

**PM_{2.5} AND DAILY MORTALITY IN THE WEST
MIDLANDS CONURBATION, UK**

REPORT

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BACKGROUND

A large number of epidemiological investigations have now reported associations between measures of particles and daily mortality and morbidity.[1,2] These associations are small and often it is not possible to separate the effects of particles from those of gases with which they are associated. Nevertheless, these associations are generally accepted as causal, at least in part, for public health purposes. The toxic component of the particle mixture is also debated. Arguments are driven mainly by theoretical possibilities with some corroboration from experimental studies, mainly in animals. There is little epidemiological evidence to test these ideas because of the lack of adequate time series of particle data partitioned by size, number, chemical composition and source.

On the basis of composition and source, PM_{10} can be divided by size of particle into the coarse fraction ($>2.5\mu m$) and the fine fraction ($<2.5\mu m$). The coarse fraction mainly arises from re-suspension of surface dust by human activity or wind, or from sea salt in coastal areas. The fine fraction is divided into "primary" and "secondary" categories. The primary fraction refers to particles arising directly from combustion sources; in the UK these are mainly traffic. The secondary fine fraction refers to particles created by chemical reactions in the air; these mainly comprise salts such as sulphates and nitrates. In the UK these are usually neutral but in some regions (such as the Eastern USA) may be in the form of acids. A study of the chemical composition of fine particles obtained in Leeds found that about half was carbonaceous material, one quarter sulphates and the remainder nitrates and other substances.[3]

At present there are theoretical and toxicological arguments in favour of the fine ($<PM_{2.5}$) fraction containing the most toxic element.[4] Epidemiological evidence, mainly from the US is accumulating which supports this view. A comprehensive investigation among a number of north American cities compared the effects of sulphates, H^+ , $PM_{2.5}$ and $PM_{2.5-10}$ on daily mortality and concluded that $PM_{2.5}$ was the most important.[5] The composition of fine

particles in the Eastern North America is likely to differ in respect of the acidity of secondary particles and the origin of primary particles (because diesel engines are used less in cars and light goods vehicles). No analysis of $PM_{2.5}$ and daily mortality has been reported from a European country.

The measure of ambient particles currently subject to standards is PM_{10} , an estimate of the weight of particles of aerodynamic diameter $<10\mu m$. In the UK, the Expert Panel on Air Quality Standards (EPAQS) has recommended a standard of $50\mu g/m^3$ as a 24 hour running average.[6] The UK National Air Quality Strategy has, as its current objective, a level of $50\mu g/m^3$ as the 99th percentile of 24hr running means.

EPAQS is considering the case for setting a standard for $PM_{2.5}$ but lacks any epidemiological evidence from a UK or similar environment upon which to base any recommendations.

$PM_{2.5}$ has been measured in Birmingham since October 1994 and we were commissioned by the Department of Health (on behalf of EPAQS), to investigate its association with daily mortality and daily hospital admissions. This report concerns the analyses of daily mortality.

QUESTIONS TO BE ADDRESSED

1. Is $PM_{2.5}$ associated with daily mortality?
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 mortality? 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 and from those of pollutant gases (NO₂, SO₂, O₃)?
6. What are the exposure response relationships?

METHODS

MORTALITY DATA

Counts of daily deaths were constructed for the period October 1994 to December 1996 (823 days) from mortality files supplied by the Office for National Statistics. Deaths from all causes, excluding accidents and other external causes (ICD9: <800), cardiovascular (390-459) and respiratory (460-519) diseases for those aged 65 and over and for all ages combined were investigated, a total of 6 separate time series. Only deaths of those people resident in, and dying in the study area were included. The study areas included 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 PM₁₀, PM_{2.5}, black smoke (BS), sulphate and an estimate of the coarse fraction (PM_{2.5-10}) derived by subtracting the PM_{2.5} from the PM₁₀ measures. In addition, estimates of primary (PFR), secondary (SFR) and coarse (CFR) particle fractions were studied.[7] These estimates were derived from a statistical model relating PM₁₀, PM_{2.5}, BS and sulphate measurements in the UK between January and March 1996. Also studied were nitrogen dioxide, ozone, sulphur dioxide and carbon monoxide

Only those pollution monitoring stations able to provide data for at least 75% of days during the study period were used, a criterion adopted from the APHEA

project. 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 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. For NO_2 , O_3 , SO_2 and CO, four, two, five and two stations respectively provided data for the computation of study-wide daily averages. 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 as indicators of average daily meteorological conditions.

STATISTICAL METHODS

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

Temporal patterns were described using non-parametric 'smoothes' of time. A smoother is a tool for summarising the trend in a variable (mortality) as a function of another (time). The relationship between the two variables is determined by the data themselves rather than as a rigid form as in linear regression. The smoother used in this study was a locally weighted smoother called 'loess'. It calculates a weighted average of each response value based upon a number (span) of 'local' predictor (date) values.

As in parametric methods it is important not to over- or under-control for temporal patterns. The degree of control is determined by the span of the smooth. For the six series studied the spans ranged from 110 to 140 days,

therefore removing any temporal associations with mortality over a longer time period. 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 (ICD9: 487) were used to control for the potential confounding effect of respiratory infections.

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) (same day, 1 day and 2 days) and cumulative lags (mean of current and previous days, mean of current and previous two days and mean of previous two days). A smooth was fitted to the data and plotted to assess the shape of the relationship.

Control for temperature was made by choosing the single or cumulative lag which showed the strongest relationship graphically and then fitting a loess smooth of temperature to the model. If the approximate F-test for the non-parametric part of the model showed no evidence of non-linearity, a parametric term for temperature e.g. linear or piecewise-linear was tried. Subsequent to adequate temperature control, relative humidity was assessed and dealt with in the same manner.

The statistical models were 'generalised additive models' or GAMs which extend additive models, comprising multiple non-parametric and parametric terms, to situations where the outcome variable is non-normally distributed, as in count data.[8]

Each final core model was re-checked by inspecting plots of the model residuals for any potential outliers or influential points and if these could be attributed to specific calendar events further dummy variables were defined as appropriate. 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.

The pollutants were fitted as linear terms. Due to 18% of $PM_{2.5}$ observations being missing, separate core models were constructed on a continuous series truncated at a length of 700 days (covering the period 1/11/94 to 30/9/96). These models were used to assess the $PM_{2.5}$ associations.

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 death was estimated. The potential for a non-linear (threshold) relationship between pollutant and risk of death was explored by fitting the pollutant as a loess in the core model and assessing the approximate F-test for the non-parametric part of the model.

Estimates of the population impact were made for all cause mortality based upon the all year, all age results for PM_{10} and $PM_{2.5}$. The annual number of premature deaths in a population of 1 million attributable to a given change in pollution levels were calculated.

All of the analyses were carried out using S-Plus 4.5 Professional (reference: Data Analysis Products Division, Mathsoft Inc., Seattle, Washington, April 1998).

RESULTS

Descriptive statistics for the outcome series, pollutants and meteorological variables are shown in Table 1 for the period October 1994 to December 1996. A daily mean of 61.4 deaths from all-causes was observed, 27.8 due to cardiovascular causes and 9.5 due to respiratory causes. Figures 1-5 illustrate the daily time-series for the outcomes, pollutant measures and meteorological variables respectively.

Table 2 presents unadjusted correlation coefficients between $PM_{2.5}$ and each outcome, pollutant and meteorological variable for all-year and the cool and warm seasons separately. The correlations cover the period November 1994 to September 1996, the period for which daily $PM_{2.5}$ measurements were available. During this period $PM_{2.5}$ measures were weakly correlated with increases in deaths for each outcome series on the same day. There were clear seasonal differences. During the warm season (April to September) the correlations between $PM_{2.5}$ and respiratory and all-cause mortality are stronger than those during the cool season (October to March). For cardiovascular mortality this seasonal pattern is much less evident. All other measured particulates are strongly correlated with $PM_{2.5}$ with the exception of the calculated coarse fraction ($PM_{10-PM_{2.5}}$). Moderate correlations are observed between $PM_{2.5}$ and the modelled primary and secondary fractions and the gases. Only weak associations are observed between $PM_{2.5}$ and the meteorological variables.

For this summary we present results for the cumulative lag of day 0 and day 1, since these are the lags associated with effects in the majority of other studies. A full description of the results for all single day and cumulative lags is presented in the Annex.

Table 3 gives the percentage change in the number of daily deaths in each outcome series associated with an increase in each pollutant from the 10th to the 90th percentile of its range together with a 95% confidence interval and P-value.

These are reported for all-year, cool and warm seasons separately. A P-value is also reported for the test for effect modification by season for each pollutant. It indicates whether or not the cool and warm season estimates are significantly different. Figure 6 illustrates these results for the measured particles for all-causes, cardiovascular and respiratory deaths respectively.

Table 4 summarises the significant results indicating the level of statistical significance and direction of associations between each outcome and each of the pollutants studied. Nothing was significant in the all-year analysis. It shows consistently positive associations during the warm season between all-cause mortality and particle measures PM₁₀, PM_{2.5}, BS, SO₄ and SFR and also the gases NO₂ and O₃.

PM_{2.5} AND OTHER DIRECTLY MEASURED PARTICLES

In the all-year analysis none of the measured particles were significantly associated with all-cause mortality. For PM_{2.5}, the association was small and positive, a 0.6% increase in mortality for a 18µg/m³ increase. This result was comparable to those for PM₁₀ and BS. For the calculated coarse fraction (PM_{2.5-10}) and SO₄ the associations with all-cause mortality were small, less than 1%, negative and insignificant. Results were similar for cardiovascular and respiratory mortality. Generally, the single-day measures and the other cumulative measures convey a similar message.

There were, however, significant seasonal differences in the associations between particle measures and all-cause mortality and, to a lesser extent, respiratory mortality. Particle measures were positively associated with all-cause mortality and respiratory mortality during the warm season and negatively (though not significantly) during the cool season. During the warm season PM_{2.5} was significantly associated with deaths from all-causes; 4.8% (95% confidence interval (CI): 1.0-8.7). Other measured particles showed comparable results. BS showed the strongest effect with both all-cause and respiratory mortality, 6.0% (95% CI: 2.1-10.0) and 9.4% (95% CI: -0.04-19.7) respectively.

Generally, the associations between particle measures and respiratory mortality in the warm season were large in magnitude but poorly estimated (wide confidence intervals) whereas for all-cause mortality the associations were slightly smaller but estimated with more precision. This is clearly illustrated in Figure 6. Also, for particles, associations with both all-cause and respiratory mortality were strongest on the same day, diminishing at lags 1 and 2 days and increasing again at lag 3 days. The cumulative measures from mean (0,1) to mean (0 to 5) were consistent in terms of magnitude and the associations tended to be stronger for the longer averaging periods.

No significant seasonal differences were found for cardiovascular admissions and any of the pollutants investigated. There was a suggestion that both NO₂ and CO in the cool season and O₃ in the warm season were associated with “same day” cardiovascular mortality, but these relationships were of low statistical significance (P<0.05).

MODELLED PARTICLES

We found that estimates of the primary fraction (PFR) and secondary fraction (SFR) were both positively associated with all-cause and respiratory mortality during the warm season. These associations were marginally statistically significant; SFR and all-cause mortality 4.6% (95% CI: 1.1-8.2) and PFR and respiratory mortality 8.3% (95% CI: 0.8-16.3). There was a tendency for the magnitude and statistical significance of these associations to increase when additional longer lags were included in the cumulative measures. All-year and cool season associations between PFR and SFR and all-cause and respiratory mortality were non-significant as were all associations between these two modelled pollutants and cardiovascular mortality.

GASES

Amongst the gases investigated only the association between O₃ and all-cause mortality approached statistical significance at the 5% level; 2.9% (95% CI: -0.1-6.0). For cardiovascular mortality there was weak evidence of associations with NO₂ and CO, and none for respiratory mortality. The seasonal results generally

showed stronger effects in the warm season but again, only O₃ approached statistical significance with all-cause mortality; 4.8% (95% CI: 0.9-8.8).

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_{2.5} measurements were available. This enabled a comparison between the estimated exposure-response of PM_{2.5} and mortality with that of PM₁₀. Figure 7 presents the exposure response curves for PM_{2.5}, PM₁₀ and all-cause mortality. The middle trail of asterisks is the estimated exposure-response, sandwiched by point-wise one-standard error confidence bands. The “rug spread” (small vertical marks along the pollutant axis) indicate the density of available data across the range observed and the vertical axis shows the relative risk. The plot should not be interpreted on a point-by-point basis but instead for its overall shape. Up to the median of measured PM_{2.5} there is a decrease in relative risk, followed by a localised increase. Beyond the 90th percentile there is a steady increase in relative risk with evidence of a flattening-off in the highest percentiles, though this observation is based on few data. The P-value for a non-linear effect was 0.005 which indicated that the dose-response relationship is significantly different from a linear one. In contrast, the dose-response with PM₁₀ shows a marked decline in relative risk up to the median of that pollutant and then a steady increase, without any hint of levelling-off beyond 45 µg/m³. This relationship was also non-linear (P=0.002).

DISCUSSION

There are a number of methodological issues which could have influenced the results of this analysis. The first to consider is the use of GAM methods as compared to other forms of statistical modelling. The GAM approach is now commonly used both in America and in Europe, where it has been adopted for the second phase of the APHEA collaboration. For one outcome series, all respiratory mortality in the 65+ group, the results from the GAM model were compared to those obtained using the parametric approach used by the APHEA project. The results of the two approaches were comparable (see Annex for further detail) which suggests that the results are not sensitive to the statistical technique employed.

As a further check, the raw data were examined in further detail. Correlation coefficients, for all year and by season, between each outcome and each pollutant were calculated. These data, which are presented in full in the Annex, show that in the cool season, particle measures were largely uncorrelated with all-cause, cardiovascular and respiratory mortality. However, in the warm season there were moderate positive correlations, typically around 0.2, between all particle measures and all cause and all respiratory mortality. This supported the pattern of results, particularly the seasonal differences, found in the more complex statistical analyses. In addition, the correlation analysis was repeated for all cause mortality only in Birmingham Health Authority using particle measures from the central Birmingham monitor. The same pattern as for the whole conurbation was found.

The results for PM₁₀ and Black Smoke observed in this study are compared with those of the earlier Birmingham study (1992-1994)[9] and of London 1992-94[10] (Table 5). It shows the percentage change in mortality associated with a 10 µg/m³ increase in PM₁₀ and BS; lag 1 day measures have been used for consistency across studies. The statistical approach was different in all three studies. All showed a positive association with PM₁₀ and all-cause mortality but the estimate from the present study was very small compared with the others.

However, each estimate fell within the 95% confidence intervals of the others, and cannot be distinguished statistically. The effect of BS was similar in Birmingham (this study) and London. The comparison for respiratory mortality showed some differences but, again, these were not statistically significant. The results for cardiovascular mortality were very similar between Birmingham and London for both PM_{10} and BS. It is concluded that the present findings are not demonstrably different from the earlier Birmingham study or London.

Given the high correlation between $PM_{2.5}$ and the other pollutants, the strong seasonal pattern found in the results and the relatively small number of days in each season within the study period, it was not appropriate to investigate the relative importance of each pollutant using two-pollutant models. 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. It may be appropriate to re-analyse some time series incorporating an additional years' data (1997), specifically to investigate two-pollutant models and the effect of this "collinearity" on the results.

The interpretation of time series studies in terms of population impact is fraught with difficulties.[11] How are effect estimates from different pollutants to be combined, which estimate, single lag or cumulative lags, should be used and how can the mortality displacement be estimated? However, bearing these limitations in mind, it is possible to translate these relative risk estimates into an estimate of the number of premature deaths avoided for a given reduction in air pollution. In a population of 1 million, assuming reductions in PM_{10} and $PM_{2.5}$ to 50% of their mean values, the annual number of deaths avoided attributed to PM_{10} was 8 (95% confidence interval: -100, 84) and to $PM_{2.5}$, 24 (95% CI: -108, 60). This assumed an average daily death rate in the population of 26.7 per day.

CONCLUSIONS

1. $PM_{2.5}$ is associated with increases in daily mortality which are largest and most statistically significant in the warm season.
2. The effects of $PM_{2.5}$ are generally similar to those of PM_{10} .
3. $PM_{2.5}$ and PM_{10} are closely correlated. Two pollutant models were not helpful in establishing which of the two pollutants dominates the association.
4. It was not clear if $PM_{2.5}$ showed stronger effects than the coarse fraction ($PM_{2.5-10}$).
5. Other measures of fine particles showed effects on mortality. Black Smoke tended to have larger effects than $PM_{2.5}$. The effects of sulphate were similar to those of $PM_{2.5}$.
6. The modelled estimates of the primary and secondary particles showed effects similar to those of the directly measured particles. Unsurprisingly there was some similarity between the results for primary particles and Black Smoke, and secondary particles and sulphate.
7. We were not able to disentangle the individual effects of intercorrelated pollutants.
8. Exposure response curves for PM_{10} showed an unclear pattern up to about $35\mu\text{g}/\text{m}^3$ but then increased linearly. For $PM_{2.5}$, the relationship was rather flat until $30\mu\text{g}/\text{m}^3$. These thresholds were well into the 90th percentile of the pollution values.

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Table 1. Descriptive Statistics for outcomes and environmental variables

		N	mean	Std.	min.	10 th	median	90 th	max.
<i>Mortality (Daily count)</i>									
All-cause, all ages	All Year	823	61.4	11.4	30	48	60	77	105
	Cool	457	65.5	11.4	38	52	64	82	105
	Warm	366	56.3	9.1	30	44	56	68	86
Cardiovascular, all ages	All Year	823	27.8	6.6	9	20	27	36	52
	Cool	457	29.8	6.6	13	21	30	38	52
	Warm	366	25.4	5.8	9	19	25	33	45
Respiratory, all ages	All Year	823	9.5	4.5	0	5	9	15	36
	Cool	457	11.1	4.9	1	6	10	17	36
	Warm	366	7.6	3.0	0	4	7	11	18
<i>Pollutants</i>									
<i>Measured Particles</i>									
PM ₁₀ (µg/m ³) 24hr	All Year	823	23.3	12.9	4	11.4	20	38.3	102.3
	Cool	457	23.6	14.6	4.0	10.6	19.2	41.6	102.3
	Warm	366	27.9	10.4	5.4	12.4	20.9	36.4	71.7
PM _{2.5} (µg/m ³) 24hr	All Year	675	14.5	9.7	2.1	6	11.7	25.8	82.8
	Cool	324	16.0	11.8	2.1	5.9	12.7	29.2	82.8
	Warm	351	13.1	7.0	3.0	6.1	11.0	21.8	47.2
PM _{2.5-10} (µg/m ³) 24hr	All Year	674	8.6	5.4	-2.2	3.0	7.7	15.2	57.2
	Cool	323	7.0	4.4	-2.2	2.2	6.5	12.7	27.0
	Warm	351	10.1	5.8	1.4	3.9	9.2	17.2	57.2
BS (µg/m ³) 24hr	All Year	823	13.2	9	1.8	5.1	10.9	23.6	71.9
	Cool	457	15.6	10.7	1.8	5.1	12.6	29.3	72.2
	Warm	366	10.2	4.7	1.9	4.9	9.9	16.0	31.5
SO ₄ (µg/m ³) 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	259	3.5	2.4	0.7	1.4	2.7	7.0	13.2
<i>Modelled Particles</i>									
Primary (µg/m ³) 24hr	All Year	804	7.0	6.5	0.6	2.1	5.3	13.8	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	8.5	25.9
Secondary (µg/m ³) 24hr	All Year	814	8.9	7.4	1.2	3.2	6.5	18.6	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	3.3	6.5	16.8	31.9
<i>Gases</i>									
NO ₂ (ppb) 1 hr	All Year	823	37.2	15.1	10.7	22.9	34.9	51.7	176.1
	Cool	457	24.5	9.4	5.7	14.1	23.8	34.1	80.1
	Warm	366	21.1	7.6	6.6	11.1	20.5	30.7	59.9
O ₃ (ppb) 8 hr	All Year	823	24.0	13.8	0.4	6.7	24	37.5	89.9
	Cool	457	24.5	9.4	5.9	14.1	23.8	34.1	80.1
	Warm	366	21.1	7.6	6.6	11.1	20.5	30.7	59.9
SO ₂ (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	12.7	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
	Cool	457	7.4	5.3	2.0	3.3	5.9	12.7	59.8
	Warm	366	6.9	3.9	1.9	3.3	5.7	11.9	25.2
<i>Meteorological (Daily)</i>									
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	13.2	18.1	22.6
Humidity (%)	All Year	823	78.7	10.0	51	65.5	79.5	91.5	99
	Cool	457	83.1	8.2	53.5	72.0	83.5	93.6	99

Warm	366	73.1	9.2	51.0	62.5	72.0	85.5	97.5
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Table 2. Correlations between PM_{2.5} and the outcome series, other pollutants and meteorological variables: all-year and by season

Variable	All-Year	Cool Season	Warm Season
All Causes	0.10	-0.05	0.18
All Cardiovascular	0.08	0.00	0.05
All Respiratory	0.07	-0.06	0.18
PM ₁₀	0.94	0.97	0.92
SO ₄	0.63	0.60	0.68
Black Smoke	0.71	0.76	0.53
PM _{2.5-10}	0.34	0.40	0.48
PFR	0.65	0.66	0.58
SFR	0.63	0.60	0.68
NO ₂	0.61	0.55	0.74
O ₃	-0.11	-0.47	0.40
SO ₂	0.52	0.49	0.56
CO	0.55	0.56	0.57
Temperature	-0.15	-0.11	-0.02
Relative Humidity	0.08	0.06	-0.09

Table 3. Summary of results for mean of current and previous days pollution levels

Cause of Death	Pollutant	All Year		Cool Season		Warm Season		Season P-value	
		% (95% CI)	P	% (95% CI)	P-value	% (95% CI)	P-value		
All-cause	PM ₁₀	0.2 (-1.8, 2.2)	0.9	-1.7 (-4.3, 1.0)	0.2	4.4 (0.9, 8.1)	0.02	0.007	
	PM _{2.5}	0.6 (-1.5, 2.7)	0.6	-1.3 (-4.4, 1.9)	0.4	4.8 (1.0, 8.7)	0.01	0.01	
	PM _{2.5-10}	-0.7 (-4.2, 2.9)	0.7	-4.6 (-9.0, 0.0)	0.05	3.3 (-0.5, 7.2)	0.09	0.01	
	BS	0.6 (-1.5, 2.7)	0.6	-0.3 (-2.9, 2.3)	0.8	6.0 (2.1, 10.0)	0.003	0.003	
	SO ₄	-0.4 (-2.3, 1.5)	0.7	-2.5 (-4.8, -0.1)	0.04	4.7 (1.1, 8.4)	0.009	0.001	
	PFR	0.9 (-0.9, 2.7)	0.3	0.6 (-1.8, 3.1)	0.6	2.8 (-0.3, 6.0)	0.07	0.1	
	SFR	-0.4 (-2.3, 1.5)	0.7	-2.5 (-4.8, -0.1)	0.04	4.6 (1.1, 8.3)	0.01	0.001	
	NO ₂	1.4 (-0.4, 3.2)	0.1	0.6 (-1.3, 2.6)	0.5	3.5 (0.1, 7.0)	0.04	0.2	
	O ₃	2.9 (-0.1, 6.0)	0.05	0.2 (-3.5, 4.0)	0.9	4.8 (0.9, 8.8)	0.01	0.1	
	SO ₂	-0.2 (-2.1, 1.8)	0.8	-0.1 (-2.5, 2.4)	0.9	-0.1 (-3.4, 3.2)	0.9	0.97	
	CO	0.8 (-0.6, 2.2)	0.3	0.8 (-0.9, 2.5)	0.4	1.8 (-1.0, 4.7)	0.2	0.3	
	Cardiovascular	PM ₁₀	1.0 (-1.9, 4.0)	0.5	0.5 (-3.3, 4.5)	0.8	2.4 (-2.7, 7.8)	0.4	0.5
		PM _{2.5}	0.9 (-2.1, 4.0)	0.6	0.8 (-3.6, 5.5)	0.7	1.7 (-3.5, 7.1)	0.5	0.7
		PM _{2.5-10}	-0.8 (-4.8, 3.5)	0.7	-3.1 (-9.5, 3.8)	0.4	2.3 (-3.1, 8.0)	0.4	0.2
BS		1.5 (-1.5, 4.7)	0.3	1.5 (-2.3, 5.5)	0.4	2.7 (-2.9, 8.6)	0.4	0.5	
SO ₄		-1.3 (-4.0, 1.5)	0.4	-2.5 (-5.8, 0.9)	0.1	1.8 (-3.1, 7.0)	0.5	0.2	
PFR		1.7 (-1.0, 4.4)	0.2	2.1 (-1.4, 5.8)	0.2	0.9 (-3.6, 5.5)	0.7	0.97	
SFR		-1.3 (-4.0, 1.5)	0.4	-2.6 (-5.9, 0.9)	0.1	1.8 (-3.1, 6.9)	0.5	0.2	
NO ₂		2.6 (-0.1, 5.4)	0.06	2.7 (-0.2, 5.7)	0.06	1.6 (-3.5, 7.0)	0.5	0.6	
O ₃		0.9 (-3.4, 5.4)	0.7	-2.5 (-7.5, 2.8)	0.4	3.4 (-2.1, 9.2)	0.2	0.1	
SO ₂		-0.2 (-3.0, 2.6)	0.9	0.8 (-2.7, 4.5)	0.7	-2.6 (-7.3, 2.3)	0.3	0.2	
CO		2.5 (0.4, 4.6)	0.02	3.0 (0.5, 5.5)	0.02	0.7 (-3.5, 5.0)	0.7	0.8	
Respiratory		PM ₁₀	-1.4 (-6.0, 3.5)	0.6	-3.3 (-8.9, 2.7)	0.3	3.1 (-5.0, 11.9)	0.5	0.2
		PM _{2.5}	-0.1 (-5.4, 5.5)	0.98	-3.1 (-9.9, 4.2)	0.4	6.5 (-2.7, 16.5)	0.2	0.1
		PM _{2.5-10}	-7.6 (-13.9, -0.9)	0.03	-12.8 (-20.9, -3.9)	0.006	-1.3 (-9.2, 7.3)	0.8	0.06
	BS	0.1 (-4.8, 5.2)	0.97	-1.3 (-6.9, 4.5)	0.6	9.4 (-0.04, 19.7)	0.05	0.04	
	SO ₄	-1.8 (-6.4, 3.0)	0.5	-4.0 (-9.2, 1.6)	0.2	2.7 (-5.5, 11.5)	0.5	0.2	
	PFR	1.0 (-3.3, 5.4)	0.7	-0.7 (-5.8, 4.8)	0.8	8.3 (0.8, 16.3)	0.03	0.03	
	SFR	-1.8 (-6.4, 3.0)	0.5	-4.0 (-9.3, 1.6)	0.2	2.7 (-5.4, 11.4)	0.5	0.2	
	NO ₂	2.8 (-1.6, 7.5)	0.2	1.3 (-3.0, 5.7)	0.6	6.9 (-1.8, 16.3)	0.1	0.3	
	O ₃	2.2 (-5.4, 10.4)	0.6	-3.3 (-11.0, 5.2)	0.4	7.1 (-2.5, 17.6)	0.2	0.1	
	SO ₂	-1.9 (-6.3, 2.7)	0.4	-2.9 (-7.9, 2.5)	0.3	1.9 (-5.8, 10.2)	0.6	0.4	
	CO	1.2 (-2.1, 4.6)	0.5	0.9 (-2.8, 4.8)	0.6	4.3 (-2.7, 11.7)	0.2	0.3	

Key:

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

PM₁₀, µg/m³, 24 hr, 24.4, 28.2, 21.6
 PM_{2.5}, µg/m³, 24 hr, 17.7, 22.7, 15.3

NO₂, ppb, 1 hr, 25.5, 22.3, 28.4
 O₃, ppb, 8 hr, 28.6, 24.1, 30.3

PM_{2.5-10}, µg/m³, 24 hr, 11.3, 9.5, 12.1

BS, 24 hr, µg/m³, 16.7, 19.7, 10.0

SO₄, 24 hr, µg/m³, 5.8, 6.3, 5.1

PFR, 24 hr, µg/m³, 10.6, 13.1, 5.9

SFR, 24 hr, µg/m³, 13.9, 15.2, 12.2

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

PFR - Primary particle fraction, SFR - Secondary particle fraction, BS - Black Smoke

SO₂, ppb, 24 hr, 8.5, 9.3, 7.

CO, ppm, 8 hr, 1.0, 1.1, 0.4

Table 4. Summary of significant results

Outcome	Cumulative Measure mean(0,1)										
	PM ₁₀	PM _{2.5}	PM _{2.5-10}	PFR	SFR	BS	SO ₄	NO ₂	O ₃	SO ₂	CO
<i>All-Cause</i>											
All-year											
Cool Season					(*)		(*)				
Warm Season	*	*			*	**	**	*	*		
<i>Cardiovascular</i>											
All-year											*
Cool Season											*
Warm Season											
<i>Respiratory</i>											
All-year			(*)								
Cool Season			(**)								
Warm Season				(*)							

Key:

(* P<0.05, **P<0.01, *** P<0.001)

N.B. cumulative lag mean(0,1) presented. Significant negative associations are enclosed in ().

Table 5. Comparison of results with other UK studies of particles

Results are from all-year analyses. The table shows the percentage change in mortality (95% confidence interval) from all causes, respiratory and cardiovascular diseases associated with an increase of 10 μgm^{-3} in the previous days measures (for consistency across studies) of PM₁₀ and BS.

Outcome	Pollutant	Present Study	Birmingham[1]	London[2]
All Cause	PM ₁₀	0.01 (-0.7, 0.7)	1.1 (0.1, 2.1)	0.3 (-0.2, 0.7)
	BS	0.2 (-0.9, 1.3)	n/a	0.7 (-0.0, 1.5)
Cardiovascular[3]	PM ₁₀	0.7 (-0.4, 1.8)	1.7 (-1.3, 4.8)	0.6 (-0.1, 1.2)
	BS	0.6 (-1.1, 2.2)	n/a	1.2 (0.1, 2.2)
Respiratory	PM ₁₀	-1.04 (-2.8, 0.7)	1.7 (-1.3, 4.8)	1.0 (-0.1, 2.1)
	BS	0.39 (-2.3, 3.1)	n/a	1.2 (-0.6, 3.0)

Key:

1 - Wordley *et al.* 1997

2 - Bremner *et al.* 1999

3 - For Wordley *et al.* result is for acute ischaemic heart disease and acute cerebrovascular disease (ICD9: 410-436).

n/a - result not available

Figure 1. Daily counts of deaths from all causes, cardiovascular disease and respiratory disease.

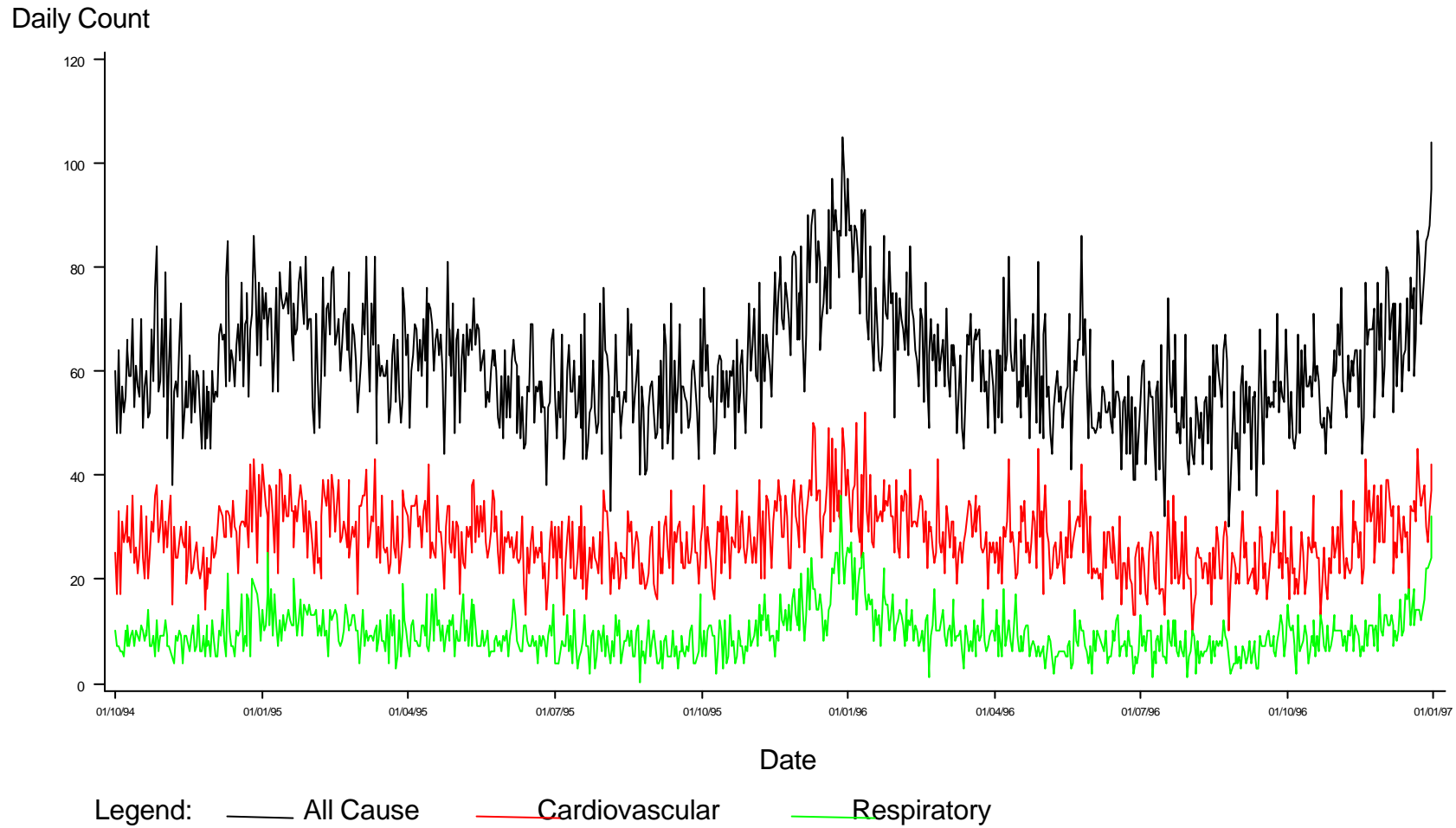


Figure 2. Daily levels of directly measured particles

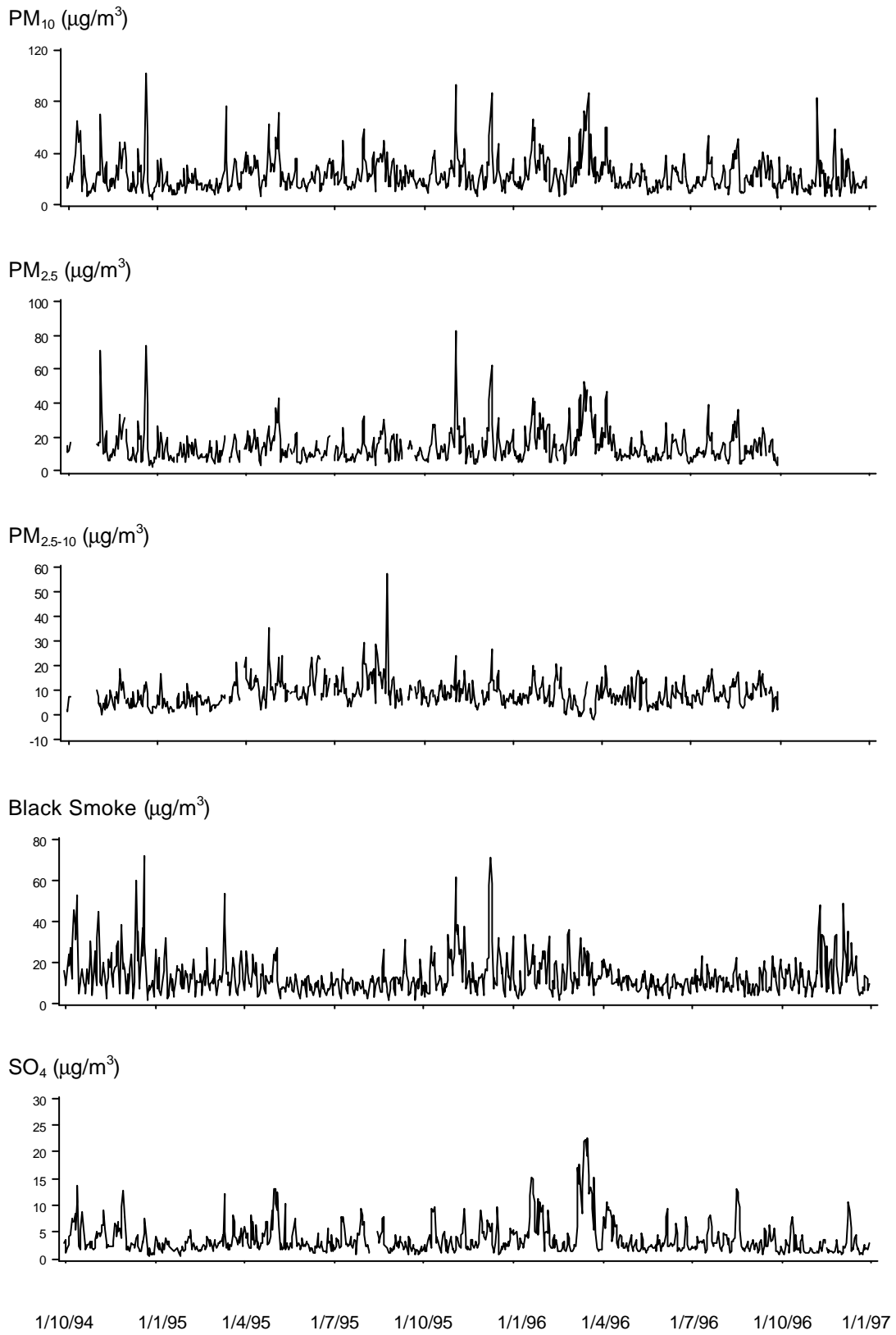
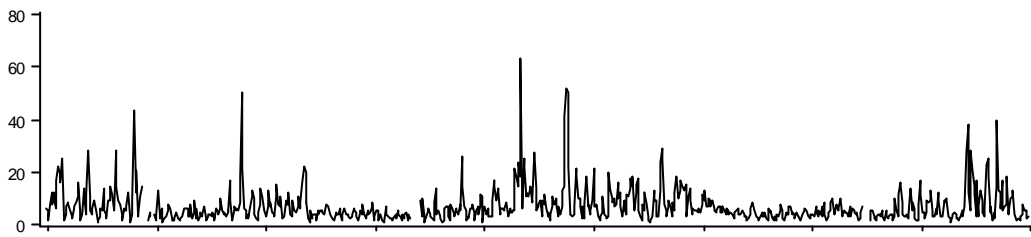
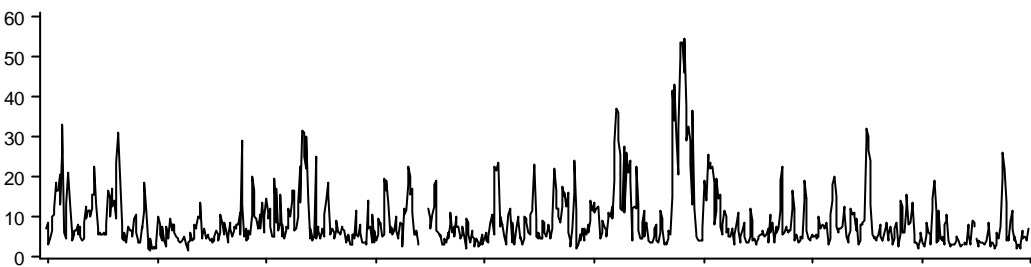


Figure 3. Daily levels of modelled particles

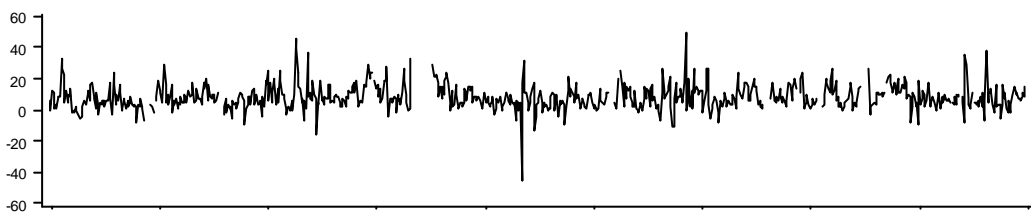
Primary Particle Fraction ($\mu\text{g}/\text{m}^3$)



Secondary Particle Fraction ($\mu\text{g}/\text{m}^3$)



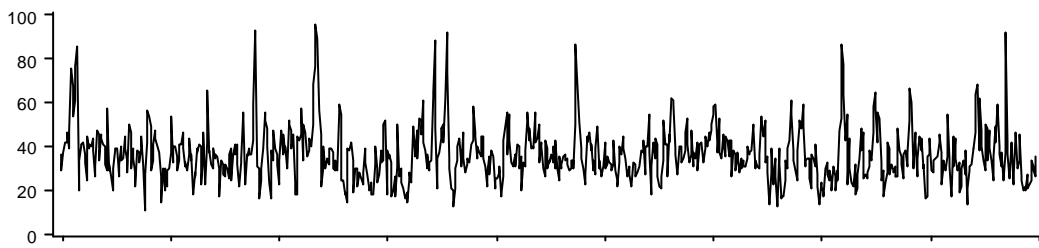
Coarse Particle Fraction ($\mu\text{g}/\text{m}^3$)



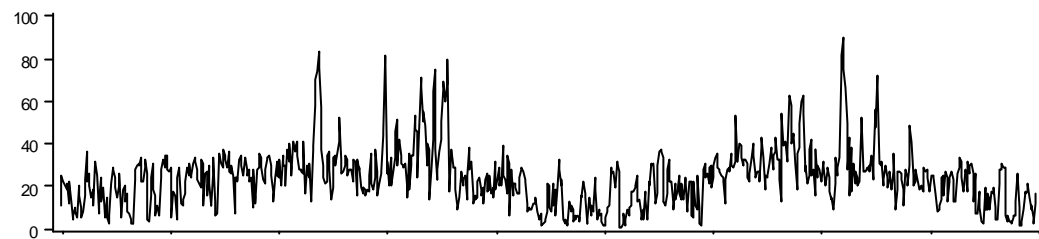
1/10/94 1/1/95 1/4/95 1/7/95 1/10/95 1/1/96 1/4/96 1/7/96 1/10/96 1/1/97

Figure 4. Daily levels of gases

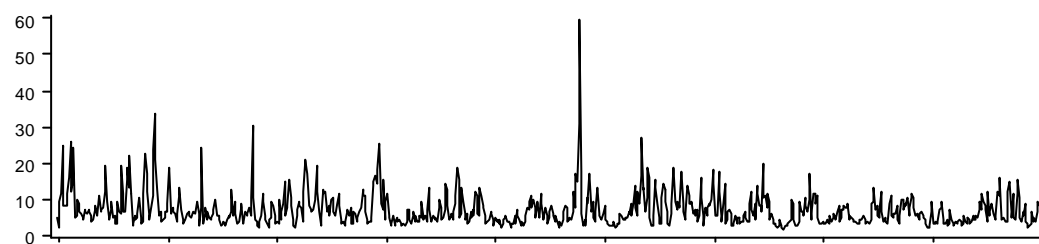
NO₂ (ppb)



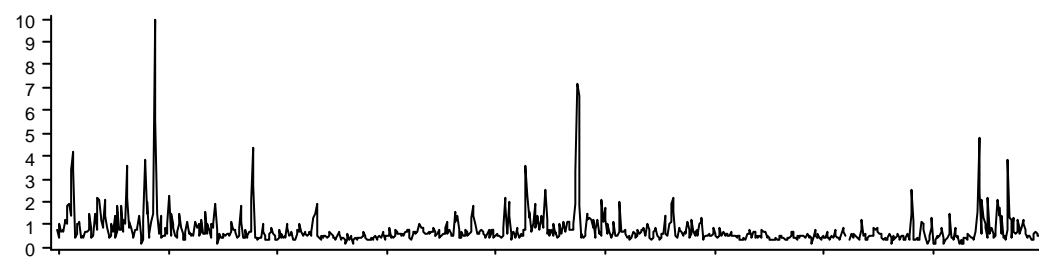
O₃ (ppb)



SO₂ (ppb)



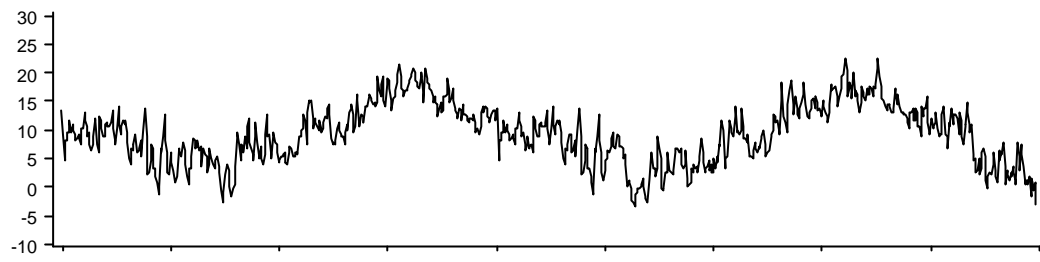
CO (ppm)



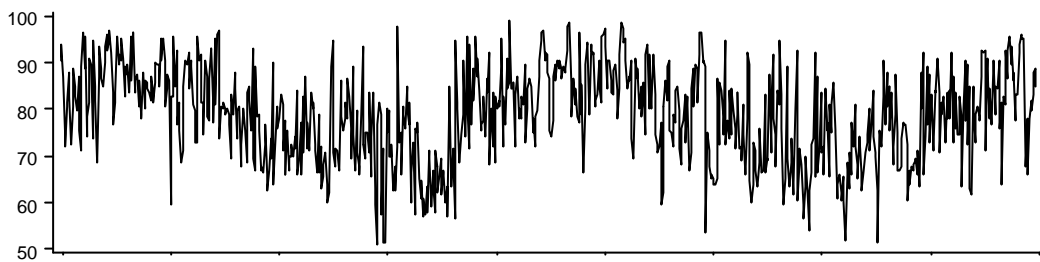
1/10/94 1/1/95 1/4/95 1/7/95 1/10/95 1/1/96 1/4/96 1/7/96 1/10/96 1/1/97

Figure 5. Daily temperature and relative humidity

Temperature (°C)



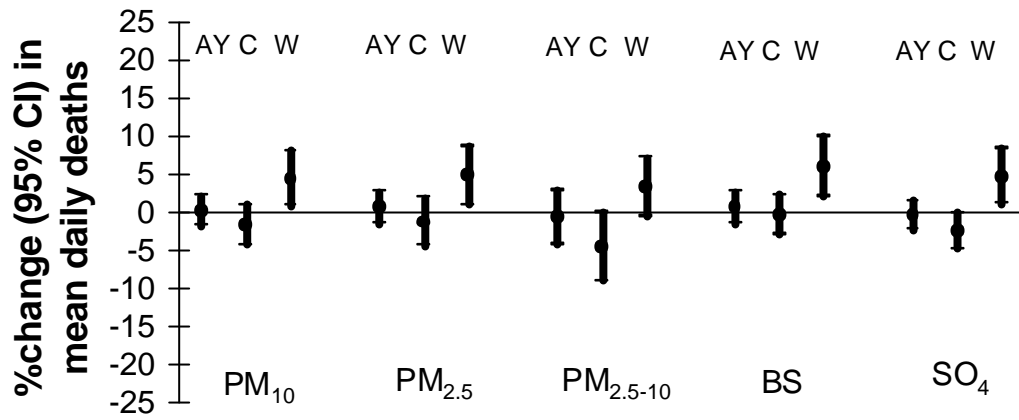
Relative Humidity (%)



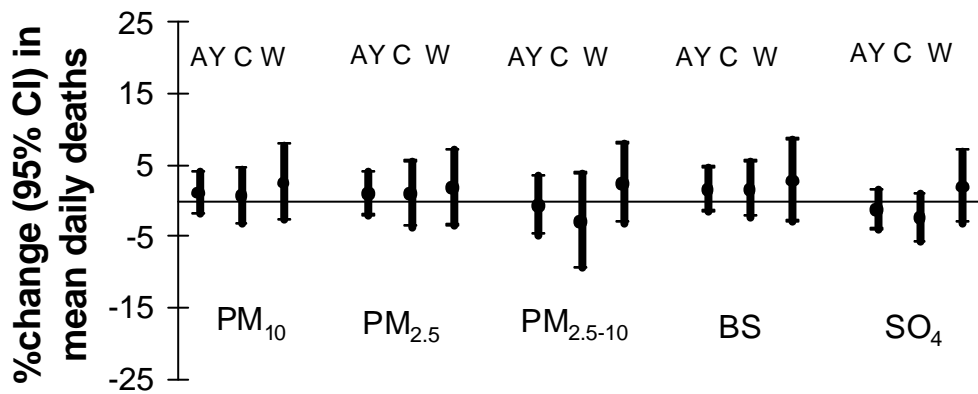
1/10/94 1/1/95 1/4/95 1/7/95 1/10/95 1/1/96 1/4/96 1/7/96 1/10/96 1/1/97

Figure 6. Comparison of all year and seasonal results for directly measured particles.

All Cause Mortality



All cardiovascular mortality



All respiratory mortality

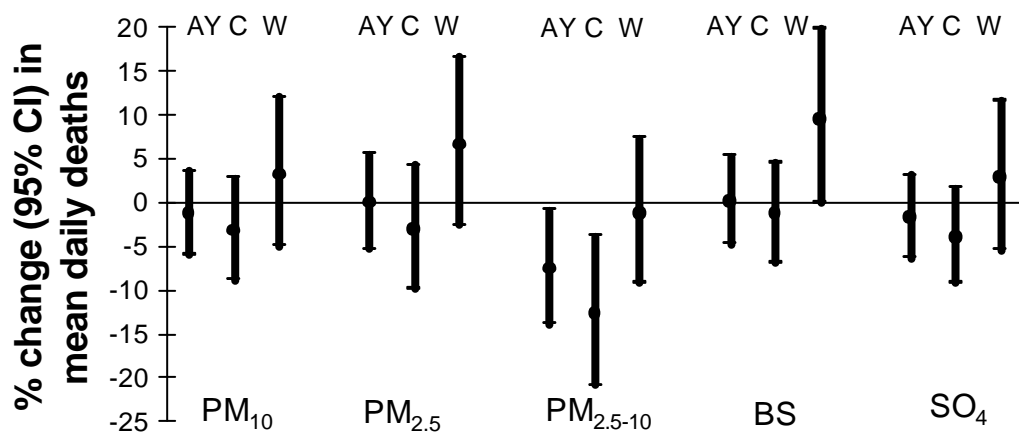
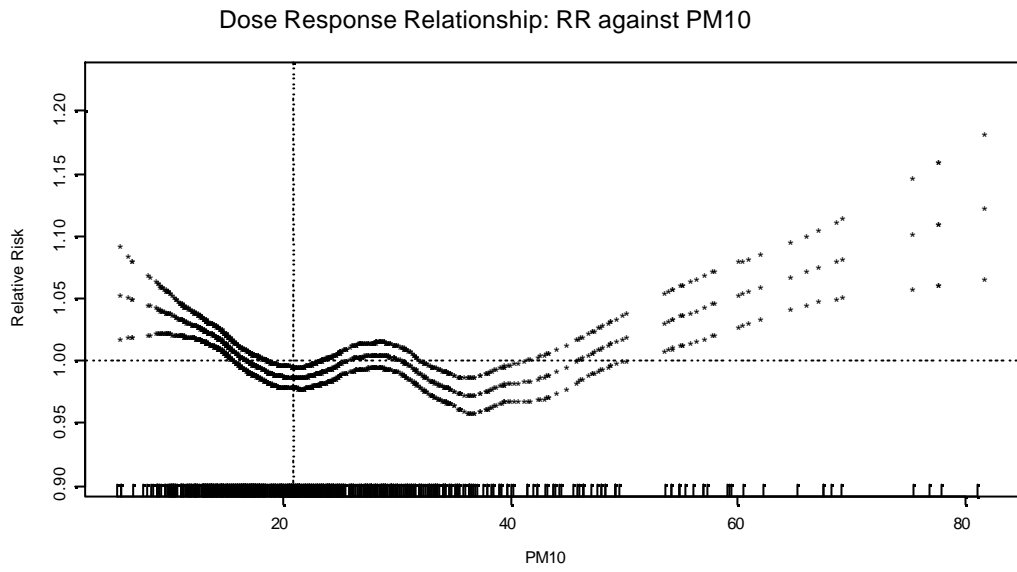


Figure 7. Exposure-response relationships for PM₁₀ and PM_{2.5}.



Dose Response Relationship: RR against PM2.5

