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Associations between short/medium-term variations in black smoke air pollution and mortality in the Glasgow conurbation, UK.

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Highlights

- A time series study of air pollution and mortality over a 25 year period.
- Compares estimates of short and medium term exposure-mortality associations.
- Effects of black smoke on mortality evident for lag periods in excess of two weeks.
- Medium term exposure-mortality associations greater than short-term associations.
- Implications for exposure averaging periods specified in air quality standards.

ABSTRACT

Objectives: To examine associations between short/medium-term variations in black smoke air pollution and mortality in the population of Glasgow and the adjacent towns of Renfrew and Paisley over a 25-year period at different time lags (0-30 days).

Methods: Generalised linear (Poisson) models were used to investigate the relationship between lagged black smoke concentrations and daily mortality, with allowance for confounding by cold temperature, between 1974 and 1998.

Results: When a range of lag periods were investigated significant associations were noted between temperature-adjusted black smoke exposure and all-cause mortality at lag periods of 13-18 and 19-24 days, and respiratory mortality at lag periods of 1-6, 7-12, and 13-18 days. Significant associations between cardiovascular mortality and temperature-adjusted black smoke were not observed. After adjusting for the effects of temperature a $10 \mu\text{g m}^{-3}$ increase in black smoke concentration on a given day was associated with a 0.9% [95% Confidence Interval (CI): 0.3-1.5%] increase in all cause mortality and a 3.1% [95% CI: 1.4-4.9%] increase in respiratory mortality over the ensuing 30-day period. In contrast for a $10 \mu\text{g m}^{-3}$ increase in black smoke concentration over 0-3 day lag period, the temperature adjusted exposure mortality associations were substantially lower (0.2 % [95% CI: -0.0-0.4%] and 0.3 % [95% CI: -0.2-0.8%] increases for all-cause and respiratory mortality respectively).

Conclusions: This study has provided evidence of association between black smoke exposure and mortality at longer lag periods than have been investigated in the majority of time series analyses.

Keywords:

time; series; black; smoke; lag; mortality.

INTRODUCTION:

There is extensive literature that, by consistency, provides evidence of a causal link between daily variations in air pollution and human mortality rates (e.g. see reviews by Pope & Dockery (2006) and Brunekreef & Holgate (2002)). Similarly, other studies have demonstrated that low air temperatures have a strong association with increased cardiorespiratory mortality (Braga et al. 2001; Pattenden et al. 2003). In earlier studies air temperature was considered a simple confounder in relation to short-term air-pollution effects (Lippmann and Ito 1995). Thus adjustments were made by including recent air temperature in the epidemiological models used, typically examining the effects of temperature-adjusted air pollution on the same day as the health outcome and/or variations that were measured up to a few days previously. However, several studies have suggested that effects on mortality of both cold temperature (Carder et al. 2005; Keatinge and Donaldson 2001) and air pollution (Carder et al. 2008; Dominici et al. 2003; Goodman et al. 2004; Schwartz 2000; Zeger et al. 1999) persist for considerably longer time periods.

We have found some evidence of interaction between (cold) temperature and particulate matter air pollution in their effect on all-cause and respiratory mortality (Carder et al. 2008). This is biologically plausible since, for example, it is known that cold will adversely affect respiratory muco-ciliary function (Williams et al. 1996) and may thus impede the clearance of pollutants. Our earlier analyses of the short-term effects of air pollution have been restricted to periods post-dating 1981 (Carder et al. 2008; Prescott et al. 1998). The aim of the study described here was to determine the effects of short/medium-term variations in black smoke air pollution and air temperature on mortality in the population of Greater Glasgow and the neighbouring towns of Renfrew and Paisley over a 25-year period, for direct comparison with the effects of long-term exposure to black smoke (Beverland et al. 2012b) in a cohort sub-group resident in the same geographical area (Beverland et al. 2012a; Yap et al. 2012). Thus, Poisson regression models were used to investigate the relationship between lagged black smoke concentrations (using a wide range of lag periods) and daily mortality, with allowance for confounding by cold temperature, over the period January 1974 to December 1998.

METHODS:

Health data: The study area encompassed a population of approximately 1.5 million residents in a densely populated and industrialised contiguous urban area of Glasgow, Paisley, and Renfrew in central Scotland, United Kingdom (the Glasgow conurbation). To provide an indication of geographical scale the Glasgow conurbation can be encompassed within a radius of 12 km, with Renfrew and Paisley encompassed by radii of 1.5 and 3.5 km, respectively, within this 12-km radius. The Information and Statistics Division of the Common Services Agency of the National Health Service in Scotland supplied mortality data for the period January 1974 to December 1998 for residents, age 50 and over, of the Glasgow conurbation.. Deaths from all non-accidental causes (referred to hereafter as all cause), cardiovascular causes (ICD-9 codes 410-414, 426-429, 434-440: essentially including cardiac and cerebral ischaemia but excluding cerebral haemorrhage), respiratory causes (ICD-9 codes 480-487 and 490-496: chronic obstructive pulmonary disease (COPD), asthma and pneumonia), lung cancer (ICD-9 code 162) and ‘other’ causes (all non-accidental causes minus codes specified above) were considered.

Lung cancer was included to match the outcomes examined in our related cohort study (Yap et al. 2012). While long-term exposure to air pollution might be causally linked to lung cancer after several years of exposure, the long latency of lung cancer implies that one might not expect deaths from lung cancer *per se* to be related to short-term changes in pollution on a scale of days or even months. However, our previous work discusses plausible explanations for the finding of a significant association between black smoke and mortality in groups containing subjects who did not have a specified cardiac or respiratory code recorded as the primary (underlying) cause of death (Carder et al. 2008). The “mode” or secondary cause of death in many diseases, regardless of the primary underlying pathology, is often respiratory and this is likely to be particularly true when the primary pathology is lung cancer (not only because of COPD and lung cancer being caused largely by smoking, but also because the presence of a bronchial tumour predisposes to distal pneumonia). It is therefore plausible that short-term exposure to air pollution contributes to this final “mode” of death.

Meteorological and air pollutant data: Hourly air temperature data for were obtained from the British Atmospheric Data Centre (BADC 2002) for Glasgow Airport. These were used to calculate the daytime mean temperature (T) for each day, taken as the average of the 7 am to 11 pm hourly values. Our previous work (Carder et al. 2005) had observed a non-linear relationship between temperature and mortality: mortality increased as temperature decreased but with a steeper increase at lower temperatures. Carder et al. (2005) examined the shape of the relationship between mortality and daytime mean temperature at each lag period by fitting cubic spline models with 7 degrees of freedom. To allow for this in a manner that would allow simple interpretations of model coefficients, two separate linear relations were assumed

over different parts of the temperature range constrained to join at a ‘knot’. The position of the knot was decided by comparing the log likelihoods of models with different choices of knot as suggested by the initial plots and choosing the one that fitted the data best—that is, at 11°C. The double linear model included two modified temperature variables referred to here as ‘high’ and ‘low’ defined as follows:

$$High = T - 11 \text{ if } T \geq 11 \text{ }^{\circ}\text{C}, 0 \text{ otherwise}$$

$$Low = T - 11 \text{ if } T < 11 \text{ }^{\circ}\text{C}, 0 \text{ otherwise}$$

We had also observed the effect of temperature on mortality to persist for periods beyond two weeks (Carder et al. 2005). Therefore, these variables were computed for T lagged by 0 (same day), 1-6 days (i.e. average of temperatures on the previous 1-6 days), 7-12 days, 13-18 days, 19-24 days and 25-30 days. Days were grouped in this way to minimise the number of variables in the regression models and thus reduce the problem of multi-collinearity (Carder et al. 2005; Carder et al. 2008).

The prevailing year-round wet climate in the West of Scotland is such that low relative humidity is seldom obvious as an environmental stressor. Low temperatures and wind-chill are more obvious stressors that may be plausibly linked to mortality in susceptible people in this geographical area. However, our earlier work found little indication that ‘wind chill’ temperature was a better predictor of mortality than ‘dry bulb’ temperature (Carder et al. 2005) so we focused our analyses with adjustments for temperature only.

Our analyses were based on daily records of black smoke concentration at a single monitoring station in central Glasgow which had few missing values and was situated in a residential area with medium to high-density housing interspersed with some industrial undertakings (classification A2/B2 according to the UK National Air Quality Archive, (DEFRA 2005)). From a review of approximately 10 potential monitoring sites in the conurbation, bearing in mind prevailing winds, population distribution and other factors, this was considered the most appropriate single site. Black smoke is a metric of the optical darkness of airborne particulate matter collected on filter media (Heal and Quincey 2012; Quincey et al. 2009). Although quantified in units of $\mu\text{g m}^{-3}$ black smoke concentrations do not equate directly to the mass of a particular size fraction of airborne particulate matter. However, consistent standard calibrations (e.g. DETR (1999)) have been used for many decades to convert reflectance to nominal concentration such that black smoke data are important measures of historic air pollution. The DETR (1999) calibration procedures were used in the computation of UK government archived black smoke data used in the present manuscript. The use of black smoke as a metric of particulate matter air pollution is well-established in the epidemiological research community and has been shown to be a good marker for traffic and other primary combustion-related urban air pollution (Hochadel et al. 2006; Hoek et al. 2001) often at least as predictive of negative health outcomes as PM_{10} or $\text{PM}_{2.5}$ (Janssen et al. 2011).

Statistical analysis: All analyses were undertaken using Splus software (Version 2000), using generalised linear (Poisson) models (GLMs) with natural cubic splines to capture seasonal and other long-term effects. The convergence tolerances of the GLM function were set to 10^{-9} with a limit of 1000 iterations (Pattenden et al. 2003). The GLMs included terms for day of week (indicator variables), and ‘season’ (a smoothed function of date with seven degrees of freedom per year – based on findings of the NMMAPS mortality reanalysis study (Dominici et al. 2000)) in addition to the 12 temperature variables described above. Models also included terms representing lagged black smoke and interactions between black smoke and temperature (Carder et al. 2008). The over-dispersion parameter (estimated from the GLM models) was close to 1 suggesting little additional variation beyond Poisson variation, and as such a simple Poisson model was assumed. For presentation of results, the percentage increase in mortality for standard exposure increments was derived from $(RR-1) \times 100\%$, where RR was the relative risk derived from $\exp(\beta)$, where β represents calculated GLM coefficients.

Lagged effects of black smoke on mortality were investigated using lag periods identical to the temperature lag periods (i.e. 0, 1-6, 7-12, 13-18, 19-24 and 25-30 days). Mean black smoke concentrations in each period were calculated and the resulting variables included in the model simultaneously. All pollution-related results were presented as the percentage increase in mortality associated with a $10 \mu\text{g m}^{-3}$ increase in black smoke concentration. To allow comparison with earlier analyses in other Scottish conurbations (including Carder et al. (2010; 2008) and Prescott et al. (1998)) black smoke effects were also investigated at lag 0-3 days, before and after adjustment for temperature at lag 0-3 days.

Interpretation of coefficients from models using temperature and black smoke lags averaged over six days: The effect that a given magnitude of black smoke concentration (or temperature) on day X may have on mortality on days X+1, X+2, ... etc would be expected to vary smoothly as the lag period increased (as assumed in distributed lag models). For simplicity of interpretation, this smooth relation is approximated here by a stepped relation changing every six days (with the exception of lag 0), with an implicit assumption that the true effect is reasonably constant within each 6-day averaging period.

Use of mean exposure variables within each exposure averaging period affects the interpretation of the corresponding coefficients in the model (see footnote to Table 2 and Carder et al. (2005)). For example, the coefficient for black smoke lagged 1–6 days can be interpreted as the increase in (log) deaths on day X comparing a scenario where each of the six previous days had a certain concentration of black smoke versus a scenario where the black smoke was $10 \mu\text{g m}^{-3}$ higher on each of the preceding six days. A

change on only one of these six days would reduce the effect to a sixth of this. To enable a direct comparison between the lag 0 coefficient and the other (six day average) coefficients, results are presented as relating to an increase of $10 \mu\text{g m}^{-3}$ on only one day within each lag period. The coefficient can also be interpreted as the total increase, across days X+1 to X+6 combined, associated with an increase of $10 \mu\text{g m}^{-3}$ on day X, compared with no increase on that day. The sum of the coefficients for the six lag periods represents the total increase across day X to X+30 combined, associated with an increase of $10 \mu\text{g m}^{-3}$ on day X compared with no increase (Carder et al. 2005).

RESULTS:

Descriptive statistics:

Table 1 shows the mean and range of daily deaths, temperature and black smoke concentration. In this study area and period the number of daily deaths in the population ranged from 12 to 79, with a mean of 34. There was evidence of a falling trend in the number of daily deaths (Fig 1). Daytime mean temperature had a substantial range from -16.6°C to 25.7°C (mean 9.5°C). Daily mean black smoke concentrations ranged from $1 \mu\text{g m}^{-3}$ to $930 \mu\text{g m}^{-3}$, with a strong seasonal cycle with greatest amplitude in the earliest years (Fig 1). Annual mean black smoke concentrations reduced substantially during the study period (Fig 1) as a result of the introduction of smoke control measures through the Clean Air Acts and vehicle emission regulations.

Association between exposure to black smoke and mortality:

When a range of lag periods were investigated generally the maximum black smoke effect occurred at lag 1-6 days prior to adjustment for temperature (Table 2). Significant associations remained between temperature-adjusted black smoke exposure and all-cause mortality at lag periods of 13-18 and 19-24 days, and respiratory mortality at lag periods of 1-6, 7-12, and 13-18 days. The lagged effects on respiratory mortality are notably higher compared to other outcomes, albeit with larger uncertainty due to the small daily counts. Significant associations between cardiovascular mortality and temperature-adjusted black smoke were not observed. The estimates in the first three lag windows (1-6, 7-12, 13-18) are reasonably consistent with Carder and others (2008) for all three causes of death, but there is less consistency for the last two lag windows and at lag 0.

For all-cause, and all of the specific causes of, mortality investigated there was evidence of a significant positive association between mortality and lagged (0-3 days) black smoke concentration prior to adjusting for the effects of temperature (data not shown). After adjusting for the effects of temperature only the

association between black smoke and all-cause mortality remained close to significant (0.17 % [95% CI: -0.01-0.35%] increase) (Table 3). Table 3 also shows the estimated effect of a $10 \mu\text{g m}^{-3}$ increase in the black smoke concentration on a given day on the percentage increase in mortality over the ensuing 30-day period (found by summing the black smoke coefficients for individual days). After adjustment for temperature effects a $10 \mu\text{g m}^{-3}$ increase in black smoke concentration on a given day was associated with a 0.9% [95% confidence interval (CI): 0.3-1.5%] increase in all cause mortality and a 3.1% [95% CI: 1.4-4.9%] increase in respiratory mortality over the ensuing 30-day period.

DISCUSSION

This study found associations between black smoke exposure and all-cause and cause-specific mortality in the population of Glasgow and the contiguous towns of Paisley and Renfrew. The largest association was observed between black smoke and respiratory mortality. Significant associations were observed between mortality and black smoke lagged by approximately one and four weeks. This observed lag structure suggests that, in this population, consideration of pollutant measurements for only a few days prior to the health outcome, as has been the norm in the majority of time-series studies, could lead to an underestimation of the full extent of the pollutant effect. The regression models also included lagged temperature measurements (one month) and as such, it is unlikely that the observed black smoke effects were due to residual confounding from temperature.

The results presented here are broadly consistent with a small number of other studies that have investigated the distributed lag structure between air pollution exposure and mortality. In a combined analysis of black smoke pollution effects in the populations of the three largest cities in Scotland (Glasgow, Edinburgh and Aberdeen) Carder et al. (2008) found maximum (although non-significant) single day associations between black smoke and mortality at lag 0, and also observed significant associations lagged by approximately two and four weeks (the latter being consistent with the findings reported here – Tables 2 and 3). In Dublin, Goodman et al. (2004) observed maximum single day associations between mortality and same-day particle exposure, with increased and significant associations over extended lag periods (subsequent from exposure) for all-cause and cause-specific mortality outcomes (Table 3). Zanobetti et al. (2002) used distributed lag models to investigate the association between mortality and PM_{10} (lagged up to 40 preceding days) across 10 European and adjacent cities within the APHEA-2 (Air Pollution and Health: A European Approach) project, and observed largest associations to be between same day PM_{10} and mortality, with effects generally decreasing then rising to another peak at approximately four weeks lag. The relative magnitudes of associations between specific mortality outcomes and lagged pollution exposure observed in the present

study and in similar studies reported in the literature are summarised in Table 3 and discussed in the following paragraphs.

When only acute effects (3-day mean for day of death and previous 2 days) were considered, Goodman et al. (2004) found that all-cause mortality increased by 0.4% for each $10 \mu\text{g m}^{-3}$ increase in black smoke concentration compared to a 1.1% increase in deaths in the 40 days after exposure. Zanobetti et al. (2002) observed a 0.70% increase in daily all-cause deaths per $10 \mu\text{g m}^{-3}$ increase PM_{10} for the day of and day before death; whereas a $10 \mu\text{g m}^{-3}$ increase PM_{10} in unrestricted distributed lag models over a 40-day preceding lag period was associated with a 1.61% increase in all-cause deaths. The black smoke all-cause mortality associations over the cumulative 30-day lag periods observed in the present study and by Carder et al. (2008) were consistent with the above cumulative 40-day lag associations observed by Goodman et al. and Zanobetti et al. In contrast the black smoke all-cause mortality association at shorter lag periods (0-3 days) in the present study was approximately 2-4 times lower than the short-lag period associations noted by Goodman et al. and Zanobetti et al. resulting in a greater contrast in exposure-mortality associations between short and medium time-scales of exposure (Table 3).

Goodman et al. (2004) observed similar exposure-cardiovascular mortality associations to those noted for all-cause mortality (0.4% increase in cardiovascular mortality for each $10 \mu\text{g m}^{-3}$ increase in 3-day running mean black smoke concentration compared to a 1.1% increase when deaths in the 40 days after exposure were considered). Similarly in further analyses of the 10 cities referred to above, Zanobetti et al. (2003) observed that the effect size estimate for the association between PM_{10} and cardiovascular deaths more than doubled over 40 day lags preceding death compared to baseline models using PM_{10} on the day of and day before death (1.97% and 0.69% respectively). In common with our earlier study over a different time period and geographical area in Scotland (Carder et al. 2008) the present study yielded a relatively small (and non-significant) association between black smoke and cardiovascular mortality over ensuing 30-day periods, albeit with the ratio of associations at short versus medium timescales in the present study being consistent with that observed by Goodman et al. and Zanobetti et al. (Table 3). This pattern of association between medium time-scale air pollution exposure and cardiovascular mortality in the Scottish population is partly inconsistent with the existing evidence base (Brook et al. 2010; Pope 2007; Pope and Dockery 2006), and merits further investigation.

For respiratory mortality, the association with black smoke observed by Goodman et al. (2004) was 0.9% for 3-day running mean exposures, but approximately four times higher (3.6%) for follow-up over 40 days. Zanobetti et al. (2003) noted associations five times higher for respiratory deaths at cumulative 40-day preceding (0-40) lag periods compared to 0-1 day lag periods (4.2% and 0.74% respectively). The

black smoke respiratory mortality associations over ensuing cumulative 30-day lag periods observed in the present study and by Carder et al. (2008) were generally consistent with the above cumulative 40-day lag associations observed by Goodman et al. and Zanobetti et al.; whereas (similar to the all-cause mortality results discussed above) the black smoke respiratory mortality association at shorter lag periods (0-3 days) in the present study was approximately 1.5-3 times lower than the associations noted by Goodman et al. and Zanobetti et al., again resulting in increased contrast in exposure-mortality associations between short and medium time-scales of exposure (Table 3).

The comparison between estimates of 30 day effects on all cause mortality observed by Carder et al. (2008) and Zanobetti et al. (2002; 2003) (Table 3B) might suggest that methodological difference in analytical approaches (multiple lag compared to distributed lag models) does not have a substantial effect on estimates, although the lack of concordance in the case of cardiovascular mortality clearly weakens any simplistic conclusion. We suspect that the relatively lower estimates in the present study are less due to methodological differences and more likely attributable to high pollution measurements in the early part of the study period (the specific effects of which are appropriate for further investigation; alongside analyses of possible effect modification by neighbourhood-scale socioeconomic conditions (Carder et al. 2010)).

The associations between black smoke exposure and mortality at 0-3 day lag (Table 3) are coherent with similarly small and generally non-significant associations between PM₁₀ exposure and cardiovascular hospital admissions noted in a study in the same geographical area between 2000 and 2006 (Willocks et al. 2012). In contrast, in a related study in the same geographical area we have observed larger magnitudes of short-term (3-day) black smoke exposure-mortality associations within the Renfrew-Paisley cohort (15,331 participants) in nested case-control analyses that accounted for spatial variations in pollution exposure and individual-level risk factors (Beverland et al. 2012a). It is possible that relatively small pollution-mortality associations at short lag times may become obscured by misclassification bias and/or inappropriate control for confounding factors (Sheppard et al. 2012) when estimated in time-series analyses using single, or small numbers of, monitoring sites. This emphasises the importance of ongoing research across a range of exposure metrics, time and spatial scales in urban areas in Scotland where there is marked spatial heterogeneity in pollution climates and socio-economic influences on health, especially in relation to the unresolved epidemiological challenge of identification of thresholds below which air pollutants have no observable effects on population health.

In conclusion, this study has provided evidence of a greater effect of black smoke on mortality at longer lag periods than have been studied in the majority of time series analyses. Our observations support the

conclusion that associations between BS and mortality persist for up to a month after exposure, but also underline the importance of appropriately adjusting for temperature effects over the same period so as to avoid over- or under-estimation of short term pollution effects. It is possible that the delayed effects are the consequence of infections or other inflammatory processes progressively developing after the initial acute exposure. It is also possible that some of the observed effects could be due to confounding, or may be modified, by unmeasured pollutants other than black smoke (Dominici et al. 2010). Collectively the evidence from this study and the other studies discussed above suggests that in these populations the full extent of short-term pollution exposure would be underestimated if the analysis was restricted only to a few days prior to health outcomes. Correspondingly public health impact assessment and related policies/practice for air pollution monitoring and management should take account of the effects of different temporal scales of exposures to air pollution.

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Disclaimer:

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Competing interest statement:

None declared

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Table 1 Mean, min and max daily number of deaths, temperature and black smoke concentration in the study area: 01/01/1974 – 31/12/1998. The mortality data is for residents, age 50 and over, of the Glasgow conurbation.

Cause of death	Total	Mean	SD	Min	Max
All cause mortality	310,312	34.0	7.9	12	79
Cardiovascular mortality	137,287	15.0	4.8	2	41
Respiratory mortality	38,340	4.2	2.6	0	28
Lung cancer	27,331	3.0	1.8	0	13
‘Other’ cause mortality	107,346	11.8	3.8	0	41
Daytime mean air temperature (°C)	-	9.5	5.2	-16.6	25.7
Daily mean black smoke ($\mu\text{g m}^{-3}$)	-	20.7	35.5	1	930

Table 2 Percentage increase in relative risk of mortality associated with a $10 \mu\text{g m}^{-3}$ increase in the daily mean black smoke concentration (lag 0, 1-6, 7-12, 13-18, 19-24, 25-30) in the Glasgow conurbation during period 1974-1998. **Values in bold are significant at $P < 0.05$.**

Lag period	All cause	Cardiovascular	Respiratory	Lung cancer	Other causes
Estimates prior to adjustment for temperature:					
0	0.02 (-0.10, 0.14)	0.08 (-0.10, 0.25)	-0.16 (-0.50, 0.17)	0.12 (-0.30, 0.54)	-0.02 (-0.22, 0.19)
1-6	0.11 (0.07, 0.14)	0.11 (0.06, 0.16)	0.20 (0.10, 0.30)	0.08 (-0.05, 0.20)	0.06 (0.00, 0.12)
7-12	0.07 (0.04, 0.11)	0.04 (-0.01, 0.09)	0.19 (0.10, 0.28)	0.09 (-0.03, 0.21)	0.05 (-0.01, 0.11)
13-18	0.06 (0.03, 0.10)	0.06 (0.01, 0.11)	0.15 (0.06, 0.24)	0.04 (-0.09, 0.16)	0.03 (-0.02, 0.09)
19-24	0.06 (0.02, 0.09)	0.05 (0.00, 0.10)	0.09 (0.00, 0.18)	0.08 (-0.04, 0.20)	0.04 (-0.02, 0.09)
25-30	-0.01 (-0.04, 0.02)	-0.02 (-0.07, 0.03)	0.05 (-0.04, 0.13)	-0.01 (-0.13, 0.12)	-0.02 (-0.08, 0.03)
Estimates following adjustment for temperature:					
0	0.01 (-0.11, 0.14)	0.02 (-0.17, 0.20)	-0.07 (-0.42, 0.29)	0.10 (-0.35, 0.55)	0.01 (-0.21, 0.23)
1-6	0.03 (-0.01, 0.07)	0.02 (-0.04, 0.08)	0.13 (0.02, 0.24)	0.02 (-0.12, 0.16)	0.00 (-0.06, 0.07)
7-12	0.03 (-0.01, 0.07)	0.00 (-0.06, 0.05)	0.12 (0.01, 0.22)	0.08 (-0.06, 0.22)	0.03 (-0.04, 0.09)
13-18	0.05 (0.01, 0.08)	0.02 (-0.03, 0.08)	0.12 (0.02, 0.22)	0.04 (-0.10, 0.18)	0.04 (-0.03, 0.10)
19-24	0.05 (0.02, 0.09)	0.05 (-0.01, 0.11)	0.07 (-0.03, 0.17)	0.11 (-0.02, 0.25)	0.03 (-0.04, 0.10)
25-30	-0.01 (-0.05, 0.02)	-0.03 (-0.08, 0.03)	0.04 (-0.06, 0.14)	-0.01 (-0.15, 0.13)	-0.03 (-0.09, 0.04)

The estimates in this table refer to the effect of a $10 \mu\text{g m}^{-3}$ increase in mean black smoke concentration on any *one* day within each lag period. The individual days within each lag period are constrained by the model to have the same effect. All lag periods are included in the model simultaneously. Thus according to the model for all cause mortality, without adjustment for temperature, a $10 \mu\text{g m}^{-3}$ increase at lag 0 would have a 0.02% effect on mortality; at each of lags 1 to 6 such an increase would have a 0.11% effect on mortality; at each of lags 7 to 12 such an increase would have a 0.07% effect on mortality; etc.

Table 3. Percentage increase in relative risk of mortality associated with a $10 \mu\text{g m}^{-3}$ increase in black smoke concentration at contrasting lag periods in present study (second column) and in similar studies reported in literature. **Values in bold are significant at $P < 0.05$.**

(A) Temperature adjusted black smoke exposure mortality associations (95% CI) at lag periods in range 0-3 days:				
	Present study:	Similar studies reported in literature:		
Cause of death	Percentage increase in mortality associated with a $10 \mu\text{g m}^{-3}$ increase in the daily mean black smoke concentration for lag 0-3 days in the Glasgow conurbation during 1974-1998	-	Percentage increase in mortality associated with a $10 \mu\text{g m}^{-3}$ increase in the 3-day running mean black smoke concentration in Dublin for 1980-1996. Data from Goodman et al. (2004)	Percentage increase in mortality associated with a $10 \mu\text{g m}^{-3}$ increase in the same and preceding day (lag 0-1) black smoke concentration in ten cities in Europe and adjacent countries for periods between 1990 and 1996. Data from Zanobetti et al. (2002, 2003).
All cause	0.17 (-0.01, 0.35)	-	0.4 (0.3, 0.6)	0.70 (0.43, 0.97)
Cardiovascular	0.10 (-0.16, 0.36)	-	0.4 (0.2, 0.7)	0.69 (0.31, 1.08)
Respiratory	0.27 (-0.22, 0.75)	-	0.9 (0.5, 1.2)	0.74 (-0.17, 1.66)
Lung cancer	0.38 (-0.25, 1.02)	-	-	-
Other causes	0.17 (-0.14, 0.48)	-	0.2 (0.0, 0.5)	-
(B) Temperature adjusted black smoke exposure mortality associations (95% CI) at lag periods of 0-30 and 0-40 days:				
	Present study:	Similar studies reported in literature:		
Cause of death	Percentage increase in mortality over ensuing 30 day period associated with a $10 \mu\text{g m}^{-3}$ increase in the daily mean black smoke concentration on a given day in the Glasgow conurbation during period 1974-1998.	Percentage increase in mortality over ensuing 30 day period associated with $10 \mu\text{g m}^{-3}$ increase in daily mean black smoke concentration on a given day for Glasgow, Edinburgh, and Aberdeen urban areas during 1981-2001. Data from Carder et al. (2008)	Percentage increase in mortality associated with a $10 \mu\text{g m}^{-3}$ increase in black smoke concentration over successive 40-day period in Dublin for 1980-1996. Data from Goodman et al. (2004)	Percentage increase in mortality associated with a $10 \mu\text{g m}^{-3}$ increase in PM_{10} concentration during preceding 40-day period (lag 0-40) in ten cities in Europe and adjacent countries for periods between 1990 and 1996. Data from Zanobetti et al. (2002, 2003)
All cause	0.91 (0.28, 1.54)	1.68 (0.72, 2.65)	1.1 (0.8, 1.3)	1.61 (1.02, 2.20)
Cardiovascular	0.27 (-0.67, 1.22)	0.43 (-0.97, 1.86)	1.1 (0.7, 1.5)	1.97 (1.38, 2.55)
Respiratory	3.10 (1.36, 4.87)	5.36 (2.93, 7.84)	3.6 (3.0, 4.3)	4.2 (1.08, 7.42)
Lung cancer	1.55 (-0.72, 3.87)	-	-	-
Other causes	0.47 (-0.61, 1.56)	2.13 (0.82, 3.47)	-0.2 (-0.7, 0.2)	-
(C) Ratios of temperature adjusted black smoke exposure mortality associations at lag periods of 0-30 and 0-40 days to equivalent associations at lag 0-3 days				
	Present study:	Above studies reported in literature:		
All cause	5.4	-	2.8	2.3
Cardiovascular	2.7	-	2.8	2.9
Respiratory	11.5	-	4.0	5.7

Figure caption:

Figure 1: Time series of black smoke and all-cause mortality in Glasgow, 1 Jan 1974 – 31 Dec 1998.

