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Do current levels of air pollution kill?
The impact of air pollution on population mortality in England

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Abstract

The current air quality limit values for airborne pollutants in the UK are low by historical standards and are at levels that are believed not to harm health. We assess whether this view is correct. We examine the relationship between common sources of airborne pollution and population mortality for England. We use data at local authority level for 1998 to 2005 to examine whether current levels of airborne pollution, as measured by annual mean concentrations of carbon monoxide, nitrogen dioxide, particulate matter less than 10 μm in diameter (PM_{10}) and ozone, are associated with excess deaths. We examine all-cause mortality and deaths from specific cardiovascular and respiratory causes that are known to be exacerbated by air pollution. The panel nature of our data allows us to control for any unobserved time-invariant associations at local authority level between high levels of air pollution and poor population health and for common time trends. We estimate multi-pollutant models to allow for the fact that three of the pollutants are closely correlated. We find that higher levels of PM_{10} and ozone are associated with higher mortality rates, and the effect sizes are considerably larger than previously estimated in time series studies for England.

Key words: airborne pollutants, population mortality, panel analysis

JEL classification: I12, I18

1. Introduction

The current levels of airborne pollutants in many OECD countries are low by historical standards. The limits on air pollution set by the regulatory authorities are also low by these standards. Yet recent research from the USA has shown that there are adverse effects from airborne pollution for infants at levels of pollution that are not dissimilar to those presently allowed in many European countries (Currie and Neidell, 2005).

In this paper we focus on one OECD country, England. England has levels of airborne pollutants that are low by historic and international standards (for example, pollutant levels are lower than those in Currie and Neidell, 2005); and the limit values allowed by the regulatory authorities are set reflecting a belief that there is a safe threshold at which no significant health effects can be observed[1]. The aim of the paper is to examine this belief by establishing whether current levels of airborne pollutants in England are associated with adverse health effects – as measured by mortality – for the population.

Adults have been the main focus of most of the research on air pollution and excess mortality. Previous studies of the impact of airborne pollutants on mortality rates are basically of two kinds. The first exploit high frequency time series data on levels of air pollution and number of deaths to examine the time series relationship. Such studies measure the acute effects of air pollution and generally focus on a single pollutant. However, the focus on a single pollutant may over-estimate its impact, as several of the common airborne pollutants are correlated, because they are components of traffic emissions. In addition, if temporarily elevated levels of pollution hasten the deaths of frail persons who would have died within days or weeks, then the effects of pollution are over-estimated. The second type of study examines the impact of living in cities with different levels of pollution. Whilst these studies capture more than the short term effects of pollution, comparisons of cities suffer from potential omitted variable bias, as it is likely that these cities are different in important ways other than in their level of pollution. So observed cross-sectional differences in deaths may not be causal (Chay and Greenstone, 2003).

In this paper, we use the following design to deal with these problems. We take as the unit of observation the primary unit of local government in the UK (the local authority) and examine the

[1] See http://www.airquality.co.uk/archive/standards.php#std and http://www.airquality.co.uk/archive/standards.php#band

relationship between annual mortality rates and annual mean concentrations of four common air pollutants over time at this level. The use of a panel allows us to fully control for time-varying determinants of death that are national in scope and factors that differ across local areas that remain fixed over time, so we can isolate the impact of pollution from other unobserved differences between local authorities. The use of a time period of a year means this design will not detect the small changes in life expectancy (changes of a few days) that may underlie the associations found in time series studies. Focussing on annual mortality rates also reduces one aspect of model uncertainty found in time series studies (see Clyde, 2000 and Koop and Tole, 2004). Additionally, annual mortality rates for local authorities are readily available, whereas daily, weekly or monthly rates are not publicly available for confidentiality reasons. Finally, the research design allows us to control for the correlation between the levels of common airborne pollutants.

Despite its advantages this design has been little used to examine pollution and mortality. In one of the few studies using this approach, Chay et al. (2003) examine the effect of particulate matter on adult mortality in the US during the 1970s. They find no impact of this pollutant on adult mortality. However, the pollutant measure used during the period covered by their study (total suspended particles) was possibly too imprecise to pick up mortality effects.

Our panel begins in 1998 after Local Air Quality Management came into effect in the UK in December 1997. It ends in 2005. Local Air Quality Management required local authorities to assess the air quality in their areas and, as a result, local authorities installed additional air pollution monitoring stations that supplement the existing national monitoring network[2]. This provides a dense network of air pollution monitors that allows us, using spatial matching methods, to assign air pollution measures for about 90% of local authorities and all of the local authorities with large populations. Our analysis focuses on the pollutants carbon monoxide (CO), nitrogen dioxide (NO_2), particulate matter less than 10 μm in diameter (PM_{10}), and ozone (O_3). European legislation sets limit values for these pollutants, because they have deleterious effects on human health[3].

[2] Note that local authorities are not the same bodies that are responsible for providing health care or meeting health targets.

[3] See Appendix A for sources and effects of these pollutants and Appendix B for the air quality standards in operation in England during our sample period.

We examine deaths from all causes and deaths from specific causes – diseases of the cardiovascular and respiratory system – that have been shown in the medical literature to be associated with air pollution (Pope and Dockery, 2006). We control for observed factors that may be correlated with pollution but are independent causes of early deaths, such as education, employment and lifestyle. We estimate multiple pollutant models to isolate the impact of specific pollutants. We also subject our results to a large number of specification tests, including 'placebo' tests for a spurious association between air pollution at local authority level and death rates by examining the association of air pollution with two causes of death which are unlikely to be driven by air pollution.

Our findings suggest that the levels of pollution currently permitted in the UK are associated with mortality rates in the population. We find significant effects of both PM_{10} and O_3 on mortality. The magnitudes of these effects are both statistically and economically significant.

2. An overview of the literature on air pollution and mortality

The literature on air pollution and mortality is dominated by two types of study – time series studies of the association between short-term variations in air pollution and mortality and cross-sectional studies of cohorts followed over time or of cities with long-term differences in pollution. Time series studies regress daily counts of deaths for a geographical area onto daily means of air pollutant concentrations, controlling for confounding factors such as temperature, humidity and barometric pressure. Exploiting short-term variation to identify pollutant effects eliminates the effects of lifestyle factors such as smoking, exercise and diet, because these factors do not change on the short run. Systematic reviews of the numerous published time series studies report significant associations between air pollutants and mortality, with mean estimates suggesting that per 10 $\mu g/m^3$ increase in NO_2, PM_{10} or O_3 or per 1 mg/m^3 increase in CO mortality increases by less than 1% (see, inter alia, Stieb et al., 2002, Bell et al., 2005, and Department of Health, 2006).

There are two problems interpreting the findings from time series studies. The daily time series design can only identify the acute effect of pollution. Part of the increase in mortality may be caused by deaths of individuals who would have died only a few days later from other causes (an

issue known as "harvesting"). So, such studies may over-estimate the impact of air pollution on health. In addition, levels of different pollutants may be strongly correlated; identifying which pollutant is causing the increased deaths is therefore difficult from studies based on short-term fluctuations in one pollutant.

Ecological studies of associations between spatial variations in air pollution and spatial variations in mortality compare mortality in highly polluted areas with mortality in less polluted areas, using population average values to control for other risk factors such as smoking, deprivation and education. Typically, they suggest that a pollutant increase of 10 $\mu g/m^3$ increases mortality by about 3% (Wilson and Spengler, 1996). But these studies face severe omitted variables problems, as they typically do not control for many individual or community level variables which may be correlated with pollution[4]. Finally, cohort studies use pollutant concentrations averaged over a year or longer periods. Few such studies exist and there are none for the UK. Two key U.S. studies estimate an increase in mortality risk of between 4% and 14% per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (Pope et al., 2002, and Dockery et al., 1993). Estimated effects on cardiopulmonary mortality are generally larger. Estimates of the effects of CO, NO_2 and O_3 tend to be insignificant (Krewski et al., 2000). The only long-term studies for Europe are one for Norway, which finds a mortality risk increase of 8% per 10 $\mu g/m^3$ increase in nitrogen oxides (NO_2 + NO) for men (Nafstad et al., 2004) and one for the Netherlands, which finds positive but insignificant effect estimates for NO_2 (Hoek et al., 2002). Because of their design, cohort studies are expensive and take long time to complete. In addition, cohort studies may suffer from omitted variable bias, as the cities or zip codes which are compared may differ from each other in important ways other than just their levels of air pollution.

Within the economics literature, there have been several studies for the US which show that current levels of pollution are associated with poor health outcomes. Currie and Neidell (2005) examine the impact of CO, PM_{10} and O_3 on infant deaths in California over the 1990s. Using individual-level weekly data, they find a significant effect of CO on infant mortality. Aggregating up their data to zip code-quarter level, however, they find no effect for CO, but a significant effect for PM_{10}. The pollution levels in California during the 1990s are higher than the pollutant

[4] A very small number of studies use exogenous changes in air pollution. Clancy et al. (2002) used the ban on coal sales in Dublin in 1990 which reduced average black smoke concentrations. Studies of extreme pollution episodes use one large fluctuation in air pollutant concentrations to identify short-term effects. A classic example is the Great Smog of London in 1954 that caused 4,000 excess deaths (Wilkins, 1954).

concentrations in England during the period we examine: the sample mean of PM_{10} in Currie and Neidell (2005) is 39.4 μg/m^3, whereas our sample mean is 24.7 μg/m^3. Chay and Greenstone (2003) exploit variation across US counties in the depth of a sudden economic recession in 1980 to 1982 to identify the effect of a medium-term reduction in total suspended particles (TSP - particles with diameter ≤ 40 μm) on infant mortality. Again, pollution levels are higher than currently in England[5]. They find a significant effect of TSP reductions on decreases in infant mortality rates.

In one section of their paper, Chay et al. (2003) use the same approach as we adopt here, using US counties as the unit of observation. They exploit within-county time-series variation in TSP levels to study the effect of air pollution on mortality in adults over 50 years and adults aged 65 to 84 years in 1969 to 1974. The average pollution level in their data is twice the level we examine[6]. However, they find no association between their measure of air pollution and mortality, perhaps because TSP are a rather crude measure of air pollution.

3. Our empirical approach

Our unit of analysis is a local authority, which is the main unit of political administration below the national level in the UK. There are 354 local authorities in England, with an average population of around 140,000 people, ranging from just over 2,000 to just over 1 million[7]. Local authorities are aggregated into 9 Government Office regions. Figure 1 shows the location and size of local authorities and the Government Office regions.

We estimate equations of the following form:

$$(1) \qquad m^j_{it} = \alpha + P'_{it}\gamma_j + Z'_{it}\beta_j + T^j + T^j_r + \mu^j_i + \varepsilon^j_{it}$$

where *i* indexes the local authority, *t* indexes the year, *r* the region and *j* the cause of death. m^j_{it} is the logarithm of one of six mortality rates (all cause; all circulatory diseases; coronary heart

[5] TSP is not measured in England during our sample period. To compare pollution levels, we convert TSP levels using 0.55 as PM_{10}/TSP ratio. In a review of studies of the acute effects of particles, Dockery and Pope (1994) use this ratio, based on guidelines from the US Environmental Protection Agency. Chay and Greenstone (2003) report TSP levels between 56.4 and 71.1 μg/m^3, which is equivalent to PM_{10} levels between 31 and 39 μg/m^3, one and a half times our sample mean of 24.7 μg/m^3.

[6] Using 0.55 as PM_{10}/TSP ratio their TSP sample mean of 93 μg/m^3 is equivalent to a PM_{10} level of 51 μg/m^3. Our PM_{10} sample mean is 24.7 μg/m^3.

[7] The smallest local authority used in the analysis here contains 34,000 people (Rutland) and the largest 1 million people (Birmingham).

disease; acute myocardial infarction; stroke; bronchitis, emphysema and other chronic obstructive pulmonary diseases), P_{it} is a vector of air pollutants (CO, NO_2, PM_{10}, O_3), Z_{it} is a vector of time-varying controls at local authority (or regional) level. T^j is a time trend, T^j_r is a regional specific time trend (regions are Government Office regions), μ^j_i is a local authority fixed effect, and ε^j_{it} is the error term for cause of death j. The coefficients of interest are the γ_j.

We first estimate the impact of each pollutant separately, but our main specifications include all pollutants together to allow for correlation between them. Identification comes from the time series variation in pollutant concentrations at local authority level. As our panel is short, within-group estimates may be biased, so we also estimate OLS models (in which the local authority fixed effect is replaced by a set of regional dummies) and three-year long-difference models (Griliches and Hausman, 1986). In all our analyses we estimate robust standard errors and weight by the size of the local authority population.

4. Data

Data on air pollution comes from the UK Air Quality Archive[8], supplemented with data from four regional air quality networks managed by the same operator and from another four regional networks managed by the Environmental Research Group at King's College London. These sources provide data on a total of 192 automatic monitoring stations, of which 90, 174, 111 and 105 record concentrations of CO, NO_2, PM_{10} and O_3, respectively. Figure 2 shows the positions of these monitors[9]. The figure also shows the population densities of local authorities; the darker the shading, the more densely populated the area. It is clear from the figure that monitors are located in more densely populated areas, so that, while there is not equal coverage across areas, those areas with few monitoring stations are also areas of small populations.

We convert measurements given in volume ratios into mass units and compute daily pollutant concentrations if only hourly readings are available (see also Appendix C). We use the daily mean of NO_2 and PM_{10} and the daily maximum 8 hr running mean of CO and O_3 (the choice of unit is determined by the relevant pollution standard) to calculate annual means. We assign these

[8] Prepared by AEA Energy & Environment on behalf of the Department for Environment, Food & Rural Affairs, www.airquality.co.uk

[9] The map does not show two monitoring stations in North Wales close to the English border, which we use for computing air pollution measures for local authorities in the West Midlands and in the North West.

annual pollutant concentrations to local authorities using a procedure similar to Currie and Neidell (2005). Using the geographical coordinates of the headquarters of a local authority, we calculate the distance between the headquarters and all monitoring stations. Then we use all monitoring stations whose distance to the headquarters is less than 30 miles (less than 10 miles for the London boroughs where there are many monitoring stations within relatively small distances) to calculate a weighted mean of the annual pollutant concentrations measured by these stations. The weight assigned to a monitor is the inverse of the distance between the headquarters and the monitor. Our measure is thus the distance-weighted mean of the annual mean pollutant concentrations at monitors in a 30 (10) mile radius of the headquarters of a local authority. We assign a measure of CO, NO_2, PM_{10} and O_3 for at least two years to 312 out of 354 local authorities. The local authorities with missing air pollution measures are less populated areas.

To assess the accuracy of our pollution measure, we use our method to predict pollutant concentrations at monitor locations and compare the predicted with the actual pollutant concentrations. For the underlying daily data the correlations are relatively high (0.59, 0.61, 0.75 and 0.84 for CO, NO_2, PM_{10} and O_3, respectively), indicating this approach will predict pollution at a location relatively well. The correlation coefficients for the annual data across all observations are lower at 0.44, 0.45, 0.40 and 0.50 for CO, NO_2, PM_{10} and O_3, respectively, due to the averaging induced by moving from daily to annual measures. However, the time series correlation between the predicted and actual annual values *within* monitoring stations is higher – 0.72, 0.47, 0.53 and 0.73 – for CO, NO_2, PM_{10} and O_3 respectively[10]. Since our identification strategy relies on time series variation within local authorities, the accuracy of our pollution measure seems reasonable.

Using measurements taken by stationary monitors at outside locations to calculate exposure to air pollution, there may be an issue of the extent to which measures of ambient air pollution predict personal exposure, as most people spend over 80% of their time indoors. Indoor air quality is often worse than outdoor air quality, because of cigarette smoke, paints, vinyl flooring, gas stoves, dust mites etc. However, empirical studies have shown that ambient levels of air

[10] Figures are mean within station correlations. The median within station correlations are higher: 0.87, 0.56, 0.64, and 0.79.

pollutants and personal exposure to air pollutants are significantly correlated[11]. Personal exposure is determined by outdoor concentrations, indoor concentrations and activity patterns, but as factors determining indoor concentrations, e.g. gas stoves and tobacco smoke, do not change over relatively short time periods the major part of the variation in personal exposure to air pollutants is determined by changes in ambient levels of pollutants[12].

Figure 3 presents quantile plots of our pollution measures, showing the time series variation in the annual pollutant levels. CO clearly declines over the years of our sample. There is also a reduction in the variation: the distance between the top two quantiles and the other three quantiles of the distribution falls over time. Measured at an annual level, no local authority exceeds the limit value, which is defined in terms of the daily maximum 8 hour running mean. The annual mean level of NO_2 initially declines before it peaks in 2003. The variation across local authorities remains pretty constant across the sample period. NO_2 exceeds the limit value of 40 $\mu g/m^3$ in many local authorities. Even in the year in which there were fewest instances of exceedances (2002), average annual levels of NO_2 were higher than the limit value in 17% of local authorities. Annual means of PM_{10} fall until 2000, remaining relatively constant since then, apart from a peak in 2003. The distribution is pretty constant over the period. PM_{10} does not exceed 40 $\mu g/m^3$, which is the limit value in force towards the end of our sample period, but it does exceed 20 $\mu g/m^3$, the limit value which will come into effect at the end of 2010. In contrast to the three other pollutants, annual means of O_3 rise over the sample period. The variance of the distribution is fairly constant. There are two clear peaks in the series which affect all local authorities, one in 1999 and another one in 2003. Both years had above average sunshine, illustrating the potential difficulty of isolating the impact of O_3 from that of weather conditions.

[11] Georgoulis et al. (2002) use measurements of personal exposure to CO for 401 individuals in five European cities during a 48 hour period and find that ambient levels of CO are a significant determinant of personal exposure to CO. Kousa et al. (2001) use the same data and find that ambient levels of NO_2 explain 11 to 19% of personal NO_2 exposure variation. However, cross-sectional correlation coefficients between personal exposure and ambient pollutant concentrations can be misleading. For example, Janssen et al. (2000) study the time-series correlation between ambient levels of $PM_{2.5}$ and personal exposure to $PM_{2.5}$ for elderly subjects with cardiovascular disease in two European cities. They find that personal exposure and ambient concentrations are highly correlated *within* subjects over time.

[12] O_3 has considerably lower indoor concentrations (Department of Health, 1997). Thus, for people who spend little time outdoors, personal exposure to O_3 and ambient levels of O_3 are not correlated. O_3 concentrations, however, are elevated in summer, and people tend to spend more time outdoors in summer. Hence, our measure of O_3 should explain at least part of the variation in personal exposure to O_3.

The top panel of Table 1 presents descriptive statistics for the pollution data. In addition to the average fall in all pollutants other than O_3, it shows that the values of the within-local authority standard deviations range from 45% to 80% of the values of the between-local authorities standard deviations. This provides support for identification of air pollution effects by exploiting within-local authority variations. CO, NO_2 and PM_{10} are positively correlated, with correlation coefficients between 0.4 and 0.6. They are negatively correlated with O_3, which tends to be higher in rural areas, with correlation coefficients between -0.2 and -0.5 (see Table A1).

The second panel of Table 1 presents the mortality rates. Sources are given in Appendix C. We examine deaths from all causes as well as deaths from specific causes for which the medical literature suggests biologically plausible mechanisms that hypothetically link air pollution and adverse effects on human health (see Pope and Dockery, 2006, and Pope et al., 2004). Mortality from all circulatory diseases comprises the ICD-10 categories I00 to I99. Mortality from coronary heart disease is a subset of mortality from all circulatory diseases (ICD-10 categories I20 to I25). Mortality from acute myocardial infarction (heart attack), in turn, is a subset of mortality from coronary heart disease (ICD-10 I21 to I22). Mortality from stroke (ICD-10 I60-I69) is another subset of mortality from all circulatory diseases. Mortality from bronchitis, emphysema and other chronic obstructive pulmonary diseases consist of the categories J40 to J44, which are a subset of diseases of the respiratory system. The subset J40 to J44 excludes asthma, pneumonia and – most important – influenza, thus avoiding confounding of the pollutant effects by epidemics, which might coincide with increased air pollution. We use directly age-standardised rates to control for different population age structures across local authorities.

Time series plots of the annual means for the six mortality rates (available from the authors) show a strong downward trend for the cardiovascular mortality rates. Many factors are likely to cause this fall, including the National Service Framework for Coronary Heart Disease (Department of Health, 2000), a ten year plan initiated in 2000 with the aim of reducing coronary heart disease in the community. On the other hand, respiratory mortality has only a slight downward trend with peaks in 1999 and 2003. Consequently, the downward trend in mortality from all causes, which encompasses both cardiovascular and respiratory mortality, is less pronounced and levels off after 2001 before continuing in 2004.

The time-varying control variables in Z_{it} in (1) are the smoking rate, the employment rate, the percentage of working-age people who hold qualifications at degree level and above, the annual mean of summer daily maximum temperature and the annual mean of precipitation. Smoking is a strong predictor of premature mortality and an important source of indoor pollution. It is therefore important to control for smoking rates. Smoking rates are for 1998 and 2000 to 2005 for Government Office regions, which we match to the 354 local authorities in England. We interpolate rates for 1999. Employment rates proxy economic conditions, which may be correlated with health. In an analysis of US data, Ruhm (2000) shows that mortality rates fall when the economy temporarily deteriorates (though Gerdtham and Johannesson (2003) show that in Sweden unemployment increases the risk of dying). Education, in contrast, has a well established positive effect on health. We measure education as the percentage of working-age people who hold qualifications at first degree level or higher.

The effects of air pollution could be confounded with weather conditions[13]. To control for these, we use surface observation data on daily maximum temperatures and daily rainfall amounts, which we assign to the headquarters of the local authorities with the same procedure we use for the pollutants. Firstly, we calculate for all weather stations the annual means of precipitation and the annual means of the daily maximum temperature during the summer months April to September. Then we determine the distance of all stations to the headquarters of a local authority. Finally, we calculate weighted means of rainfall and temperature, using the annual means of all stations within a 10 miles radius and a 20 miles radius, respectively. The inverse of the distance between the headquarters and the weather station provides the weight. These measures should capture the effects of heat waves (for example, the summer of 2003) and very wet years.

The third panel in Table 1 presents descriptive statistics for the controls. Mean smoking rates fell from 27.4% in 1998 to 23.6% in 2005, possibly reflecting the government's efforts to reduce smoking prevalence (Department of Health, 1998). Mean employment rates and mean degree-level qualification rates increased between 1998 and 2005. Mean temperatures have increased during the sample period, with peaks in 1999 and 2003. Precipitation seems to have fallen, but

[13] For instance, during heat waves, O_3 levels rise because of the greater sunshine. Without controls for temperature, it may appear that O_3 increases mortality, while in fact the heat caused excess deaths. On the other hand, to the extent that weather is associated with the level of pollution but does not have an independent effect on deaths, inclusion of weather variables will reduce the amount of variation in our pollution measures and make it more difficult to detect their effects.

the trend is less clear. As for the pollutants and the mortality rates, there is significant within local authority variation.

5. Results

(a) Cross-sectional associations

Figure 4 maps the cross-sectional spatial distributions of mean all cause mortality and mean local authority pollutant concentrations. Five different shades indicate the quintiles of the respective distribution. The figure shows a similar spatial distribution for three of the pollutants – CO, NO_2 and PM_{10} – which are higher in urban areas, while O_3, is higher in rural areas. There is no clear north-south divide in this rural-urban split of pollution. In contrast, all cause mortality shows a marked north-south split, death rates being higher in the north and lower in the more affluent south. So in the raw data, averaged over the sample period, there is little correspondence between the spatial distribution of mortality rates and of air pollutant concentrations.

Table 2 examines this further by reducing the information on variation shown in the maps to a split of the sample into tertiles of the pollutant distributions and showing mean mortality from all causes across these tertiles. There is some indication that higher concentrations of CO and NO_2 are associated with higher mortality rates. For example, the mean mortality rate for observations in the highest third of the NO_2 distribution is 1.8% higher than the mean rate for the lowest third. The relationship, however, is not linear, with the mean rate for the middle third being greater than the mean rate for the highest third. In contrast, highest concentrations of O_3 are associated with lower death rates. There is no clear relationship between PM_{10} and mortality, with the mean mortality rate for the middle third being smallest and the rates for the lowest and the highest third being similar.

(b) The relationship between each pollutant and all cause mortality

We start with an analysis of all cause mortality to see if air pollution has any impact on this aggregate measure. We then focus on the specific causes of deaths for which the medical literature suggests they are causally related to air pollution (Pope et al., 2004). We begin by examining the separate association between each pollutant and mortality. The first column of Table 3 presents the raw correlations, estimated by an OLS regression of the log of all cause mortality on a constant and the pollutant. We then control for trend, region and region-specific

trends and present OLS, within-group and long-difference estimates. We then add the time-varying controls for weather and for lifestyle differences between local authorities. We multiply the outcome variable by 100 and divide the NO_2, PM_{10} and O_3 levels by 10, so the coefficients are estimates of the percentage change in the mortality rate per 10 $\mu g/m^3$ increase in NO_2, PM_{10} or O_3 or per 1 mg/m^3 increase in CO.

The first block of Table 3 shows the estimates for CO. This shows no association between CO and all cause mortality, apart from a slightly significant positive coefficient in the OLS equation with controls for time, region, weather and lifestyle, but this is not robust to the inclusion of local authority fixed effects. The second block shows the results for NO_2. The raw association is positive but not significant. The coefficient estimates are significantly positive after controlling for trend and region, but adding the controls for lifestyle and weather makes the within-group and long-difference estimates insignificant. The results for PM_{10} in the third block show a positive but insignificant raw association and significantly positive coefficients for all other specifications. The within-group and long-difference estimates are similar. The within-group estimate suggests that a 10 $\mu g/m^3$ increase in PM_{10} is associated with a 2.8% increase in all cause mortality.

The final block shows the results for O_3. The raw correlation is negative and significant, showing the association seen in Figure 4: rural areas, which have lower mortality rates, have higher O_3 concentrations. Adding time varying controls does not change this negative sign, though the point estimate is considerably smaller. Allowing for local authority fixed effects, however, changes the direction of the association. Both the within-groups and the long-difference estimates indicate a positive effect of O_3 on all cause mortality. The within-groups point estimate is a 0.7% increase in all cause mortality for a 10 $\mu g/m^3$ increase in O_3.

(c) The relationship between all pollutants simultaneously and all cause mortality

Table 4 repeats the analyses of Table 3, but includes all pollutants simultaneously to allow for correlation between the pollutant levels. It confirms that CO has no independent effect on death rates. For NO_2 the within-group and long-difference estimates are again significantly positive when controlling only for trend, region and regional trend, but become insignificant when adding the controls for lifestyle and weather.

The coefficient on PM_{10} remains significantly positive in all specifications, though it falls by 2 to 40%. The within-group estimate from the specification with all controls suggests that per 10 $\mu g/m^3$ increase in PM_{10} the all cause mortality rate increases by 2.7%. The corresponding long-difference estimate suggests an impact of 2.4%, not significantly below the within-group estimate. The associations between O_3 and mortality in the multi-pollutant model are similar to those estimated by the single-pollutant model, though the negative coefficient estimate in the OLS specification with all controls becomes insignificant. Using the within-group specification with all controls, the estimated impact of O_3 is 0.8% per 10 $\mu g/m^3$ increase. The corresponding long-difference estimate is not significantly different from zero. However, the long-difference sample is much smaller, so the effect of O_3 might be masked by the control for summer temperatures in this smaller sample. We therefore give more credence to the within-group estimates[14].

The association of the controls with all cause mortality are shown in the final three columns of Table 4. As expected, smoking rates are positively associated with higher death rates. The estimate, however, is (marginally) significant only in the OLS specification. The employment rate and the degree-level qualification rate are negatively associated with death rates, but the coefficients are significant only in the OLS specification, indicating that these variables are capturing unobserved differences between local authorities rather than the effect of time variation in employment and education on death rates[15].

(d) The relationship between pollutants and specific causes of mortality

The medical literature suggests that the association between air pollution and mortality is driven by deaths from cardiovascular and respiratory causes (see, for example, Bell et al, 2005 and Pope et al., 2002). Several pathophysiological pathways that link particulate matter and mortality from cardiovascular diseases have been suggested (see Pope and Dockery (2006) and Department of

[14] Estimates using differences two periods apart support this argument, as they are closer to the within-group estimates. The point estimates are, in fact, larger than the within-group estimates: NO_2 0.59 (s.e. = 0.28), PM_{10} 2.50 (s.e. = 0.63), O_3 0.81 (s.e. = 0.32), 1,701 observations, 304 groups.

[15] We also tested the robustness of our results to defining economic activity in terms of unemployment instead of employment and to inclusion of an additional control for local pay rates (the log of average male pay). We found very similar results: no measures of economic conditions were significantly associated with all cause mortality in models which controlled for local authority fixed effects.

Health 2006). The two main hypotheses are the clotting hypothesis and the neural hypothesis[16]. From the first, we would expect to find positive associations between PM_{10} and mortality from coronary heart disease in particular, but also stroke, heart failure and atherosclerosis (Pope et al., 2004). Therefore, we examine mortality from all circulatory diseases, coronary heart disease, acute myocardial infarction (heart attack) and stroke. Data on mortality from heart failure and atherosclerosis on local authority level are not publicly available. We are not able to examine the pathways suggested by the neural hypothesis.

Table 5 presents these estimates. The first column repeats the within-group estimates for all cause mortality from Table 4 for comparison. The results show that PM_{10} is positively associated with all four cardiovascular mortality rates. We find a large and highly significant positive effect on mortality from coronary heart disease (a subset of mortality from all circulatory diseases), for which we should find a strong effect according to the clotting hypothesis. The estimates suggest that a 10 $\mu g/m^3$ increase in PM_{10} increases each of the four specific mortality rates by around 4 to 5%. O_3 is positively associated with mortality from bronchitis, emphysema and other chronic obstructive pulmonary diseases, suggesting that the association between O_3 and mortality is driven by mortality from respiratory causes. The coefficient is significant at the 10% level only, perhaps because the relatively small death rates (around 30 per 100,000 population) do not allow the effect to be estimated precisely enough.

Thus, we find that pollution levels are associated with those specific causes of death that are indicated in the literature on the pathways by which pollution leads to death. Further, our estimates suggest that the effects of pollution on these specific causes of death account for a high fraction of the estimated effect of pollution on all-cause mortality. Using the sample mean for each specific mortality rate from Table 1 and applying our estimates from Table 5, the overall estimated effect of PM_{10} on coronary heart disease and stroke accounts for 80% of our estimated

[16] The clotting hypothesis suggests that particles penetrating into the lungs cause an inflammatory response in the lungs. The inflammation in turn might trigger changes in the control of blood clotting, causing, for example, thrombosis. Alternatively, the inflammation might change chemical factors in the blood that affect the stability of the atheromatous plaques in the arteries that supply blood to the heart muscle. The atheromatous plaques might rupture, causing a blockage of the artery. The neural hypothesis proposes that inhaled particles might trigger a reflex that leads to subtle changes in the heart rhythm, making the heart more susceptible to dangerous changes in the rhythm that potentially cause sudden death. Therefore, we would expect to find positive associations between particulate matter and mortality from dysrhythmias, heart failure and cardiac arrest (Pope et al., 2004). Data on mortality from these causes on local authority level are not publicly available, so we cannot examine this potential pathway.

effect of PM_{10} on all circulatory diseases, while the effect on mortality from circulatory disease accounts for 60% of our estimated effect on all deaths.

(e) The relationship between all pollutants and all cause mortality for different age groups

The literature suggests that children and elderly persons are most likely to be susceptible to air pollution (Pope and Dockery, 2006). So if our results indicate some causal link, we should find greater effects for these age groups. Table 6 presents within-group estimates of the association between air pollutants and all cause mortality by broad age groups: under 15 years, between 15 to 64 years, 65 to 74 years and older than 75 years. As directly age-standardised rates are not publicly available for the older than 75 years group, we use non-age-standardised data and control for population age structure by including controls for proportions of age groups in 5-year age bands on the right-hand side.

For comparison, the first column of Table 6 presents estimates for all ages using the all cause mortality rate that is not age-standardised. The coefficients are similar to those obtained using age-standardised rates. Columns 2 to 5 of Table 6 show that the effects of PM_{10} and O_3 are largest for the most vulnerable groups. The PM_{10} estimates are largest for the youngest age group. The absolute impact is smaller, because of the very low death rates in this age group. At the mean of the under 15 years old mortality rate, 44 per 100,000, a 10 $\mu g/m^3$ increase in PM_{10} increases the number of deaths by 4, whereas the coefficient estimate for the over 75 years old suggests that at the mean mortality rate of 10,556 per 100,000 a 10 $\mu g/m^3$ increase in PM_{10} increases the number of deaths by 331. The coefficient on O_3 is significant only for the over 75 years old, suggesting that the coefficient estimate for O_3 in the all-ages specification is driven by this age group. Disaggregation by age also shows an effect of NO_2 (again for the elderly) and for CO (in this case for those aged 15 to 64).

6. Robustness checks

Our method involves assignation of air pollution levels to local authorities and the estimation of a linear relationship between pollution and death rates. We subject these assumptions to robustness tests. We further explore whether our results are indicative of a causal relationship by first undertaking 'placebo tests' and second by examining whether confounding factors could account for the association we find between pollution and mortality. The results of our robustness tests

are summarized in Table 7. The baseline estimates in row 1 are the within-group estimates from the specification with all four pollutants simultaneously and the full set of controls in the last block of Table 4.

(a) The assignation of air pollution to areas

Our air pollution measure is the distance-weighted mean of the annual mean pollutant concentrations at monitors within a 30 mile radius (10 miles for London) of the headquarters of a local authority. Row 2 of Table 6 presents estimates using a 20 mile radius (5 miles for London). The number of local authorities to which we can assign an air pollution measure drops from 312 to 267. The coefficients on PM_{10} and O_3 fall by 20% and 5%, respectively, but they are still significantly positive.

To calculate our air pollution measures we used monitoring stations that are situated in different environments, for example in urban areas, at roadsides or in rural areas. If a local authority has mainly roadside or kerbside monitoring stations, actual exposure might be lower than our measures suggest. Row 3 of Table 7 examines robustness of our results when we omit readings from kerbside and roadside stations. The number of local authorities to which we can assign a pollution measure drops from 312 to 243. Though the coefficients on PM_{10} and O_3 drop by 20% and 5%, respectively, they are still significantly positive.

The summer of 2003 was unusually hot. This was also a year with higher death rates and higher O_3 and PM_{10} levels. Row 4 examines the robustness of our results to omission of this year. The estimated impact of both PM_{10} and O_3 falls by around a third, as might be expected given this year is an outlier, but PM_{10} remains well defined[17]. More generally, to test that our results are not driven by areas with high levels of pollution which may not be representative of England, we omit observations with one or more pollutants in the top 10% of the pollutant distribution. Row 5 shows that the results are robust to this omission.

[17] If we allow for a full set of year dummies the coefficient on PM_{10} falls to 1.12 (s.e. = 0.54) and the coefficient on O_3 falls to -0.24 (s.e. = 0.32). However, both weather coefficients have incorrect signs (the coefficient on hot weather is significantly negative and the coefficient on precipitation is positive and significant) and several of the year dummies are not significantly different from each other. We conclude that we cannot identify separate year, pollution and weather effects. A more parsimonious time specification that fits the time pattern in death rates (a spline with knots in 1999 and 2003, both of which are years with higher death rates and higher temperatures) gives significant positive coefficients for both pollutants and summer temperature (PM_{10} 2.18 (s.e. = 0.55), O_3 0.80 (s.e. = 0.29), summer temperature 0.66 (s.e. = 0.20)).

Our assignment of pollution measures to local authorities is based on distance to monitoring stations, without taking into account wind direction, which is predominantly from the west in England. Stations located in the South West, in particular, will have measures predominantly based on stations to their east. To examine whether this is a problem, Row 6 omits observations in the South West. Our results are little affected by omitting these areas.

Air pollution – at least from CO_2, NO_2 and PM_{10} – might be an urban phenomenon. We therefore checked that our results were not solely due to London by omitting all London observations. Row 7 shows that the estimates for PM_{10} and O_3 fall by around 20% but remain significantly positive. The estimate for NO_2 increases by 30% and becomes significantly positive[18].

(b) Dynamics and non-linearities

We were concerned that we might have mis-specified the dynamic structure of the model. Row 8 therefore includes the lagged levels as well as the current levels of the pollutants. The estimated effects of current PM_{10} and O_3 change slightly but remain statistically significant[19]. We also conditioned on lagged mortality. Again, our results were robust to this, suggesting that the local authority fixed effects do a good job of picking up unobserved heterogeneity between local authorities.

Our model assumes that the impact of air pollution on mortality is linear. We investigate non-linearities using splines in the levels of PM_{10} and O_3 in a within-group specification controlling for CO, NO_2, trend, region-specific trends and our full set of covariates. We place two knots at the 33rd and 66th percentiles, dividing the pollutant data into tertiles. Table 8 presents the results. For PM_{10} the coefficients for the middle tertile and the highest tertile are larger than the coefficient for the lowest tertile, though the relationship is not linear, with the largest estimate for the middle tertile. For O_3 there seems to be a negative relationship, with the coefficient for the

[18] We also checked if there are regional differences in the impact of PM_{10} on mortality. The region pattern is quite complex, but there is a group of regions where the impact of PM_{10} is largest, both for all cause mortality and the specific mortality rates. These regions are South West, East Midlands and North East.

[19] The coefficients on the lagged pollutants are small and insignificant for three of the four pollutants. For O_3, however, the coefficient on the lag is similar and of opposite sign to that of current O_3. This result might indicate that the impact of O_3 is to bring mortality that would have otherwise occurred forward (harvesting). Conditional on a positive association with the current level of pollution, a negative coefficient on the lagged level could indicate harvesting, since individuals who died last year are not available to die this year. However, the issue of harvesting has less force for annual data as – by definition – the mortality rates and the measures of pollution average out short run increase and decreases. In our data years with higher than average O_3 are preceded by years with lower than average O_3: it seems likely that in this short time series this is what the lagged coefficient is picking up.

lowest tertile being larger than the coefficient for the middle tertile and the coefficient for the middle tertile being larger then coefficient for the highest tertile. The estimates for the middle and the highest tertile, however, are not significantly different from zero.

We also tested whether the annual maxima of the weekly means of the pollutant concentrations have an impact on mortality to determine whether the long-term average level of air pollution or short-term peaks drive the relationship between air pollution and mortality. Row 9 in Table 7 presents the results. The first line shows the coefficients for the annual mean pollutant concentrations, the second line the coefficients for the annual maxima of the weekly mean pollutant concentrations. The coefficient for the annual mean level of PM_{10} drops by around one third, but is still significantly positive. The level of PM_{10} in the week with the highest PM_{10} level is positively associated with the annual mortality rate, though the size of the effect is only one fifth of the effect of the annual mean level of PM_{10}. The coefficient for the annual mean level of O_3 drops by less than 10%, and the coefficient for the maximum weekly level of O_3 is not significantly different from zero. The coefficient for annual mean NO_2 becomes significantly positive, but the coefficient for maximum weekly NO_2 has an unexpected negative sign and largely offsets the effect of annual mean NO_2, leaving the joint effect similar to the baseline estimate[20]. These tests show that there is some evidence of non-linear effects, but they do not change our main finding that PM_{10} and O_3 are positively associated with mortality.

(c) Placebo tests

It is possible that the association of mortality with pollution does not result from pollution effects, but that our pollution measures are proxies for some omitted factor which is correlated with pollution, but itself is the cause of deaths. To some extent, this is already dealt with by using local authority fixed effects and region-specific time trends. Any non-time-varying factors – such as poor health care services or the presence of health risks in urban settings – will be controlled for by the fixed effects, and the region-specific trends will pick up changes over time at regional level. However, it is possible that there are omitted time-varying factors at local authority level that are correlated with changes in pollution and that are driving our results.

[20] The somewhat odd results for NO_2 may be due to collinearity. In fact, annual mean NO_2 and maximum weekly NO_2 are strongly correlated, with $r = 0.86$. The correlation coefficients for the other pollutants are smaller: annual mean CO and maximum weekly CO: $r = 0.65$, annual mean PM_{10} and maximum weekly PM_{10}: $r = 0.65$, annual mean O_3 and maximum weekly O_3: $r = 0.57$.

One way of testing for this is to examine mortality from causes that are unlikely to be affected by the within local authority time series variation in pollution. If the coefficients for the air pollutants are similar to those found in the baseline specification, then this suggests that some omitted factor may be driving the association we find between air pollution and mortality rates. Two candidate causes are chronic liver disease (including cirrhosis) and infectious and parasitic diseases. Rows 10 and 11 report the coefficients on the pollutants for the baseline specification with age-standardised mortality rates from liver disease and infectious and parasitic diseases as the dependent variable. The baseline specification (as all others in the table) includes the full set of controls to allow for the fact that mortality from liver disease and from infectious and parasitic diseases may be associated with the economic cycle and weather. The results show none of the coefficients on the pollutants are statistically significant, apart from a marginally significant coefficient on O_3 for mortality from liver disease.

(d) Mitigating response to pollution: Population mobility

Our estimates are weighted by the size of the local authority population, giving more importance to local authorities with bigger populations and consequently more reliable mortality measures. The population size, however, might have an independent impact on mortality other than affecting the precision of the mortality rate. For example, a population could shrink because healthy people leave. Consequently, the proportion of frail people would increase, causing an increase in mortality. If healthy people leave because of upward-trended air pollution, the increase in mortality might wrongly be assigned to the rise in air pollution rather than the fall in population. To test this, row 12 in Table 7 controls for the population size. The coefficients on PM_{10} and O_3 are unaffected. The coefficient on population size is significantly negative. Assuming that changes in the population size are mainly caused by migration, this result supports the idea that healthy people are more mobile, leaving a more frail population behind. These moves, however, do not appear to be a response to pollution levels.

(e) Magnitudes

Our results are statistically significant, but are they economically significant? The within-group estimates from the penultimate column of Table 4 can be used to examine the effect of a change in PM_{10} and O_3 on mortality. We focus on all cause mortality.

Assuming no behavioural response (an issue we return to below) a 10 $\mu g/m^3$ increase in PM_{10}, holding all other pollutants fixed, is associated with a 2.7% increase in the all cause mortality rate. As the mean all cause mortality rate is 660 per 100,000 population, this increase equals around 18 more deaths per 100,000 persons. The 10^{th} percentile of the PM_{10} distribution is 20.9 $\mu g/m^3$, the 90^{th} percentile is 29.0 $\mu g/m^3$, and so a move from the 10^{th} percentile to the 90^{th} percentile of the PM_{10} distribution would be associated with around 14 more deaths per 100,000 population. A 10 $\mu g/m^3$ increase in O_3, holding all other pollutants fixed, is associated with a 0.8% increase in the all cause mortality rate. The 10^{th} percentile of the O_3 distribution is 47.1 $\mu g/m^3$, the 90^{th} percentile is 66.4 $\mu g/m^3$, so a move from the 10^{th} to the 90^{th} percentile would be associated with 10 more deaths per 100,000 population.

Alternatively, the difference between the 90^{th} percentile and the 10^{th} percentile of all cause mortality is 225 deaths per 100,000 population. So, a fall from the 90^{th} to the 10^{th} percentile of PM_{10} would account for about 6% of the spread in all cause mortality, while moving from the 90^{th} to the 10^{th} decile of the O_3 distribution would account for around 4% of the spread in all cause mortality.

These effects can be compared to those from the cohort and time series studies. We would expect our estimates to lie between those of the cohort studies, which measure the impact of air pollution over a long period (and cannot control for unobserved heterogeneity across individuals), and the time series estimates, which measure the immediate response to a change in air pollution. The American Cancer Society Cohort Study estimates that a 10 $\mu g/m^3$ increase in fine particles, $PM_{2.5}$, would lead to a 6% increase in all cause mortality (Pope et al., 2002). The health effects from fine particles are worse than the effects from coarser particles, which the PM_{10} measure includes but the $PM_{2.5}$ measure excludes. Thus, we would expect our estimate to be lower. A meta-analysis of the time series studies (Stieb et al. 2002) reports that multi-pollutant models estimate a 0.4% increase in mortality per 10 $\mu g/m^3$ increase in PM_{10}. Our estimates indicate a 10 $\mu g/m^3$ increase in PM_{10} is associated with a 2.7% increase in mortality. So our estimate is about half the size of that from the cohort study – which has no UK counterpart – and nearly seven times as large as those from times series studies that have been undertaken for the UK[21].

[21] Our estimate of the impact of PM_{10} over a year is similar to the impact of a PM_{10} reduction caused by a 13-month strike at a steel mill in Utah (Pope, 1996).

There is no single estimate of the effect of O_3 from the American Cancer Society Cohort Study. Time series studies estimate a 0.3% death rate increase per 10 $\mu g/m^3$ increase in O_3 in single-pollutant models and a 0.1% increase in multi-pollutant models (Stieb et al., 2002). In our analysis, a 10 $\mu g/m^3$ increase in O_3 is associated with a 0.8% increase in mortality. Again, our estimate is considerably higher than those from UK studies undertaken to date.

The extent to which we can use our estimates to quantify the effects of a change in pollution depends on whether individuals are likely to take actions to protect themselves from increases in pollution levels. Neidell (2004) finds that people in California respond to information about air pollution (smog alerts) with avoidance behaviour. In England air pollution alerts have to be issued when NO_2 levels exceed 400 $\mu g/m^3$ or when O_3 levels exceed 360 $\mu g/m^3$ (240 $\mu g/m^3$ since September 2003). Since these thresholds came into force in 2001 no alert has been issued. And while air pollution forecasts are freely available via a variety of sources[22], anecdotal evidence shows that use of this information is limited. For example, in 2006 the Sussex Air Quality Partnership piloted a service for respiratory sensitive people that sends air quality forecasts to mobile phones. The study found that the service raised awareness of pollution episodes and produced health behaviour modifications (Sussex-air, 2008). However, the same information had been freely available before the service was introduced. Individuals appeared to respond to air quality forecasts only when they received them as personalised messages.

Assuming the extreme position of no behavioural response, our estimates can be used to give a back-of-the-envelope calculation of the benefits of the reduction in the limit value for PM_{10} to 20.0 $\mu g/m^3$ by 2010. We estimate a 10 $\mu g/m^3$ increase in PM_{10}, holding all other pollutants fixed, is associated with a 2.7% increase in all cause mortality. Therefore, reducing PM_{10} pollution from our sample mean of 24.7 $\mu g/m^3$ to 20.0 $\mu g/m^3$ (a fall of just under 20%) would be associated with 8.4 fewer deaths per 100,000 population. The population of England is just over 50m, so this translates into around 4,200 fewer deaths per annum over the whole population of England. Putting a monetary value on these lives saved is less straightforward, because we do not know the life expectancy of those who die prematurely. A value per year of life can be taken from the implicit figure used by the UK body responsible for authorisation of the use of new drugs and therapies in the NHS, which is around £30,000 (Devlin and Parkin, 2004). If we assumed that

[22] Teletext, the World Wide Web, a Freephone telephone service and weather forecasts in newspapers, on TV and radio.

those who died had another 10 years to live and were healthy, the value of the 42,000 life years gained is around £1,260 million. If those who died were less healthy, then our estimate is too high. But as we do not take into account any of the non-mortality costs associated with air pollution, this figure is more likely to be a lower bound[23], [24].

7. Conclusions

We identify the impact of airborne pollutants on mortality from time series variation in annual average pollution levels in English local authorities. Our results suggest that higher levels of PM_{10} and O_3 are associated with higher mortality rates, that these pollutants are associated with higher death rates amongst those groups that are likely to be affected by pollution and from those causes that the medical literature indicates are most likely to be associated with pollution. In addition, we find no association between pollution and causes of death that are not affected by pollutants. This suggests that although we cannot exploit a natural experiment and have to rely on annual time series variation at the local authority level, we do identify a causal relationship.

Finally, this paper finds an association between pollution and mortality at average levels of pollution that are lower than in California, the area to which many of the economics studies have referred. In addition, our estimates of the deaths arising from current levels of airborne pollution are considerably higher than those which have been estimated previously using UK data. They are, in fact, closer to those derived from the much less common – and far more expensive – cohort studies, none of which have been undertaken for the UK.

[23] If the short run effect of pollution is to kill the frail, our estimates are an upper bound. We repeat this exercise for deaths in the age group 15 to 64 years old, as these individuals are least frail. We estimate that a 10 $\mu g/m^3$ increase in PM_{10} increases mortality in this age group by 2.3%, so a 4.7 $\mu g/m^3$ drop in PM_{10} evaluated at the mean mortality rate for this age group, 247 per 100,000 population, would result in 2.7 fewer deaths per 100,000 population. The population of 15 to 64 years old is around 32 million, so the drop in mortality translates into 864 fewer deaths per annum. Assuming these individuals gain only 10 years of life – so this estimate will give a lower bound – these 8,640 additional life years are worth £ 260 million.

[24] This benefit figure is one and a half times the size of the £791 million expenditure on protection of ambient air and climate by the UK general government sector (£250 million) and UK industry (around £541 million) in 2004 (http://www.statistics.gov.uk/downloads/theme_environment/EA_Jun08.pdf and http://www.defra.gov.uk/environment/statistics/envsurvey/expn2004/eerp2004.pdf). It is in a similar ballpark to estimates of the annualised cost of fitting all new cars and lorries with devices that reduce emissions (Department of Food, Environment and Rural Affairs, 2007), though it is estimated that this action will decrease PM_{10} by only 0.8 and not the 4.7 needed to reach the new standard.

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Appendix A: Sources of CO, NO_2, PM_{10} and O_3 and their effects on human health

CO is a colourless, odourless, poisonous gas, which reduces the body's ability to use oxygen. CO results from combustion processes under insufficient oxygen supply. Burning fuel containing carbon in idling or slow moving motor vehicles contributes the largest share of CO. A smaller share results from processes involving combustion of organic matter, e.g. power stations and waste incinerators. CO survives in the atmosphere for approximately one month before it oxides to carbon dioxide.

NO_2 is a brown, reactive gas with a detectable smell, which is highly toxic in significant concentrations. Relatively high concentrations of NO_2 cause inflammation of the airways and can produce broncho-constriction in both asthmatics and non-asthmatics (Department of Health, 1997). NO_2 occurs as a primary pollutant (emitted directly from a source) and as a secondary pollutant (formed in the air by reactions of primary pollutants). As a primary pollutant, NO_2 is mainly emitted from the tailpipe of diesel vehicles, especially when they move slowly. As a secondary pollutant, NO_2 is mainly formed by oxidation of nitric oxide, which is produced by burning fuel at high temperatures. Road transport produces the largest share of NO_2. Other important sources of NO_2 are power stations and natural gas space heating (Air Quality Expert Group, 2004). NO_2 converts to nitrates (e.g. nitric acid), which rain or gravity return from the atmosphere to Earth.

Particulate matter has an unspecified chemical composition. Its most important characteristic is the size of the particles. Coarse particles with a diameter of 2.5 to 100 μm consist mainly of soil and sea salt elements and are produced by mechanical processes (e.g. suspension of soil in farming and mining, construction, stone abrasion, and sea spray). Coarse particles settle out quickly by gravity. Fine particles with a diameter of 0.1 to 2.5 μm consist of primary particles that result from combustion processes and secondary particles that are, for instance, formed by condensation of low volatile compounds and ammonia. Fine particles are too small to settle out by gravity and too large to coagulate into larger particles, therefore they can stay in the atmosphere over days to weeks and travel hundreds to thousands of kilometres before rain returns them from the atmosphere to Earth. Ultra-fine particles with a diameter of 0.01 to 0.1 μm have a short residence time in the atmosphere because of their Brownian motion. Particles with a diameter less than 10 μm (PM_{10}) are inhalable, but 60 to 80% of particles with a diameter of 5 to 10 μm are trapped in the nose and pharynx (Wilson and Spengler, 1996). Smaller particles penetrate the trachea and the primary bronchi. Very small particles penetrate deep into the lungs.

O_3 is a bluish, unstable gas with a pungent odour, which is toxic even at low concentrations. It is the "most potent (…) pro-inflammatory pollutant of the common range of air pollutants" (Department of Health, 1997). O_3 is a secondary pollutant that is formed by the action of sunlight on volatile organic compounds in presence of NO_2. It can travel large distances. Nitric oxide, which has high concentrations in urban areas, scavenges O_3, resulting in much higher O_3 levels in rural areas than in urban areas. As the formation of O_3 requires sunlight, O_3 levels are highest in summer.

Appendix B: Current air quality standards

(a) Annual
The annual mean of NO_2 must not exceed 40 $\mu g/m^3$ by 31 December 2005. The annual mean of PM_{10} must not exceed 40 $\mu g/m^3$ by 31 December 2004 and 20 $\mu g/m^3$ by 31 December 2010.

(b) Daily
The daily maximum of the running 8 hour mean of CO must not exceed 10 mg/m^3 by 31 December 2003. The 24 hr mean of PM_{10} must not exceed 50 $\mu g/m^3$ more than 35 times per year by 31 December 2004. The daily maximum of the running 8 hr mean of O_3 must not exceed 100 $\mu g/m^3$ more than 10 times per year by 31 December 2005.

Appendix C: Data sources

(a) Air pollution
Data was downloaded from the web sites of the following networks:

- Automatic Urban and Rural Network (www.airquality.co.uk)
- London Air Quality Network (www.londonair.org.uk)
- Hertfordshire & Bedfordshire Air Pollution Monitoring Network (www.hertsbedsair.org.uk)
- Kent and Medway Air Quality Monitoring Network (www.kentair.org.uk)
- Sussex Air Quality (www.sussex-air.net)
- South Cambridgeshire District Council (http://scambs-airquality.aeat.co.uk)
- Oxford Airwatch (www.oxford-airwatch.aeat.co.uk)
- Newham Council (http://apps.newham.gov.uk/pollution/)
- Air Quality Monitoring in Slough (www.aeat.co.uk/netcen/aqarchive/slough/site_map.html)

We dropped provisional values, keeping only ratified values. Some data came in volume ratios, which we converted into mass units, using the conversion factors used for reporting data to the European Commission:

- CO: 1 ppm = 1.16 mg/m^3
- NO_2: 1 ppb = 1.91 $\mu g/m^3$
- O_3: 1 ppb = 2.00 $\mu g/m^3$

We multiply data on PM_{10} from TEOM analysers by 1.3 and data from BAM analysers by 0.83 to obtain gravimetric equivalent measures.

Annual means of pollutant concentrations at station level are based on at least 100 observations.

(b) Mortality and covariates

Variable	Source	Years covered
Mortality rates (per 100,000)		
Mortality from all causes	Directly age-standardised rates from Clinical and Health Outcomes Knowledge Base (www.nchod.nhs.uk), calculated using data on registered deaths from Office for National Statistics (ONS) and 2001 Census based mid-year population estimates from ONS	1998-2005
Mortality from all circulatory diseases		
Mortality from coronary heart disease		
Mortality from acute myocardial infarction		
Mortality from stroke		
Mortality from bronchitis, emphysema and other chronic obstructive pulmonary diseases		
Mortality from chronic liver disease incl. cirrhosis		
Mortality from infectious and parasitic diseases		
Mortality from lung cancer		
Covariates		
Smoking rate, regional level	Clinical and Health Outcomes Knowledge Base	1998, 2000-2005
Employment rate	Labour Force Survey (www.nomisweb.co.uk)	1998-2005
Percentage of working age people educated to degree level or higher		
Annual mean of summer daily max. temperature	Met Office – MIDAS Land and Surface Station Data	1998-2005
Annual mean of precipitation		

Table A.1: Correlation between annual pollutant concentrations

Correlation	CO	NO_2	PM_{10}	O_3
CO	1			
NO_2	0.6	1		
PM_{10}	0.4	0.6	1	
O_3	-0.3	-0.5	-0.2	1

Figure 1: English local authorities and Government Office regions

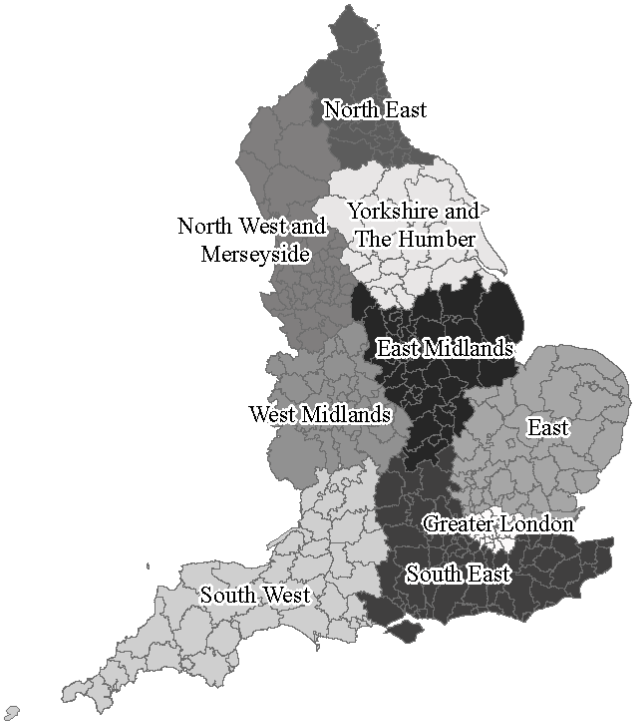


Figure 2: Positions of monitoring stations in England

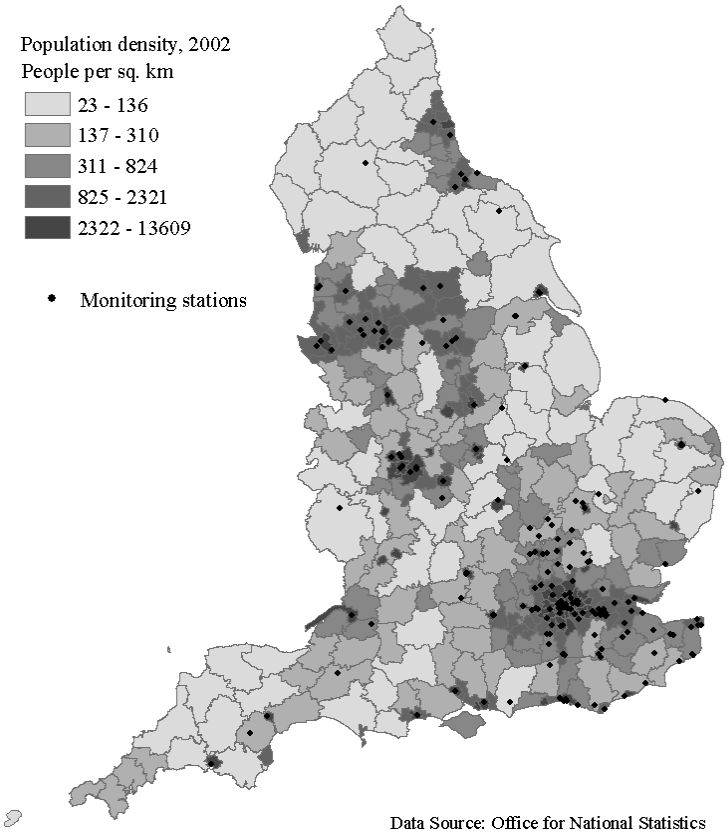
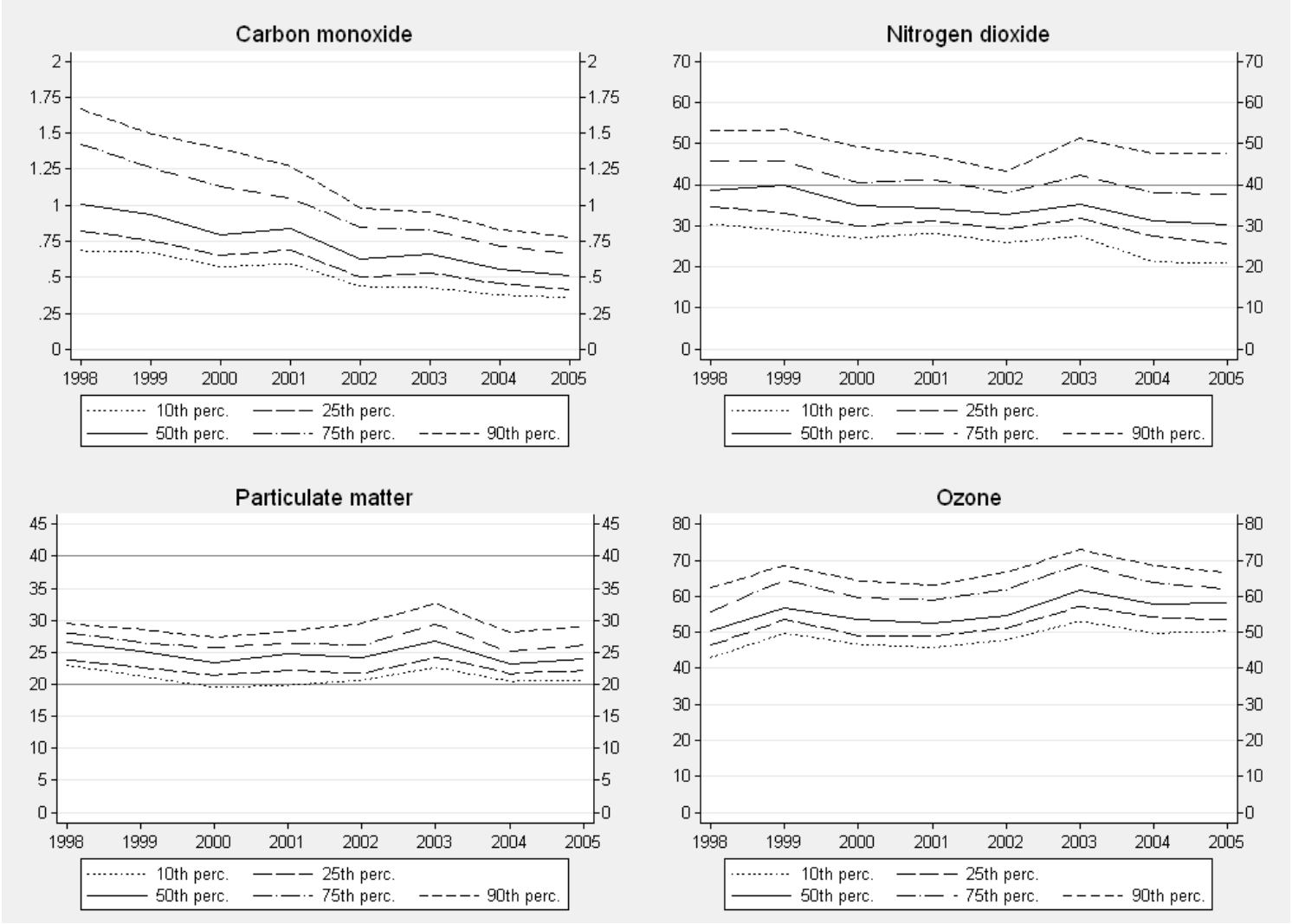
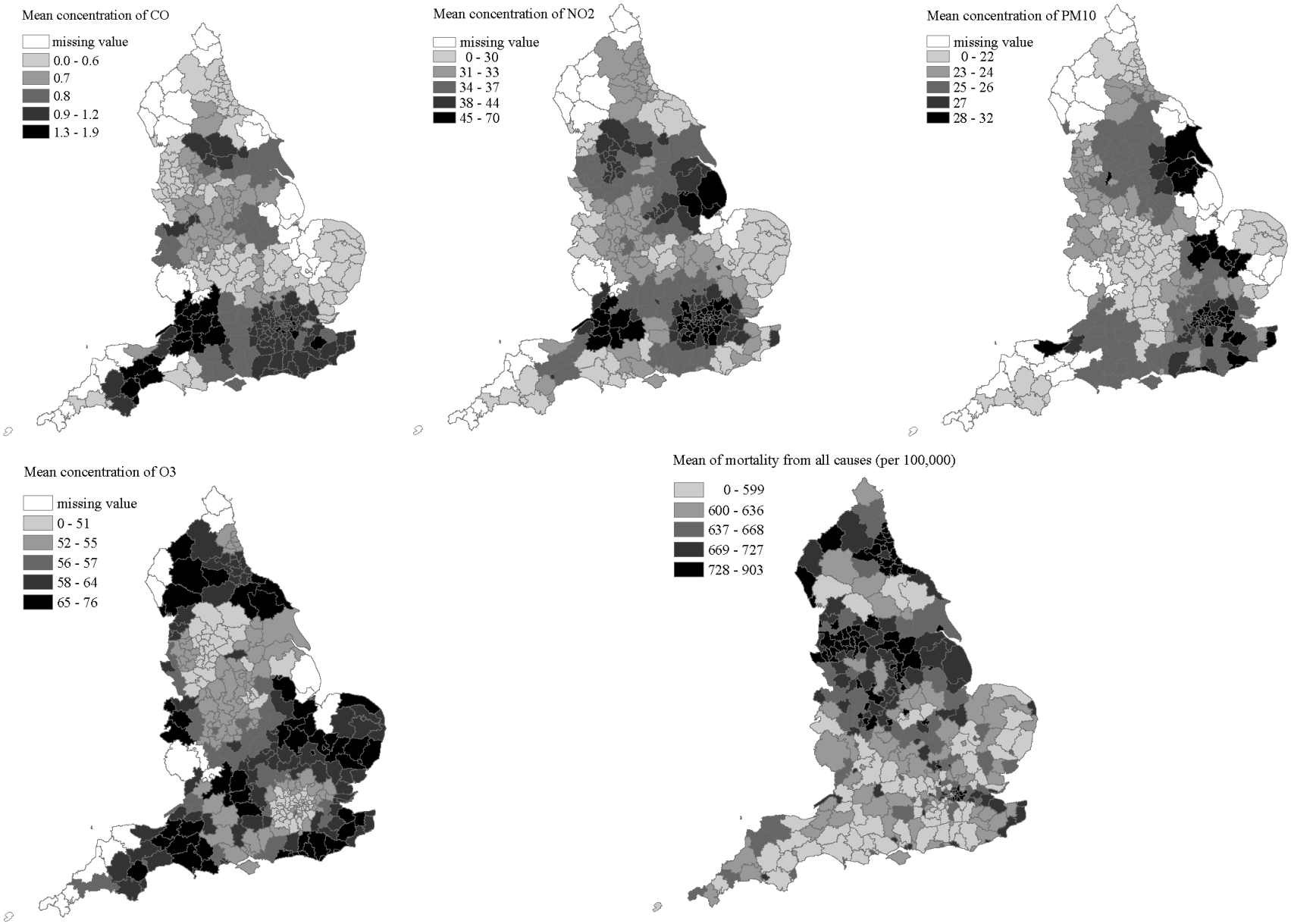


Figure 3: Quantile plots of annual pollutant concentrations in English local authorities



Note: Grey lines indicate annual limit values: the annual mean of NO_2 must not exceed 40 μg/m^3 by 31 December 2005, the annual mean of PM_{10} must not exceed 40 μg/m^3 by 31 December 2004 and 20 μg/m^3 by 31th December 2010.

Figure 4: Cross-sectional distribution of mortality from all causes, CO, NO_2, PM_{10} and O_3



T*able 1:* Descriptive statistics for the estimation sample (n = 2338, groups = 312)

Variable	Mean	Std. dev.	Between local author. std. dev.	Within local auth. std. dev.	Mean in 1998	Mean in 2005
Pollutants						
CO (mg/m^3)	0.80	0.34	0.26	0.23	1.13	0.55
NO_2 ($\mu g/m^3$)	36.6	9.1	8.5	3.8	41.0	33.2
PM_{10} ($\mu g/m^3$)	24.7	3.3	2.9	1.7	26.3	24.2
O_3 ($\mu g/m^3$)	55.9	7.5	6.6	4.1	49.9	57.5
Mortality rates						
Mortality from all causes (per 100,000)	660.1	86.2	78.2	39.9	712.7	605.0
Mortality from all circulatory diseases (per 100,000)	243.7	42.0	31.6	28.4	286.3	202.2
Mortality from coronary heart disease (per 100,000)	124.9	28.6	22.1	18.5	153.0	99.7
Mortality from acute myocardial infarction (per 100,000)	54.8	17.1	13.1	11.3	71.3	41.1
Mortality from stroke (per 100,000)	63.7	11.9	8.2	8.7	73.2	53.0
Mortality from bronchitis, emphysema and other COPD (per 100,000)	29.2	9.8	8.8	4.6	31.5	27.3
Control variables						
Smoking rate (%)	25.7	2.4	1.7	1.7	27.4	23.6
Employment rate (%)	76.0	6.3	5.8	2.4	75.6	76.2
NVQ 4+ level rate (%)	24.4	7.9	7.6	3.2	22.2	26.6
Annual mean of summer daily maximum temperature (deg C)	18.6	1.2	1.0	0.6	17.8	18.7
Annual mean of precipitation (mm)	2.2	0.63	0.51	0.38	2.4	1.7
Other mortality rates for robustness tests						
Mortality from chronic liver disease including cirrhosis (per 100,000)	8.9	4.4	3.5	2.7	7.8	9.6
Mortality from infectious and parasitic diseases (per 100,000)	5.7	2.9	2.0	2.1	5.0	7.2

Table 2: Means of pollutants and all cause mortality by tertiles of pollutant distributions for the estimation sample (n = 2338)

Ranked by	Variable	Lowest 1/3	Middle 1/3	Highest 1/3
CO	CO (mg/m^3)	0.5	0.7	1.2
	Mortality from all causes (per 100,000)	656.9	662.5	661.0
NO_2	NO_2 ($\mu g/m^3$)	27.5	35.1	47.3
	Mortality from all causes (per 100,000)	649.3	670.1	661.0
PM_{10}	PM_{10} ($\mu g/m^3$)	21.2	24.5	28.4
	Mortality from all causes (per 100,000)	662.4	654.1	663.9
O_3	O_3 ($\mu g/m^3$)	48.1	55.2	64.4
	Mortality from all causes (per 100,000)	688.6	656.8	634.7

Table 3: Estimates of the association between air pollutant concentrations and all cause mortality rates in single-pollutant models

ln(all cause mort.) * 100	OLS	OLS	WG	Long diff.	OLS	WG	Long diff.
		Controlling for trend, region and regional trend			Controlling for trend, region, regional trend, smoking rate, employment rate, degree level qualification rate, temperature and precipitation		
CO	0.39	1.14	-0.13	-0.06	3.40*	-0.42	-0.90
	(1.58)	(2.28)	(0.61)	(0.75)	(1.74)	(0.65)	(0.77)
R^2	0.00	0.49	0.94	0.02	0.71	0.95	0.10
NO_2 / 10	0.43	1.58**	1.36***	1.40***	2.16***	0.42	0.21
	(0.76)	(0.75)	(0.24)	(0.24)	(0.49)	(0.27)	(0.25)
R^2	0.00	0.49	0.94	0.04	0.71	0.95	0.09
PM_{10} / 10	1.15	6.84***	4.07***	4.22***	3.96***	2.80***	2.38***
	(1.93)	(1.50)	(0.47)	(0.51)	(1.27)	(0.51)	(0.60)
R^2	0.00	0.50	0.94	0.07	0.71	0.95	0.11
O_3 / 10	-5.04***	-0.86	1.92***	1.57***	-1.47**	0.73**	0.12
	(0.85)	(0.85)	(0.23)	(0.26)	(0.61)	(0.29)	(0.33)
R^2	0.08	0.49	0.94	0.05	0.71	0.95	0.09
Observations	2338	2338	2338	1404	2338	2338	1404
Groups	312	312	312	301	312	312	301

Note: WG = within groups. Coefficients are percentage changes in all cause mortality rate per 1 mg/m^3 increase in CO and per 10 $\mu g/m^3$ increase in NO_2, PM_{10} and O_3. Observations weighted by square root of mid-year population estimates. Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 4: Estimates of the association between air pollutant concentrations and all cause mortality rates in multi-pollutant models

ln(all cause mort.) * 100	OLS	OLS	WG	Long diff.	OLS	WG	Long diff.
		Controlling for trend, region and regional trend			Controlling for trend, region, regional trend, smoking rate, employment rate, degree level qualification rate, temperature and precipitation		
CO	0.45	-2.27	-0.39	-0.79	0.27	-0.34	-0.94
	(1.63)	(2.30)	(0.72)	(0.80)	(1.68)	(0.69)	(0.78)
NO_2 / 10	-2.82***	0.49	0.64**	0.64***	1.50**	0.34	0.17
	(0.94)	(0.75)	(0.27)	(0.24)	(0.62)	(0.28)	(0.26)
PM_{10} / 10	1.85	6.72***	3.03***	3.36***	2.33*	2.74***	2.37***
	(1.92)	(1.55)	(0.47)	(0.52)	(1.37)	(0.51)	(0.59)
O_3 / 10	-6.60***	-0.59	1.57***	1.06***	-0.55	0.80***	0.18
	(0.80)	(0.80)	(0.24)	(0.27)	(0.66)	(0.29)	(0.34)
Smoking rate					0.18*	0.07	0.20
					(0.10)	(0.08)	(0.13)
Employm. rate					-0.80***	0.00	0.05
					(0.06)	(0.04)	(0.04)
Degree qua-lification rate					-0.46***	-0.06*	-0.04
					(0.07)	(0.03)	(0.03)
Summer tem-perature					0.96**	0.90***	0.94***
					(0.39)	(0.20)	(0.23)
Precipitation					1.30**	0.46	-0.12
					(0.54)	(0.35)	(0.36)
R^2	0.10	0.50	0.95	0.09	0.71	0.95	0.11
Observations	2338	2338	2338	1404	2338	2338	1404
Groups	312	312	312	301	312	312	301

Note: WG = within groups. Coefficients are percentage changes in all cause mortality rate per 1 mg/m^3 increase in CO and per 10 $\mu g/m^3$ increase in NO_2, PM_{10} and O_3. Observations weighted by square root of mid-year population estimates. Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 5: Within-group estimates of the association between air pollutant concentrations and a range of mortality rates in a multi-pollutant model

	ln(all cause mortality) * 100	ln(mortality from all circulatory diseases) * 100	ln(mort. from coronary heart disease) * 100	ln(mortality from acute myocardial infarction) * 100	ln(mortality from stroke) * 100	ln(mort. fr. bronchitis, emphysema and other COPD) * 100
CO	-0.34	-0.02	1.18	-1.51	-3.18	-3.82
	(0.69)	(1.53)	(1.27)	(2.36)	(4.04)	(3.51)
NO_2 / 10	0.34	0.34	0.27	-1.91	0.82	1.91
	(0.28)	(0.49)	(0.65)	(1.27)	(0.91)	(1.27)
PM_{10} / 10	2.74***	4.38***	4.90***	5.03**	4.08**	1.80
	(0.51)	(0.78)	(1.05)	(2.09)	(1.78)	(2.49)
O_3 / 10	0.80***	-0.01	-0.39	-1.37	0.02	2.40*
	(0.29)	(0.54)	(0.69)	(1.18)	(0.90)	(1.23)
R^2	0.95	0.92	0.91	0.86	0.76	0.83
Obs.	2338	2338	2338	2338	2338	2338
Groups	312	312	312	312	312	312

Note: COPD = chronic obstructive pulmonary diseases. Coefficients are percentage changes in the mortality rate per 1 mg/m^3 increase in CO and per 10 $\mu g/m^3$ increase in NO_2, PM_{10} and O_3. Controls are trend, region-specific trends, smoking rate, employment rate, degree-level qualification rate, annual mean of daily maximum temperature in summer and annual mean of precipitation. Observations weighted by square root of mid-year population estimates. Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 6: Within-group estimates of the association between air pollutant concentrations and all cause mortality for different age groups in a multi-pollutant model

	All ages	< 15	15 to 64	65 to 74	> 75
CO	-1.02*	5.08	2.65**	-1.05	-1.60*
	(0.56)	(5.57)	(1.18)	(1.13)	(0.85)
NO_2 / 10	0.72**	-1.77	-0.25	0.88	0.82**
	(0.29)	(2.68)	(0.61)	(0.58)	(0.37)
PM_{10} / 10	2.46***	9.30**	2.27**	1.63	3.14***
	(0.53)	(4.57)	(1.05)	(1.00)	(0.64)
O_3 / 10	0.69**	2.41	-0.12	0.51	0.90***
	(0.29)	(2.34)	(0.60)	(0.54)	(0.32)
R^2	0.97	0.52	0.88	0.91	0.80
Observations	2338	2331	2338	2338	2338
Groups	312	312	312	312	312

Note: Coefficients are percentage changes in the mortality rate per 1 mg/m^3 increase in CO and per 10 $\mu g/m^3$ increase in NO_2, PM_{10} and O_3. Mortality rates are not age-standardised. Controls are proportions of age groups (5 year age bands), trend, region-specific trends, smoking rate, employment rate, degree-level qualification rate, annual mean of daily maximum temperature in summer and annual mean of precipitation. Observations weighted by square root of mid-year population estimates for respective age group. Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 7: Robustness tests in a multi-pollutant fixed effects model for all cause mortality

		CO		NO_2 / 10		PM_{10} / 10		O_3 / 10		Coefficient on additional control variable		Obs.	Groups
1	Baseline	-0.34	(0.69)	0.34	(0.28)	2.74***	(0.51)	0.80***	(0.29)			2338	312
2	Monitoring stations within 20 mile/5 mile radius	-0.36	(0.62)	0.16	(0.31)	2.20***	(0.50)	0.76**	(0.31)			1933	267
3	Drop kerbside and roadside monitoring stations	-2.37**	(1.19)	0.50	(0.39)	2.16***	(0.53)	0.77**	(0.34)			1778	243
4	Drop observations for 2003	-0.29	(0.73)	0.28	(0.30)	1.88***	(0.53)	0.51	(0.33)			2037	312
5	Drop obs. in top 10% of pollutant distributions	-1.02	(1.01)	0.41	(0.38)	2.26***	(0.75)	0.63*	(0.35)			1789	288
6	Drop observations in South West	-0.73	(0.82)	0.28	(0.34)	2.58***	(0.52)	0.93***	(0.33)			2132	283
7	Drop observations in London	-0.47	(0.83)	0.65**	(0.31)	2.22***	(0.51)	0.63**	(0.29)			2081	279
8	Include lagged pollutants	-0.37	(0.98)	0.67**	(0.30)	1.90***	(0.68)	0.72**	(0.31)			2043	312
9	Include annual max. of weekly pollutant levels	0.09	(0.77)	1.01***	(0.33)	1.77***	(0.61)	0.70**	(0.34)			2338	312
	Coeff. on ann. max. of weekly poll.:	0.03	(0.14)	-0.52***	(0.2)	0.36**	(0.15)	-0.02	(0.06)				
10	Dep. var.: ln(mort. fr. chronic liver disease) * 100	-4.42	(5.87)	-0.36	(2.73)	0.10	(5.09)	4.63*	(2.80)			2331	312
11	Dep. var.: ln(mort. fr. infectious diseases) * 100	5.20	(6.71)	-2.99	(3.24)	-2.41	(6.60)	1.73	(3.15)			2325	312
12	Include population size / 1000	-0.30	(0.69)	0.38	(0.29)	2.96***	(0.51)	0.83***	(0.29)	-0.12**	(0.06)	2338	312

Note: Coefficients are percentage changes in all cause mortality rate per 1 mg/m^3 increase in CO and per 10 $\mu g/m^3$ increase in NO_2, PM_{10} and O_3. Observations weighted by square root of mid-year population estimates. Baseline specification includes time trend, region specific time trends, smoking rate, employment rate, degree-level qualification rate, annual mean of daily maximum temperature in summer and annual mean of precipitation. Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%

Table 8: Within-group estimates of the association between air pollutant concentrations and all cause mortality using a spline with 2 knots at the 33rd and the 66th percentile

Pollutant	Lowest tertile	Middle tertile	Highest tertile
PM_{10}	2.24** (1.05)	3.48*** (1.13)	2.69*** (0.69)
O_3	0.97** (0.49)	0.88 (0.54)	0.62 (0.45)

Note: Coefficients are percentage changes in the mortality rate per 10 $\mu g/m^3$ increase in pollutant concentration. Controls are CO, NO_2, trend, region-specific trends, smoking rate, employment rate, degree-level qualification rate, annual mean of daily maximum temperature in summer and annual mean of precipitation. Observations weighted by square root of mid-year population estimates. Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.