	0.95	0.8
	NO <sub>2</sub>	2.3 ( -0.6, 5.3)	0.1	1.8 ( -1.1, 4.8)	0.2	4.5 ( -1.9, 11.4)	0.2	0.6
	O <sub>3</sub>	-5.2 ( -9.7, -0.5)	0.03	-4.7 ( -10.0, 1.0)	0.1	-5.9 ( -12.6, 1.3)	0.1	0.99
	SO <sub>2</sub>	4.6 ( 1.4, 7.8)	0.004	3.8 ( 0.1, 7.7)	0.05	8.7 ( 2.4, 15.4)	0.006	0.1
	CO	1.5 ( -0.6, 3.6)	0.2	1.8 ( -0.7, 4.3)	0.2	2.4 ( -2.7, 7.8)	0.4	0.5
	All Respiratory Ages 15-64	PM <sub>10</sub>	0.1 ( -4.0, 4.4)	0.96	0.6 ( -4.9, 6.3)	0.8	-0.8 ( -7.8, 6.7)	0.8
PM <sub>10</sub> <sup>*</sup>		-2.5 ( -6.9, 2.2)	0.3	-4.6 ( -11.0, 2.3)	0.2	0.4 ( -6.8, 8.0)	0.9	0.4
PM <sub>2.5</sub>		-2.1 ( -6.4, 2.4)	0.4	-4.4 ( -10.5, 2.1)	0.2	1.7 ( -5.8, 9.7)	0.7	0.3
PM <sub>2.5-10</sub>		-4.9 ( -9.9, 0.4)	0.07	-2.2 ( -9.2, 5.3)	0.6	-6.9 ( -14.0, 0.9)	0.08	0.5
PFR		2.0 ( -1.7, 5.8)	0.3	2.6 ( -2.3, 7.8)	0.3	0.9 ( -5.3, 7.6)	0.8	0.9
SFR		0.5 ( -3.4, 4.6)	0.8	-1.0 ( -5.9, 4.2)	0.7	3.8 ( -3.5, 11.6)	0.3	0.3
BS		1.2 ( -3.1, 5.6)	0.6	1.1 ( -4.3, 6.7)	0.7	2.9 ( -4.9, 11.4)	0.5	0.6
SO <sub>4</sub>		0.5 ( -3.4, 4.7)	0.8	-1.0 ( -5.9, 4.2)	0.7	3.9 ( -3.5, 11.7)	0.3	0.3
NO <sub>2</sub>		0.0 ( -3.7, 3.8)	0.997	-0.5 ( -4.5, 3.7)	0.8	1.2 ( -5.6, 8.5)	0.7	0.7
O <sub>3</sub>		-2.8 ( -7.7, 2.4)	0.3	-7.7 ( -14.0, -0.9)	0.03	0.6 ( -6.8, 8.5)	0.9	0.08
SO <sub>2</sub>		-0.9 ( -4.8, 3.3)	0.7	0.0 ( -5.0, 5.3)	0.99	-2.7 ( -9.2, 4.2)	0.4	0.5
CO		-0.7 ( -3.6, 2.3)	0.7	-0.1 ( -3.6, 3.5)	0.96	-4.7 ( -10.2, 1.2)	0.1	0.1
All Respiratory Ages 65+		PM <sub>10</sub>	-1.1 ( -4.3, 2.1)	0.5	-4.4 ( -8.5, -0.1)	0.05	7.5 ( 1.3, 14.1)	0.02
	PM <sub>10</sub> <sup>*</sup>	-1.1 ( -4.6, 2.6)	0.6	-6.0 ( -10.9, -0.8)	0.02	8.5 ( 2.1, 15.3)	0.008	0.0008
	PM <sub>2.5</sub>	-1.3 ( -4.7, 2.2)	0.5	-5.7 ( -10.5, -0.6)	0.03	8.7 ( 2.2, 15.7)	0.008	0.0009
	PM <sub>2.5-10</sub>	-1.9 ( -6.0, 2.5)	0.4	-7.1 ( -12.4, -1.5)	0.01	5.1 ( -1.7, 12.3)	0.1	0.007
	PFR	-0.1 ( -2.8, 2.8)	0.97	-0.7 ( -4.4, 3.1)	0.7	5.4 ( 0.1, 11.0)	0.04	0.04
	SFR	-1.3 ( -4.4, 2.0)	0.4	-4.9 ( -8.8, -0.9)	0.02	9.0 ( 2.5, 15.9)	0.006	0.0005
	BS	-0.3 ( -3.5, 3.0)	0.9	-1.1 ( -5.2, 3.1)	0.6	8.1 ( 1.4, 15.4)	0.02	0.01
	SO <sub>4</sub>	-1.3 ( -4.4, 2.0)	0.5	-4.9 ( -8.7, -0.9)	0.02	9.0 ( 2.5, 16.0)	0.006	0.0005
	NO <sub>2</sub>	1.0 ( -1.8, 3.9)	0.5	-0.8 ( -3.7, 2.2)	0.6	7.9 ( 2.0, 14.1)	0.009	0.01
	O <sub>3</sub>	0.2 ( -4.1, 4.8)	0.9	-2.2 ( -7.5, 3.4)	0.4	1.6 ( -5.1, 8.6)	0.7	0.4
	SO <sub>2</sub>	-2.0 ( -4.9, 1.1)	0.2	-2.4 ( -6.1, 1.4)	0.2	-0.1 ( -5.4, 5.6)	0.98	0.5
	CO	0.0 ( -2.1, 2.1)	0.99	0.0 ( -2.6, 2.6)	0.98	2.7 ( -2.2, 7.9)	0.3	0.3

**Table 7 Summary of results for asthma and COPD**

<i>Diagnosis</i>	<i>Pollutant</i>	<i>% Change in admiss. (95% CI) P-Value</i>		<i>% Change in admiss. (95% CI) P-Value</i>		<i>% Change in admiss. (95% CI) P-Value</i>		<i>(Season P-Value)</i>	
		<i>All Year</i>		<i>Cool Season</i>		<i>Warm Season</i>			
Asthma Ages 0-14	PM <sub>10</sub>	8.3 ( 1.7, 15.3)	0.01	7.9 ( -0.6, 17.1)	0.07	13.0 ( 0.9, 26.5)	0.03	0.3	
	PM <sub>10</sub> <sup>*</sup>	7.9 ( 0.4, 15.9)	0.04	8.3 ( -2.5, 20.4)	0.1	9.4 ( -2.7, 23.0)	0.1	0.7	
	PM <sub>2.5</sub>	6.0 ( -0.9, 13.4)	0.09	3.7 ( -6.4, 14.9)	0.5	14.2 ( 1.5, 28.4)	0.03	0.1	
	PM <sub>2.5-10</sub>	7.1 ( -2.1, 17.2)	0.1	7.5 ( -4.5, 21.0)	0.2	5.7 ( -7.8, 21.2)	0.4	0.8	
	PFR	6.8 ( 1.2, 12.7)	0.02	7.3 ( -0.2, 15.4)	0.06	9.6 ( 0.9, 19.0)	0.03	0.2	
	SFR	2.0 ( -4.0, 8.4)	0.5	4.3 ( -3.3, 12.5)	0.3	-0.4 (-11.1, 11.7)	0.95	0.6	
	BS	7.4 ( 0.7, 14.5)	0.03	7.6 ( -0.8, 16.6)	0.08	10.0 ( -1.3, 22.6)	0.09	0.3	
	SO <sub>4</sub>	2.1 ( -3.9, 8.6)	0.5	4.4 ( -3.3, 12.6)	0.3	-0.2 (-11.0, 12.0)	0.98	0.6	
	NO <sub>2</sub>	4.0 ( -2.0, 10.2)	0.2	3.1 ( -3.1, 9.8)	0.3	4.8 ( -6.2, 17.1)	0.4	0.9	
	O <sub>3</sub>	-12.9 (-21.2, -3.8)	0.007	-9.8 (-20.0, 1.8)	0.09	-15.6 (-26.5, -3.0)	0.02	0.7	
	SO <sub>2</sub>	10.9 ( 4.5, 17.8)	0.0007	7.9 ( 0.0, 16.5)	0.05	19.3 ( 7.9, 31.9)	0.0006	0.05	
	CO	3.9 ( -0.5, 8.5)	0.08	3.6 ( -1.7, 9.2)	0.2	7.7 ( 0.4, 15.6)	0.04	0.1	
	Asthma Ages 15-64	PM <sub>10</sub>	-2.3 (-10.0, 6.1)	0.6	-3.7 (-13.8, 7.5)	0.5	0.6 (-12.8, 16.0)	0.9	0.7
		PM <sub>10</sub> <sup>*</sup>	-7.7 (-16.0, 1.4)	0.1	-13.3 (-24.7, 0.0)	0.05	0.1 (-13.3, 15.5)	0.99	0.3
PM <sub>2.5</sub>		-8.4 (-16.3, 0.3)	0.06	-14.5 (-25.5, -1.9)	0.03	0.8 (-13.3, 17.2)	0.9	0.2	
PM <sub>2.5-10</sub>		-10.7 (-19.9, -0.5)	0.04	-12.9 (-25.4, 1.7)	0.08	-7.5 (-21.1, 8.5)	0.3	0.5	
PFR		-0.8 ( -7.8, 6.7)	0.8	0.4 ( -8.9, 10.6)	0.9	-6.5 (-18.1, 6.7)	0.3	0.3	
SFR		-4.2 (-11.6, 3.9)	0.3	-6.0 (-15.2, 4.3)	0.2	-0.7 (-14.0, 14.5)	0.9	0.6	
BS		-2.8 (-10.7, 5.8)	0.5	-1.2 (-11.2, 9.9)	0.8	-11.6 (-24.7, 3.7)	0.1	0.2	
SO <sub>4</sub>		-4.2 (-11.7, 3.9)	0.3	-6.0 (-15.2, 4.2)	0.2	-0.7 ( -14.0, 4.7)	0.9	0.6	
NO <sub>2</sub>		-3.3 (-10.4, 4.4)	0.4	-3.3 (-11.0, 5.1)	0.4	-2.3 (-15.0, 12.4)	0.7	0.8	
O <sub>3</sub>		-1.7 (-11.2, 8.9)	0.8	-3.7 (-16.3, 10.7)	0.6	-0.2 (-13.8, 15.6)	0.98	0.7	
SO <sub>2</sub>	2.4 ( -5.5, 10.9)	0.6	3.6 ( -6.2, 14.5)	0.5	1.0 (-11.9, 15.8)	0.9	0.8		
CO	-4.9 (-10.6, 1.1)	0.1	-4.2 (-10.9, 3.1)	0.3	-12.5 (-22.6, -1.0)	0.03	0.07		
COPD Ages 65+	PM <sub>10</sub>	-1.8 ( -6.9, 3.5)	0.5	-5.0 (-11.5, 2.1)	0.2	5.3 ( -4.3, 15.7)	0.3	0.1	
	PM <sub>10</sub> <sup>*</sup>	-2.9 ( -8.3, 2.9)	0.3	-7.2 (-14.9, 1.1)	0.09	4.9 ( -4.5, 15.2)	0.3	0.08	
	PM <sub>2.5</sub>	-3.9 ( -9.0, 1.6)	0.2	-8.4 (-15.7, -0.3)	0.04	4.8 ( -4.8, 15.3)	0.3	0.07	
	PM <sub>2.5-10</sub>	-1.7 ( -8.9, 5.3)	0.6	-4.7 (-13.2, 4.6)	0.3	1.5 ( -8.5, 12.6)	0.8	0.4	
	PFR	1.2 ( -3.2, 5.8)	0.6	0.6 ( -5.2, 6.8)	0.8	6.2 ( -1.9, 14.9)	0.1	0.2	
	SFR	-0.3 ( -5.3, 4.9)	0.9	-4.5 (-10.6, 2.0)	0.2	9.9 ( 0.1, 20.6)	0.05	0.02	
	BS	1.5 ( -3.7, 7.0)	0.6	0.9 ( -5.6, 7.8)	0.8	8.2 ( -2.1, 19.5)	0.1	0.2	
	SO <sub>4</sub>	-0.3 ( -5.3, 4.9)	0.9	-4.5 (-10.6, 2.0)	0.2	9.9 ( 0.1, 20.8)	0.05	0.02	
	NO <sub>2</sub>	2.5 ( -2.1, 7.3)	0.3	1.0 ( -3.8, 6.0)	0.7	6.7 ( -2.4, 16.7)	0.2	0.3	
	O <sub>3</sub>	0.2 ( -7.0, 8.0)	0.96	-2.8 (-11.5, 6.7)	0.6	1.9 ( -8.5, 13.4)	0.7	0.5	
SO <sub>2</sub>	-4.2 ( -8.9, 0.8)	0.1	-4.7 (-10.6, 1.6)	0.1	-3.9 (-11.9, 4.9)	0.4	0.99		
CO	1.0 ( -2.5, 4.6)	0.6	1.5 ( -2.7, 5.9)	0.5	-1.4 ( -8.7, 6.5)	0.7	0.6		

**Table 8 Dual pollutant models for one particle measure in the presence of another. Percentage change in relative risk for a 10<sup>th</sup>-90<sup>th</sup> centile increase in row pollutant, in presence of column pollutant, 95% confidence interval and P-value**

Outcome	Pollutant	Single	PM <sub>2.5</sub>	PM <sub>2.5-10</sub>	SO <sub>4</sub>	Black Smoke
All Cardiovascular Diseases, All Ages	PM <sub>2.5</sub>	-0.5 (-2.6, 1.6) P = 0.6	*****	0.6 (-2.0, 3.2) P = 0.7	-1.5 (-4.2, 1.3) P = 0.3	-3.2 (-6.0, -0.2) P = 0.04
	PM <sub>2.5-10</sub>	-0.7 (-3.7, 2.3) P = 0.2	-2.2 (-5.3, 0.9) P = 0.2	*****	-2.6 (-5.4, 0.2) P = 0.07	-2.7 (-5.3, 0.0) P = 0.05
	SO <sub>4</sub>	0.3 (-1.5, 2.1) P = 0.7	1.4 (-1.3, 4.1) P = 0.3	1.4 (-0.9, 3.7) P = 0.2	*****	0.0 (-2.1, 2.1) P = 0.98
	Black Smoke	1.0 (-1.0, 3.1) P = 0.3	3.9 (0.7, 7.3) P = 0.02	2.2 (-0.1, 4.6) P = 0.06	1.4 (-0.9, 3.8) P = 0.2	*****
Cardiac Disease, All Ages	PM <sub>2.5</sub>	-0.4 (-2.8, 2.2) P = 0.8	*****	0.2 (-2.8, 3.3) P = 0.9	-0.7 (-3.8, 2.6) P = 0.7	-3.2 (-6.6, 0.3) P = 0.07
	PM <sub>2.5-10</sub>	-0.9 (-4.3, 2.7) P = 0.6	-1.2 (-4.8, 2.6) P = 0.5	*****	-1.3 (-4.7, 2.1) P = 0.4	-1.9 (-5.0, 1.3) P = 0.2
	SO <sub>4</sub>	0.9 (-1.2, 2.9) P = 0.4	0.4 (-2.7, 3.6) P = 0.8	0.5 (-2.2, 3.3) P = 0.7	*****	-0.6 (-3.0, 2.0) P = 0.7
	Black Smoke	1.7 (-0.6, 3.9) P = 0.2	4.2 (0.4, 8.2) P = 0.03	2.3 (-0.5, 5.1) P = 0.1	1.9 (-0.8, 4.7) P = 0.2	*****
All Respiratory Diseases, All Ages	PM <sub>2.5</sub>	1.2 (-0.9, 3.4) P = 0.3	*****	1.8 (-0.9, 4.6) P = 0.2	0.9 (-1.9, 3.7) P = 0.6	-1.7 (-4.9, 1.6) P = 0.3
	PM <sub>2.5-10</sub>	0.2 (-2.5, 3.1) P = 0.9	-1.6 (-5.0, 2.0) P = 0.4	*****	-0.9 (-4.1, 2.4) P = 0.6	-1.5 (-4.4, 1.6) P = 0.3
	SO <sub>4</sub>	0.8 (-1.3, 2.9) P = 0.4	0.5 (-2.3, 3.5) P = 0.7	1.4 (-1.2, 4.0) P = 0.3	*****	0.3 (-2.1, 2.6) P = 0.8
	Black Smoke	2.1 (-0.1, 4.2) P = 0.06	3.8 (0.5, 7.3) P = 0.03	2.9 (0.5, 5.3) P = 0.02	2.4 (0.1, 4.7) P = 0.04	*****
All Respiratory Disease, Ages 0-14	PM <sub>2.5</sub>	3.4 (-0.1, 7.0) P = 0.05	*****	3.0 (-1.3, 7.4) P = 0.2	3.9 (-0.5, 8.4) P = 0.08	1.6 (-3.5, 7.1) P = 0.5
	PM <sub>2.5-10</sub>	4.4 (-0.3, 9.4) P = 0.07	2.9 (-2.9, 9.0) P = 0.33	*****	4.0 (-1.5, 9.7) P = 0.2	3.5 (-1.6, 8.8) P = 0.2
	SO <sub>4</sub>	1.5 (-1.7, 4.9) P = 0.4	0.4 (-4.1, 5.0) P = 0.9	1.6 (-2.6, 5.8) P = 0.5	*****	1.5 (-2.2, 5.4) P = 0.4
	Black Smoke	3.9 (0.7, 7.3) P = 0.02	3.3 (-1.9, 8.8) P = 0.2	3.6 (0.0, 7.4) P = 0.05	4.1 (0.6, 7.7) P = 0.02	*****

NB: All models were fitted for the 700-day series except for the single-pollutant models for SO<sub>4</sub> and Black Smoke

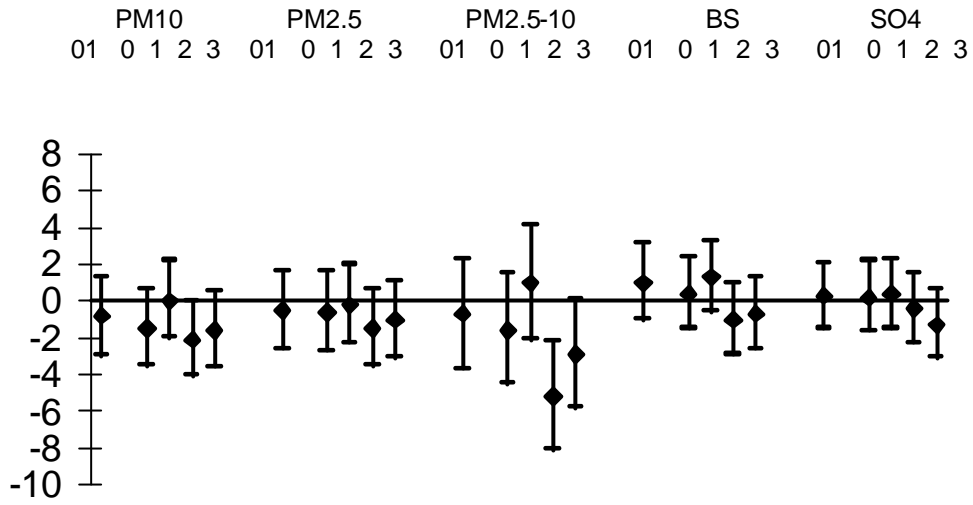
**Table 9 Comparison of results with a London study of emergency hospital admissions**

Results are from all-year analyses. The table shows the percentage change in admissions (95% confidence interval) associated with an increase of  $10 \mu\text{g}/\text{m}^3$  in the cumulative mean(0,1) measure (for comparability of the two studies) of  $\text{PM}_{10}$  and BS.

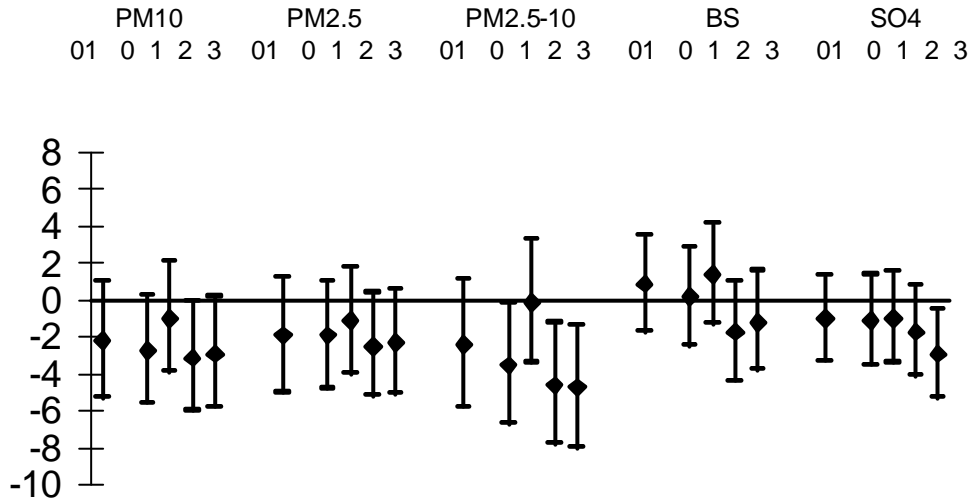
Outcome	Pollutant	Present Study	London [7]
All Cardiovascular, All Ages	$\text{PM}_{10}$	-0.3 (-1.0, 0.5)	0.6 ( 0.1, 1.1)
	BS	0.6 (-0.6, 1.9)	1.1 ( 0.1, 2.0)
All Respiratory, All Ages	$\text{PM}_{10}$	0.6 (-0.3, 1.5)	1.3 ( 0.5, 2.0)
	BS	1.2 (-0.1, 2.5)	1.0 (-0.2, 2.2)

Figure 1. All Cardiovascular Disease – Percentage change in daily number of emergency admissions associated with cumulative (lag 0,1) and single day lags (0-3) for PM2.5, PM10, PM2.5-10, Black Smoke and Sulphate

All-Year



Cool Season



Warm Season

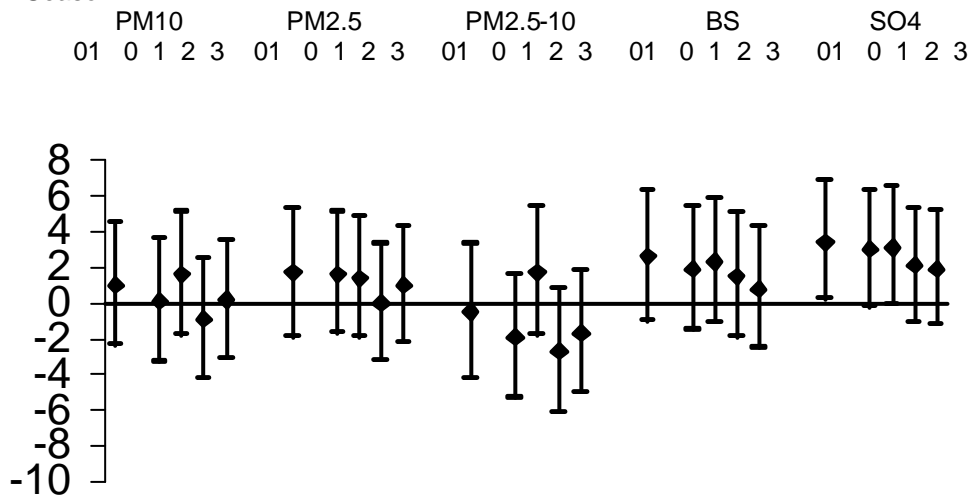


Figure 2. All Respiratory Disease – Percentage change in daily number of emergency admissions associated with cumulative (lag 0,1) and single day lags (0-3) for PM2.5, PM10, PM2.5-10, Black Smoke and Sulphate.

