Are current levels of air pollution in England too high? The impact of pollution on population mortality

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Editorial Note

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Abstract

We examine the relationship between common sources of airborne pollution and population mortality in present day England. The current air quality limit values are low by both historical and international standards, and these are set at levels which are believed not to be harmful to health. We assess whether this view is correct. We use data at local authority level for the period 1998 to 2004 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. We exploit the panel nature of our data to control for any unobserved time-invariant associations at local authority level between high levels of pollution and poor population health and estimate multi-pollutant models to allow for the fact that three of the pollutants are closely correlated. We find higher levels of PM_{10} and ozone are associated with higher mortality rates. The size of the effects we find translates into around 4,500 deaths per annum.

Key words: airborne pollutants, adult mortality, geographical analysis
JEL classification: I12, I18

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1. Introduction

The current levels of airborne pollutants in many OECD countries are low by historical standards. The limits on 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 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 epidemiological 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 pollution and deaths to examine the time series relationship. Such studies measure the acute effects of pollution and generally focus on a single source of pollution. However, the focus on a single source of pollution may over-estimate the impact of this pollutant, as several of the common airborne pollutants are correlated, because they are closely related to 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 very likely that these cities may be 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 relationship between annual mortality rates and annual mean concentrations of four common sources of air pollution 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. We are also able to control for the correlation between the levels of common airborne pollutants.

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

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 source of pollution on adult mortality. However, as they note, the airborne 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 2004. 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. 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. We examine deaths from all causes and specific deaths - of the cardiovascular and respiratory system - that are most likely to be caused by exposure to airborne pollutants (Pope and Dockerty 2006). We control for observed factors that may be correlated with pollution but are independent causes of early deaths, such as income and lifestyle. We estimate multiple pollutant models to isolate the impact of specific pollutants. We subject our results to a large number of specification tests, including a 'placebo' test for a spurious association between pollution at local authority level and death rates by examining the association of pollution with a cause of death which is unlikely to be driven by pollution, suicide.

Our findings suggest that the levels of pollution currently permitted in the UK are associated with higher mortality rates in the population.[2] We find significant effects of levels of both particulate matter less than 10μm in diameter (PM_{10}) and ozone on mortality. The magnitudes of these effects are both statistically and economically significant. They are also much closer to those derived from the few studies of the impact of life time exposure to pollution (none of which have been estimated using UK data) than those from the much more common approach (widely used in the UK) of studying the impact of daily variation in pollution levels.

2. Background

(a) Air pollutants and their effects on human health

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). These four pollutants as well as sulphur dioxide, lead, and the carcinogens benzene and 1,3-butadiene are "Air Quality Strategy Pollutants" for which European legislation sets limit values.[3]

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2 The vast majority of deaths are accounted for by adults: in 2004, for example, children made up less than 1.1% of the total deaths and only 0.09% of deaths from circulatory diseases.

3 Sulphur dioxide used to be a major component of the air pollution mix until the 1950s but

CO is a colourless, odourless, poisonous gas. It reduces the body's ability to use oxygen, because it bonds with haemoglobin more easily than 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. It 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. Particles cause inflammation of the airways, and they may alter the circulation of red blood cells and platelets (Air Quality Expert Group, 2005).

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since the Clean Air Act 1956 it is a less significant pollutant. Urban lead levels have been substantially reduced since the introduction of unleaded petrol and industry plants are now the main source of lead. Benzene and 1,3-butadiene levels are measured by only a few monitoring stations, which precludes inclusion in our analysis. Air quality standards in operation in the UK during our data period are given in Appendix A.

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.

In our analyses, we follow the literature and use measurements taken by stationary monitors at outside locations to calculate exposure to air pollution. As people spend over 80% of their time indoors, there is an issue of the extent to which measures of ambient air pollution predict personal exposure. 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 pollutants and personal exposure to air pollutants are significantly correlated.[4] 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.[5]

(b) An overview of the literature on air pollution and mortality

The literature on air pollution and mortality is dominated 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

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4 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.

5 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.

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 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.

Cohort studies use pollutant concentrations averaged over a year or longer periods. They control for confounding factors by using individual-level data on lifestyle, susceptibility, socio-economic status, occupational exposure etc. 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).[6] Because of their design, cohort studies are expensive and take long time to complete. In addition, the cohort studies may also 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 pollution.

Another important strand of the literature are ecological studies of associations between spatial variations in air pollution and spatial variations in mortality. These studies compare mortality in 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). 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.[7]

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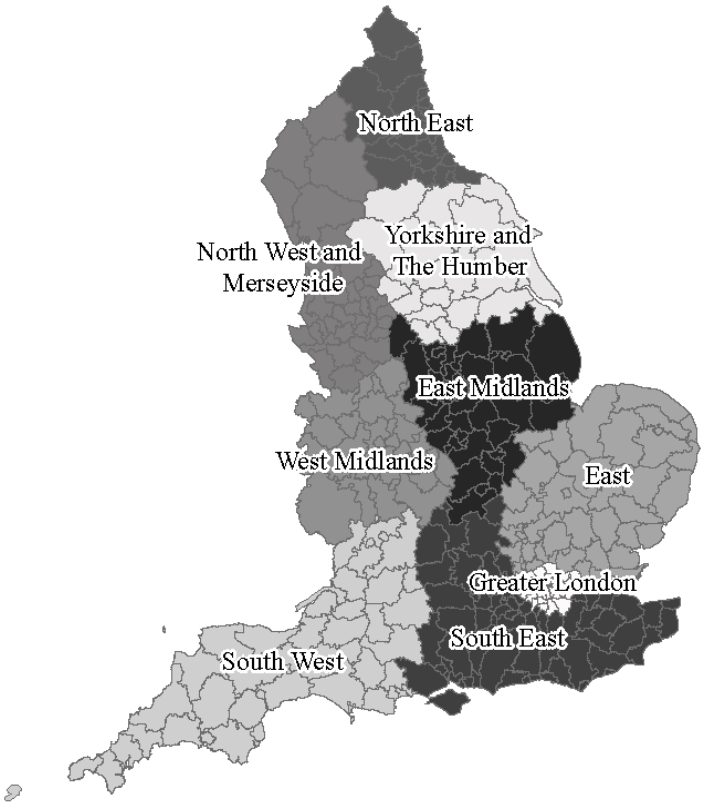
6 An alternative measure of exposure to air pollution, living near a major road, has a significant effect on cardiopulmonary mortality.

7 A very small number of studies use exogenous changes in air pollution. For example, Clancy et al. (2002) used the ban on coal sales in Dublin in 1990 which reduced average black smoke concentrations. Chay and Greenstone (2003) use a sudden recession as an instrument to identify the effect of a medium-term reduction of pollution on infant mortality. 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).

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.[8] Local authorities are aggregated into 9 Government Office regions. Figure 1 shows the location and size of local authorities and the Government Office regions.

Figure 1: English local authorities and Government Office regions



We estimate equations of the following form:

$$(1) \qquad M^{j}_{it} = \alpha + P'_{it}\gamma_j + Z'_{it}\beta_j + T^{j} + T_r^{j} + \mu^{j}_{i} + \varepsilon^{j}_{it}$$

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 five mortality rates (all cause; all circulatory diseases; coronary heart disease; acute myocardial infarction; bronchitis, emphysema and other chronic obstructive pulmonary diseases), P_{it} are the logarithms of a set of pollutants (CO, NO_2, PM_{10}, O_3), Z_{it} are the logarithms of a set of time-varying controls at local authority (or regional) level. T^{j} is a time trend, T_r^{j} is a regional specific time

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

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 pollution at local authority level. In a relatively short panel in which the outcomes and the level of pollution are both strongly trended, the time effects may be collinear with pollution effects, making identification of the latter difficult; but we show below that there is within local authority variation to be explained. As our panel is short, within group estimates may be biased, so we assess the robustness of these by also estimating 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

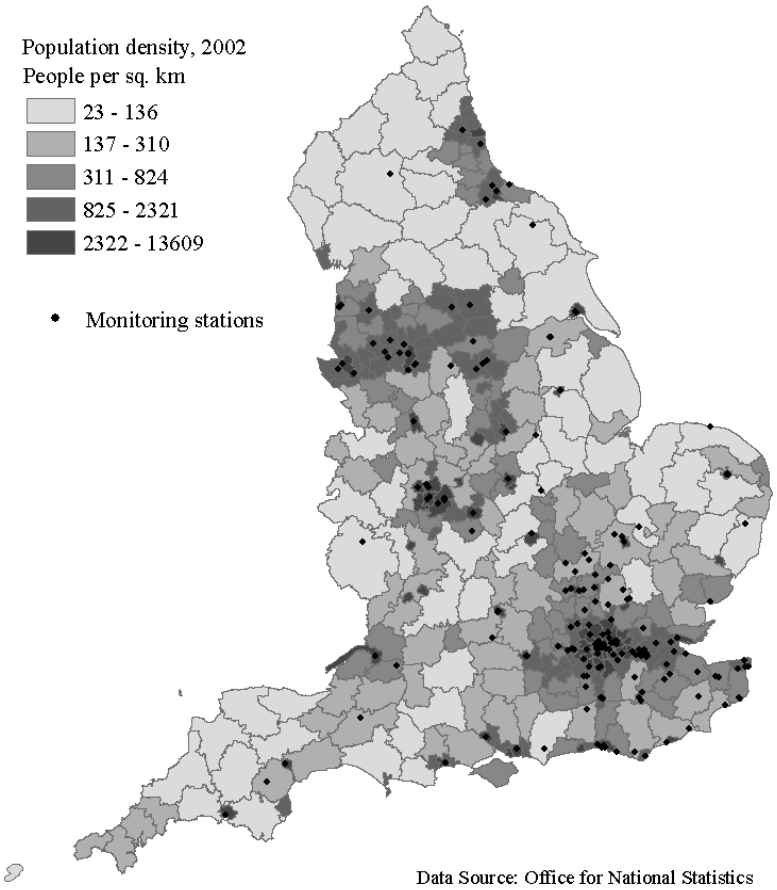
Data on air pollution comes from the UK Air Quality Archive,[9] supplemented with data from four regional air quality networks managed by the same operator and from another four regional networks managed by the Environmental Research Group at King's College London. These sources provide data on a total of 190 automatic monitoring stations, of which 87, 165, 108 and 99 record concentrations of CO, NO_2, PM_{10} and O_3, respectively. Figure 2 shows the positions of these monitors. 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 B). 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 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

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

the distance-weighted mean of the annual mean pollutant concentrations at monitors in a 30 (10) miles 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 319, 331, 319 and 335 out of 354 local authorities, respectively. The local authorities with missing air pollution measures are all less populated areas.

Figure 2: Positions of monitoring stations in England



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.[10] 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.[11] Since

[10] Figure A.1 shows the weekly CO series: comparing the weekly and the annual, the effect of annual averaging is clearly to remove seasonal patterns and to reduce variation.

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

our identification strategy relies on time series variation within local authorities, the accuracy of our pollution measure seems reasonable.

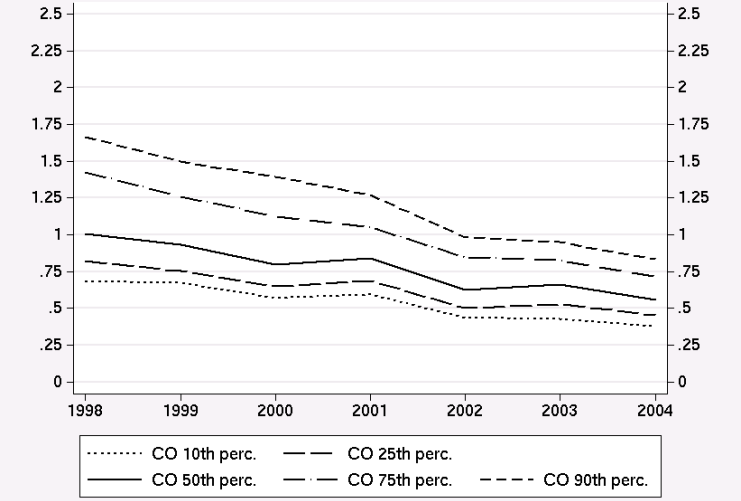
Figure 3 shows the time series variation in annual pollution at local authority level. CO clearly declines over the years of our sample. There is also a reduction in the variation: the distance between the top two quantiles and the other three quantiles of the distribution falls over time. Measured at 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 annual mean standard 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 when this fall levels off before a marked rise in 2003. The distribution is pretty constant over the period. PM_{10} does not exceed the annual mean standard of 40 g/m^3 that was in force towards the end of the period we examine, but it does exceed the standard of 20 $\mu g/m^3$, 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 in 2003. Both these years are years of above average sunshine, illustrating the potential difficulty of isolating the impact of O_3 from that of weather conditions.

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 pollution effects by exploiting within-local authority variations. Table A.1 presents the correlation between the pollutants and shows that CO, NO_2 and PM_{10} are closely correlated, whereas O3, which tends to be higher in rural areas, is negatively correlated with the other three pollutants.

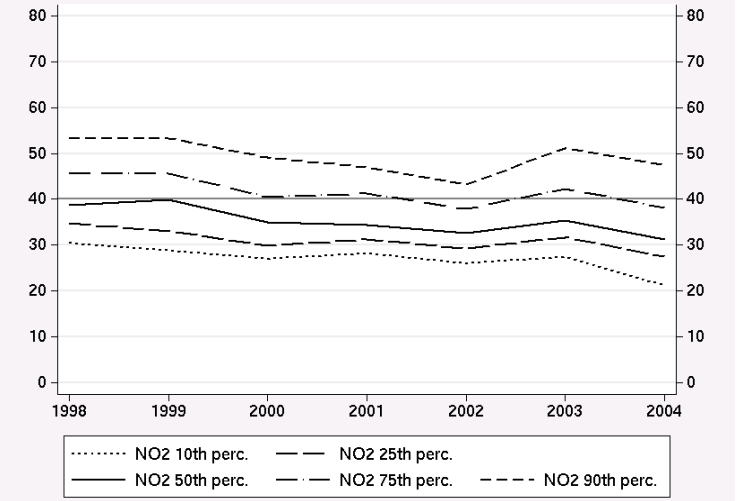
The second panel of Table 1 presents the mortality rates. Sources are given in Appendix B. We focus on deaths from causes that have been shown to be linked to levels of pollution in the medical literature. All cause mortality encompasses all deaths. Mortality from all circulatory diseases comprises the ICD-10 categories I00 to I99. Mortality from coronary heart disease is a subset of mortality from all circulatory diseases (all deaths in the ICD-10 categories I20 to I25). Mortality from acute myocardial infarction, in turn, is a subset of mortality from coronary heart disease, containing the categories I21 to I22. 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.

Figure 3: Annual pollutant concentrations at local authority level in µg/m^3

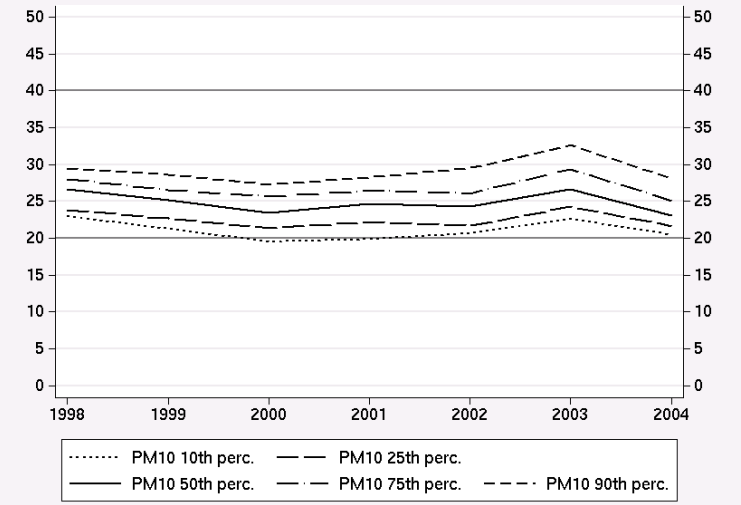
(a) CO



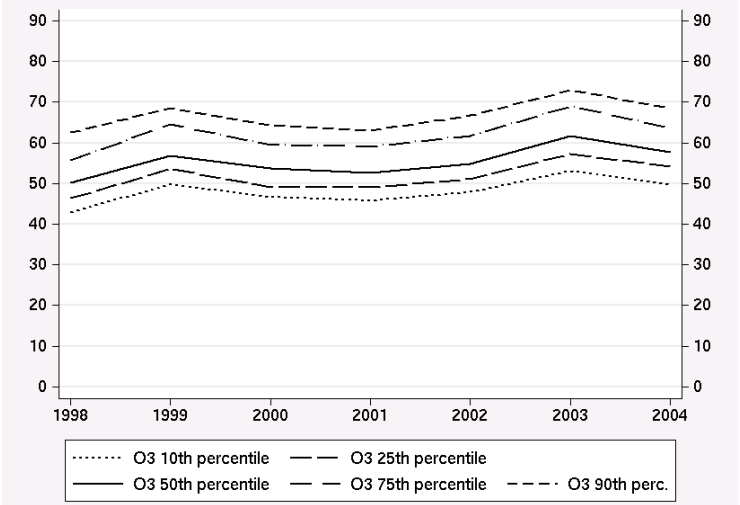
(b) NO_2



(c) PM_{10}



(d) Ozone



Notes: Grey lines indicate air quality standards defined on annual means: the annual mean of NO_2 must not exceed 40 µg/m^3 by 31th December 2005, the annual mean of PM_{10} must not exceed 40 g/m^3 by 31th December 2004 and 20 µg/m^3 by 31th December 2010.

Table 1: Descriptive statistics for the estimation sample (2038 observations, 309 groups)

Variable	Mean	Std.dev.	Between local authorities std. dev.	Within local authorities std. dev.	Mean in 1998	Mean in 2004
Pollutants						
CO (mg/m^3)	0.84	0.34	0.27	0.22	1.13	0.59
NO2 ($\mu g/m^3$)	37.1	8.8	8.2	3.7	41.0	33.5
PM10 ($\mu g/m^3$)	24.8	3.3	2.8	1.7	26.3	23.4
O3 ($\mu g/m^3$)	55.7	7.6	6.8	4.3	49.8	58.0
Mortality rates						
Mortality from all causes (per 100,000)	668.3	84.5	77.4	35.7	712.7	619.9
Mortality from all circulatory diseases (per 100,000)	249.8	40.3	32.0	25.0	286.3	215.3
Mortality from coronary heart disease (per 100,000)	128.6	27.9	22.4	16.7	153.0	107.7
Mortality from acute myocardial infarction (per 100,000)	56.8	16.8	13.4	10.4	71.3	45.4
Mortality from bronchitis, emphysema and other COPD (per 100,000)	29.5	9.8	8.8	4.5	31.5	26.5
Control variables						
Smoking rate (%)	26.1	2.3	1.8	1.5	27.4	24.6
Employment rate (%)	76.3	6.5	5.9	2.7	75.6	76.4
NVQ 4+ level rate (%)	24.0	7.7	7.1	3.0	22.2	25.9
Annual mean of summer daily maximum temperature (deg C)	18.5	1.2	1.0	0.7	17.8	18.8
Annual mean of precipitation (mm)	2.3	0.63	0.51	0.35	2.4	2.2
Additional controls for robustness tests						
Household food energy derived from saturated fatty acids (%)	14.7	0.37	0.35	0.12	14.7	14.8 (2003)
Smoking rate on Health Authority level (%)	24.8	5.9	3.3	4.9	26.8	23.7 (2003)
Mortality from lung cancer (per 100,000)	39.4	11.5	10.4	5.0	42.4	37.3
Other mortality rate for robustness tests						
Mortality from suicide (per 100,000)	6.2	2.9	1.7	2.4	6.6	6.0

The five mortality rates fell over the period. Many factors are likely to cause this fall, including – for heart and respiratory diseases – 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. The time series for the five mortality rates (available from the authors) have a strong downward trend which is very similar for the three cardiovascular mortality rates. 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 2004 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. 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, controlling for weather may dilute the effects of air pollutants if certain weather conditions cause elevated pollutant levels without having an independent effect on mortality.

To control for weather conditions, 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.

The third panel in Table 1 presents descriptive statistics for the controls. Mean smoking rates fell from 27.4% in 1998 to 24.6% in 2004, possibly reflecting the government's efforts to reduce smoking prevalence (Department of Health, 1998).

Mean employment rates and mean NVQ 4+ level rates increased between 1998 and 2004. Mean temperatures have increased during the sample period, with peaks in 1999 and 2003. Precipitation seems to have fallen, but 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 NO_2 and PM_{10} are associated with higher mortality rates. For example, the mean mortality rate for observations in the highest third of the NO_2 distribution is 1.5% 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 CO and O_3 are associated with lower death rates.

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

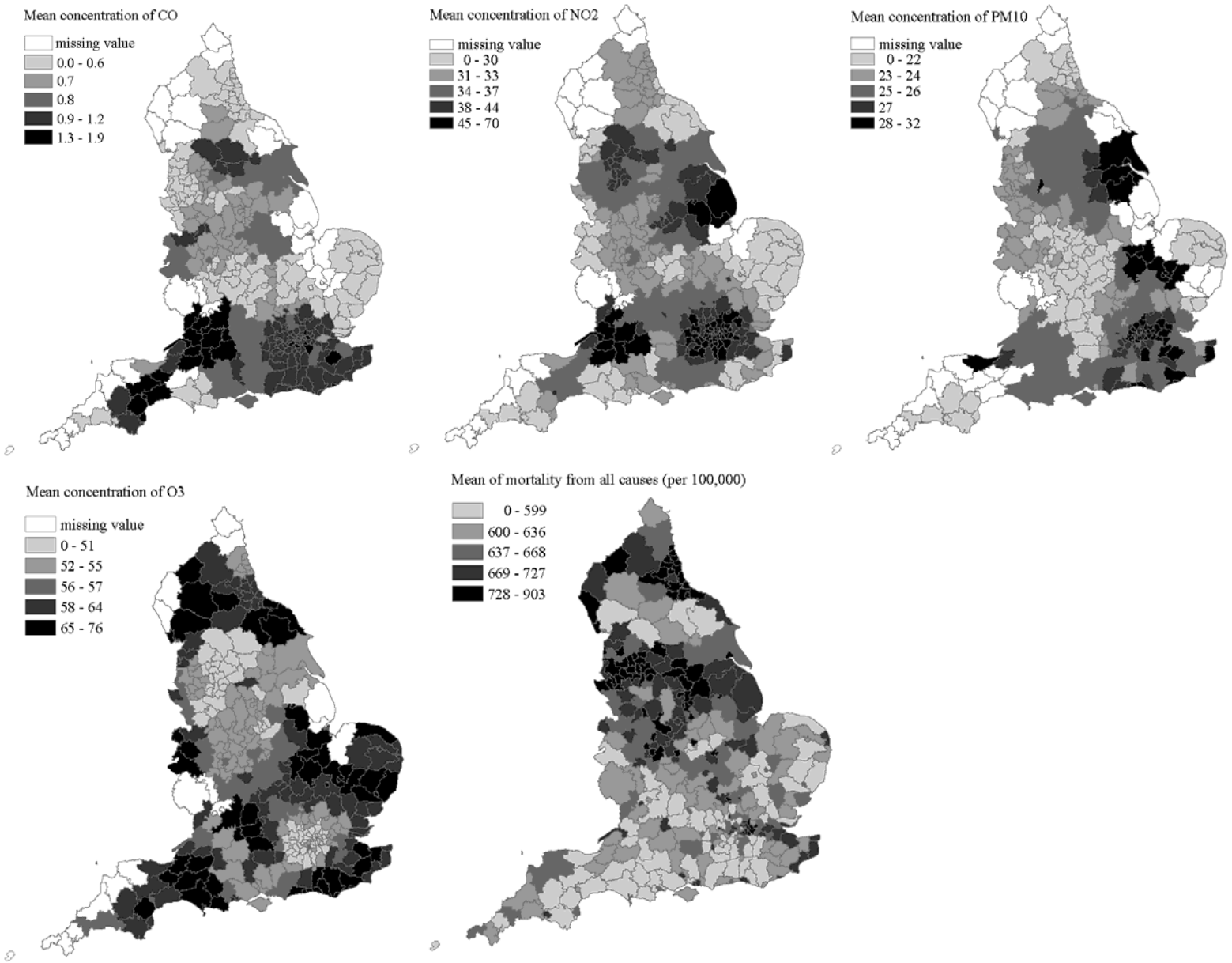


Table 2: Means of pollutants and all cause mortality by tertiles of pollutant distributions for the estimation sample (2038 observations)

Ranked by	Variable	Lowest 1/3	Middle 1/3	Highest 1/3
CO	CO (mg/m^3)	0.5	0.8	1.2
	Mortality from all causes (per 100,000)	669.5	671.7	663.6
NO_2	NO_2 ($\mu g/m^3$)	28.3	35.6	47.3
	Mortality from all causes (per 100,000)	660.2	674.5	670.1
PM_{10}	PM_{10} ($\mu g/m^3$)	21.3	24.6	28.4
	Mortality from all causes (per 100,000)	668.8	663.4	672.5
O_3	O_3 ($\mu g/m^3$)	47.8	54.9	64.4
	Mortality from all causes (per 100,000)	694.3	666.7	643.7

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

We begin by examining the separate association between each pollutant and all cause mortality. The first column of Table 3 presents the raw correlation, estimated by the OLS regression of the log of all cause mortality on a constant and the log of each pollutant. We then control for trend, region and region-specific trends and present OLS, within group and long difference estimates. We then add time-varying controls for lifestyle differences between local authorities, using smoking, employment and education as our covariates.

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 and lifestyle, but this is not robust to inclusion of local authority fixed effects. The second block shows the results for NO_2. The raw association is positive but not significant, but the association is significantly positive after controlling for the covariates. Higher levels of NO_2 are associated with higher mortality rates and the within group and the long difference estimates are very similar. The estimates in the final two columns show that a 1% increase in NO_2 is associated with just under a 0.04% increase in all cause mortality.

The third block shows the results for PM_{10}. These show the same pattern as for NO_2: a positive but insignificant raw association and significantly positive coefficients for all other specifications. Again, the within group and long difference estimates are very similar. The final two columns show that a 1% increase in PM_{10} is associated with a 0.1% increase in all cause mortality, so the point estimate of the effect of PM_{10} is over twice the size of that of NO_2.

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

	OLS	**OLS**	**WG**	**Long diff.**	**OLS**	**WG**	**Long diff.**
ln(all cause mortality)		Controlling for trend, region and regional trends			Controlling for trend, region, regional trends, smoking rate, employment rate and NVQ 4+ level rate		
ln(CO)	**-0.015**	**0.014**	**0.000**	**0.009**	**0.025***	**-0.003**	**0.006**
	(0.014)	(0.019)	(0.005)	(0.006)	(0.013)	(0.005)	(0.006)
ln(smoking rate)					0.111***	0.103***	0.183***
					(0.028)	(0.022)	(0.045)
ln(employment rate)					-0.552***	-0.025	0.007
					(0.046)	(0.026)	(0.029)
ln(NVQ 4+ level rate)					-0.095***	-0.007	-0.008
					(0.014)	(0.007)	(0.008)
R-squared	0.00	0.47	0.94	0.04	0.68	0.94	0.06
Observations	2092	2092	2092	1151	2092	2092	1151
Groups	317	317	317	303	317	317	303
ln(NO_2)	**0.040**	**0.051****	**0.039*****	**0.040*****	**0.067*****	**0.037*****	**0.035*****
	(0.024)	(0.020)	(0.007)	(0.008)	(0.014)	(0.007)	(0.009)
ln(smoking rate)					0.111***	0.095***	0.169***
					(0.026)	(0.022)	(0.044)
ln(employment rate)					-0.539***	-0.023	0.009
					(0.045)	(0.026)	(0.028)
ln(NVQ 4+ level rate)					-0.101***	-0.004	-0.005
					(0.013)	(0.007)	(0.008)
R-squared	0.01	0.48	0.94	0.06	0.69	0.94	0.07
Observations	2153	2153	2153	1183	2153	2153	1183
Groups	328	328	328	313	328	328	313
ln(PM_{10})	**0.027**	**0.168*****	**0.108*****	**0.107*****	**0.089*****	**0.104*****	**0.098*****
	(0.046)	(0.036)	(0.011)	(0.012)	(0.027)	(0.011)	(0.011)
ln(smoking rate)					0.103***	0.076***	0.143***
					(0.026)	(0.021)	(0.039)
ln(employment rate)					-0.533***	-0.002	0.023
					(0.045)	(0.026)	(0.028)
ln(NVQ 4+ level rate)					-0.096***	-0.006	-0.007
					(0.013)	(0.007)	(0.008)
R-squared	0.00	0.48	0.94	0.10	0.68	0.94	0.11
Observations	2088	2088	2088	1150	2088	2088	1150
Groups	315	315	315	309	315	315	309

	OLS	OLS	WG	Long diff.	OLS	WG	Long diff.
ln(all cause mortality)		Controlling for trend, region and regional trends			Controlling for trend, region, regional trends, smoking rate, employment rate and NVQ 4+ level rate		
ln(O_3)	**-0.269*** **	**-0.080***	**0.109*** **	**0.069*** **	**-0.082*** **	**0.106*** **	**0.054*** **
	(0.043)	(0.045)	(0.012)	(0.016)	(0.031)	(0.012)	(0.016)
ln(smoking rate)					0.130***	0.080***	0.147***
					(0.028)	(0.021)	(0.044)
ln(employment rate)					-0.544***	-0.020	0.008
					(0.044)	(0.025)	(0.027)
ln(NVQ 4+ level rate)					-0.099***	-0.009	-0.008
					(0.013)	(0.007)	(0.008)
R-squared	0.09	0.47	0.94	0.06	0.68	0.94	0.07
Observations	2210	2210	2210	1224	2210	2210	1224
Groups	331	331	331	323	331	331	323

Notes: Observations are weighted by the square root of mid-year population estimates for the local authorities. Robust standard errors in parentheses.* significant at 10%; ** significant at 5%; *** significant at 1%

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 ozone on all cause mortality, which is also robust to the time varying controls. The within groups point estimate is a 0.1% increase in all cause mortality for a 1% increase in O_3; the long difference estimates are around half this magnitude.

The association of the controls with all cause mortality are shown in the final three columns of the table. As expected, smoking rates are positively associated with higher death rates. The estimate is significant in the OLS, in our preferred within group specification and in the long difference estimates. The point estimate for the within group estimates indicate that a 1% increase in smoking results in a 0.1 increase in death rates. The long difference estimates are about twice this magnitude. Higher employment and education are negatively associated with death rates, but the coefficients are only significant and of any size in the OLS estimates, 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.[12]

12 We also tested the robustness of these 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. See robustness checks section below.

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

The first three blocks of Table 4 repeat the analyses of Table 3, but include all pollutants simultaneously to allow for correlation between the levels of pollutants. Column 1 shows the raw association, columns 2 to 4 show the OLS, within group and long differences regressions without time-varying controls, columns 5 to 7 show the OLS, within group and long differences regressions with time varying controls other than weather and the final three columns add in controls for weather.

Table 4 confirms that CO has no independent effect on death rates. Apart from the raw association, none of the estimates are significantly different from zero. On the other hand, the other pollutants all remain significantly associated with mortality rates, though the associations are a little smaller than when we examine the pollutants in isolation. The coefficients for NO_2 fall by most (by around a third), while those for PM_{10} and O_3 fall by around 20%. Nevertheless, after controlling for local authority fixed effects and the effects of employment, education and smoking, the estimated impact of NO_2 is about 0.02, that of PM_{10}, 0.08, and that of O_3, 0.09 (though only 0.03 in the long difference estimates).

The final block of Table 4 controls for weather. As noted above, weather is an important determinant of the level of pollution but could also have an independent effect on mortality. We thus include controls for the annual mean of the daily maximum temperature in summer and the annual mean of precipitation. These measures should capture the effects of heat waves (for example, the summer of 2003) and very wet years. To the extent that weather is associated with the levels of pollution but does not have an independent effect on deaths, inclusion of the weather variables will reduce the amount of variation in our pollution measures and make it more difficult to detect their effects.

The results show that our estimated effect of NO_2 is not robust to controls for temperature and precipitation. The coefficient falls by around a half and is not statistically significant. However, the within group estimates for PM_{10} and O_3 are robust to controls for weather. For PM_{10} the within group estimates are very similar with and without weather measures. The effect of controlling for weather on O_3 is in the expected direction: all cause mortality rose in 2003, when the UK experienced an unusually hot summer and this hot weather also produced higher than normal levels of O_3. However, even allowing for this, there still appears to be an effect of O_3 on mortality rates.

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

	OLS	**OLS**	**WG**	**Long diff.**	**OLS**	**WG**	**Long diff.**	**OLS**	**WG**	**Long diff.**
ln(all cause mortality)		Controlling for trend, region and regional trends			Controlling for trend, region, regional trends, smoking rate, employment rate and NVQ 4+ level rate			Controlling for trend, region, regional trends, smoking rate, employment rate, NVQ 4+ level rate, annual mean of daily maximum temperature in summer, annual mean of precipitation		
ln(CO)	**-0.037****	**-0.015**	**-0.006**	**-0.005**	**-0.004**	**-0.008**	**-0.006**	**-0.001**	**-0.008**	**-0.007**
	(0.017)	(0.022)	(0.006)	(0.007)	(0.014)	(0.005)	(0.007)	(0.014)	(0.005)	(0.007)
ln(NO_2)	**-0.056**	**0.019**	**0.024*****	**0.023****	**0.073*****	**0.024****	**0.021****	**0.063*****	**0.011**	**0.004**
	(0.035)	(0.028)	(0.009)	(0.010)	(0.020)	(0.009)	(0.011)	(0.023)	(0.010)	(0.012)
ln(PM_{10})	**0.035**	**0.162*****	**0.082*****	**0.093*****	**0.035**	**0.079*****	**0.089*****	**0.037**	**0.071*****	**0.048*****
	(0.050)	(0.041)	(0.011)	(0.013)	(0.033)	(0.011)	(0.012)	(0.035)	(0.013)	(0.015)
ln(O_3)	**-0.338*****	**-0.040**	**0.094*****	**0.045*****	**-0.030**	**0.092*****	**0.034****	**-0.038**	**0.046*****	**-0.004**
	(0.045)	(0.048)	(0.013)	(0.016)	(0.034)	(0.013)	(0.016)	(0.041)	(0.017)	(0.019)
ln(smoking rate)					0.112***	0.064***	0.114***	0.106***	0.065***	0.091**
					(0.028)	(0.021)	(0.041)	(0.028)	(0.020)	(0.039)
ln(employment rate)					-0.527***	-0.006	0.022	-0.527***	-0.004	0.019
					(0.043)	(0.025)	(0.028)	(0.043)	(0.025)	(0.028)
ln(NVQ 4+ level rate)					-0.106***	-0.008	-0.006	-0.108***	-0.007	-0.005
					(0.013)	(0.007)	(0.008)	(0.013)	(0.007)	(0.008)
ln(annual mean of summer								0.181**	0.171***	-0.000
daily maximum temperature)								(0.090)	(0.045)	(0.003)
ln(annual mean precipitation)								0.028*	0.007	-0.040***
								(0.016)	(0.008)	(0.009)
R-squared	0.10	0.48	0.95	0.11	0.69	0.95	0.12	0.70	0.95	0.14
Observations	2038	2038	2038	1123	2038	2038	1123	2038	2038	1123
Groups	309	309	309	298	309	309	298	309	309	298

Notes: Observations are weighted by the square root of mid-year population estimates for the local authorities.
Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%

The long difference estimates are smaller, but remain statistically significant for PM_{10} though not for O_3. However, the long difference sample is much smaller and the coefficient on summer temperature is of the wrong sign. This suggests that the effect of O_3 is being masked by the control for summer temperatures in this smaller sample: we therefore give more credence to the within group estimates.[13]

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

Table 5 examines the relationship between the pollutants and specific causes of death, selected on the basis that these causes of death are argued to be associated with high levels of pollution. The causes are all circulatory diseases, and then two subsets of this – coronary heart disease and, nested within coronary disease, acute myocardial infarction – and bronchitis, emphysema and other chronic obstructive pulmonary disease (COPD).[14] We present only the within group estimates. The first column repeats the estimates from Table 4 for all cause mortality for comparison. The top panel of the table does not include controls for weather, the bottom does. All other controls are included in both panels and all pollutants are included in each regression.

The top row of both panels shows that allowing for the simultaneous effects of other pollutants there is no association between any of the causes of death and CO levels. The other pollutants, however, are associated with specific causes of death that stem from breathing or circulatory problems. With no controls for weather, NO_2 is positively associated with deaths from bronchitis and emphysema, PM_{10} is associated with deaths from all four specific causes and O_3 is associated with deaths from all circulatory diseases and with death from bronchitis, emphysema and other COPD. Allowing for weather reduces the size and statistical significance of the associations between these specific causes and the levels of NO_2 and O_3. If we allow for weather, neither of these two pollutants is significantly associated with any of the four specific causes. But even with controls for weather, the associations between PM_{10} levels and all causes of death except COPD remain statistically significant. These coefficients indicate that a 1% increase in PM_{10} is associated with a 0.1% increase in mortality from all these causes.

(e) Magnitudes

Our results are statistically significant, but are they also economically significant? We can examine the effect of a change in these sources of pollution on all cause mortality using the within group estimates from the penultimate column of Table 4 for PM_{10} and O_3.

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13 Estimates using differences two periods apart support this as they are closer to the within group estimates. The point estimates are, in fact, larger than the within group estimates: NO_2 0.031 (s.e. = 0.01), PM_{10} 0.086 (s.e. = 0.017), O_3 0.093 (s.e. = 0.018), 1,410 observations, 299 groups.

14 If the focus of the analysis was hospital admissions we could also examine admissions for other chronic diseases of the respiratory system such as asthma. However, the mortality rate from asthma is too low to examine the impact on deaths.

A 10% increase in PM_{10}, holding all other pollutants fixed, is associated with a 0.7% increase in the all cause mortality rate. As the mean all cause mortality rate is 668 per 100,000 population, this increase equals around 5 more deaths per 100,000 persons. The decile ratio of PM_{10} in our sample is 1.39, and so a move from the bottom to the top decile of PM_{10} pollution would be associated with a 2.8% increase in the all cause mortality rate (0.07% x 39), which is around 19 more death per 100,000 persons.

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

		ln(all cause mortality)	ln(all circula-tory diseases mortality)	ln(coronary heart disease mortality)	ln(acute myocardial infarction mortality)	ln(bronchitis, emphysema and other COPD mort.)
Without weather controls	**ln(CO)**	**-0.008**	**-0.010**	**0.011**	**0.000**	**-0.026**
		(0.005)	(0.010)	(0.012)	(0.025)	(0.025)
	ln(NO_2)	**0.024****	**0.015**	**0.006**	**-0.052**	**0.117*****
		(0.009)	(0.016)	(0.021)	(0.039)	(0.043)
	ln(PM_{10})	**0.079*****	**0.139*****	**0.136*****	**0.207*****	**0.128****
		(0.011)	(0.016)	(0.023)	(0.048)	(0.055)
	ln(O_3)	**0.092*****	**0.044****	**0.006**	**0.006**	**0.332*****
		(0.013)	(0.019)	(0.026)	(0.050)	(0.055)
	R-squared	0.95	0.92	0.91	0.85	0.84
Controlling for annual mean of daily maximum temperature in summer and annual mean of precipitation	**ln(CO)**	**-0.008**	**-0.011**	**0.010**	**-0.007**	**-0.029**
		(0.005)	(0.010)	(0.012)	(0.025)	(0.025)
	ln(NO_2)	**0.011**	**0.003**	**0.003**	**-0.094****	**0.037**
		(0.010)	(0.017)	(0.023)	(0.043)	(0.044)
	ln(PM_{10})	**0.071*****	**0.126*****	**0.123*****	**0.103***	**0.050**
		(0.013)	(0.019)	(0.027)	(0.054)	(0.061)
	ln(O_3)	**0.046*****	**0.007**	**0.004**	**-0.053**	**0.078**
		(0.017)	(0.027)	(0.036)	(0.068)	(0.076)
	R-squared	0.95	0.92	0.91	0.86	0.84
	Obs.	2038	2038	2038	2038	2038
	Groups	309	309	309	309	309

Notes: Controls for time trend, region specific time trends, smoking rate, employment rate and NVQ 4+ level rate; additional controls for weather in lower panel. Observations are weighted by the square root of mid-year population estimates for the local authorities.
Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.

A 10% increase in O_3, holding all other pollutants fixed, is associated with a 0.46% increase in the all cause mortality rate. The decile ratio for O_3 is 1.43, so a move from the bottom to the top decile of O_3 is associated with a 2.0% increase in the all cause mortality rate (0.046% x 43). This translates into just under 14 extra deaths per 100,000 persons.

Alternatively, the difference between the top decile and the bottom decile of all cause mortality is 220 deaths per 100,000 population. So a fall from the 90^{th} to the 10^{th} decile of PM_{10} would account for about 9% of the decile 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 6% of the decile spread in all cause mortality.

We can also compare our estimates 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 most influential cohort study, the American Cancer Society Cohort Study, estimates that a 10 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 particulates are worse than that from the coarser particulates which the PM_{10} measure we use here captures but the $PM_{2.5}$ measure excludes. Thus we would expect our estimate to be lower. A meta-analysis of the time series studies by Stieb et al. (2002) reports that single-pollutant models suggest that a 10 $\mu g/m^3$ increase in PM_{10} increases mortality by 0.6% and that multi-pollutant models estimate a 0.4% increase in mortality per 10 $\mu g/m^3$ increase in PM_{10}. In our analysis, a 10 $\mu g/m^3$ increase in PM_{10} corresponds to a 40.3% increase in PM_{10} (evaluated at the mean of 24.8 $\mu g/m^3$), giving an estimated effect of 2.9% (0.071% x 40.3).[15] So our estimate is about half the size of that from the cohort study – which has no UK counterpart – and is nearly seven times as large of that from the times series studies that have been undertaken for the UK.

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 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 increase in O_3 corresponds to an 18% increase in O_3 (evaluated at the mean of 55.7 $\mu g/m^3$), giving an estimated effect of 0.8% (0.046% x 18). Again, our estimates are considerably higher than those from the time series studies which have been undertaken in the UK.

[15] 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).

6. Robustness checks

A sample of the large number of robustness checks we performed is summarized in Table 6. The baseline estimates in row 1 are the within groups estimates from the last block of Table 4, which include the full set of controls and all four pollutants.

(a) Other measures of smoking and diet

Our baseline regression controls for smoking at regional level. As correlations between smoking, diet and pollution may account for the relationship between mortality and pollution, we tested the robustness of our results to measures of smoking at a lower geographical level (health authority); to cumulative smoking patterns as measured by lung cancer mortality rates (see Hansell et al., 2003); and to other measures of diet. We are limited by a paucity of data on diet at local authority level and so use measures that are available at regional level only.

Row 2 of Table 6 adds controls for smoking at health authority level to the base specification. There are 9 regions but approximately 100 health authorities: in principle the more disaggregated data is more desirable, but the health authority estimates are based on surveys with relatively small samples and are therefore less reliable. The coefficients on PM_{10} and O_3 do not change considerably, whereas the coefficient on CO becomes more negative and the coefficient on NO_2 becomes more positive. However, these changes are probably not due to the addition of smoking at health authority level as an extra control, but to the use of a slightly shorter data period, as the estimates using the same time period as for which smoking rates at health authority level are available (1998 to 2003) are very similar to those in row 3.[16] The coefficient on smoking measured at health authority level is not significant, which may be due to the greater measurement error in the data at this more detailed level.

Row 3 adds the local authority lung cancer mortality rate as a measure of cumulated smoking. The coefficients on the pollutants are robust to the inclusion of this measure and the coefficient on the lung cancer mortality rate is significantly positive. The coefficient on our regional smoking rate also does not change.

Row 4 controls at regional level for the percentage of household food energy derived from saturated fatty acids. The estimated effect of fat consumption on mortality is positive as expected and the coefficients on PM_{10} and O_3 do not change considerably.

[16] The coefficients from the base specification in row 1 for the time period 1998 to 2003 are: CO – 0.012 (s.e. = 0.006), NO_2 0.027 (s.e. = 0.011), PM_{10} 0.065 (s.e. = 0.015), O_3 0.048 (s.e. = 0.019).

Table 6: Robustness tests in a multi-pollutant fixed effects model for all cause mortality

		ln(CO)	ln(NO_2)	ln(PM_{10})	ln(O_3)	Coeff. on additional control	Sample period	Observa-tions	Groups
1	Baseline	-0.008 (0.005)	0.011 (0.010)	0.071*** (0.013)	0.046*** (0.017)		1998-2004	2038	309
2	Include log of smoking rates on health authority level	-0.011* (0.006)	0.028** (0.011)	0.064*** (0.015)	0.049** (0.020)	0.002 (0.003)	1998-2003	1730	305
3	Include log of lung cancer mortality as marker for smoking	-0.008 (0.005)	0.010 (0.010)	0.071*** (0.012)	0.048*** (0.017)	0.057*** (0.006)	1998-2004	2038	309
4	Include log of percentage of household food energy derived from saturated fatty acids	-0.008 (0.005)	0.011 (0.010)	0.064*** (0.013)	0.047*** (0.017)	0.292** (0.119)	1998-2004	2038	309
5	Include log of population size	-0.006 (0.005)	0.013 (0.010)	0.081*** (0.013)	0.050*** (0.018)	-0.378*** (0.134)	1998-2004	2038	309
6	Drop observations for 2003	-0.007 (0.006)	0.008 (0.010)	0.053*** (0.015)	0.034* (0.019)		1998-2002 + 2004	1737	309
7	Drop observations with one or more pollut-Ants in top 10 % of pollutant distributions	-0.009 (0.006)	0.008 (0.012)	0.081*** (0.016)	0.040* (0.021)		1998-2004	1656	292
8	Drop observations in South West	-0.010* (0.006)	0.008 (0.012)	0.070*** (0.014)	0.053*** (0.020)		1998-2004	1859	280
9	Drop observations in London	-0.011** (0.005)	0.017 (0.010)	0.064*** (0.013)	0.032* (0.018)		1998-2004	1814	277
10	Include lagged pollutants	-0.004 (0.005)	0.015 (0.011)	0.050*** (0.015)	0.057*** (0.017)		1998-2004	2001	307
11	ln(mortality from suicides)	0.073 (0.063)	-0.030 (0.130)	-0.259 (0.164)	-0.133 (0.184)		1998-2004	2023	309

Notes: Observations are weighted by the square root of mid-year population estimates for the local authorities. Baseline specification includes time trend, region specific time trends, smoking rate, employment rate, NVQ 4+ level 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%

(b) Population changes

Our estimates are weighted by the size of the local authority population, thus 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. Row 5 in Table 6 controls for the population size. The coefficients on PM_{10} and O_3 are unaffected. The coefficient on population size is significantly negative. If we can assume that changes in the size of the population are mainly due to migration, this supports the idea that healthy people are more mobile, leaving a more frail population behind.

(c) Omission of outliers and mis-measurement of air pollution

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 6 examines the robustness of our results to omission of this year. The estimated impact of both PM_{10} and O_3 fall by around a quarter, as might be expected given this year is an outlier, but PM_{10} remains well defined and O_3 is significant at the 10% level.[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 as a whole, we omit observations with one or more pollutants in the top 10% of the pollutant distribution. Row 7 shows that the results are robust to this and, if anything, a little stronger for PM_{10}.

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 8 omits observations in the South West. Our results are little affected by omitting these areas.

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[17] If we allow for a full set of year dummies the coefficient on PM_{10} falls to 0.026 (s.e. = 0.014) and the coefficient on O_3 falls to 0.01 (s.e. = 0.018). However, both weather coefficients have incorrect signs (the coefficient on hot weather is 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 two 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} 0.039 (s.e. = 0.014), O_3 0.051 (s.e. = 0.017), summer temperature 0.22 (s.e. = 0.05)).

The higher density of monitoring stations in London meant we used a smaller radius (10 miles) to assign pollution levels in the capital. In addition, it could be argued that air pollution – at least from CO_2, NO_2 and PM_{10} – is really an urban phenomenon. We therefore checked that our results were not solely due to London by omitting all London observations. Row 9 shows that the estimates are basically unchanged from those of Row 1 for CO, NO_2 and PM_{10}, while the effect of O_3 falls by around a third but remains significant.

We were concerned that we might have mis-specified the dynamic structure of the model. Row 10 therefore includes the lagged levels as well as the current levels of the pollutants. These change the estimated effects of current PM_{10} and O_3 a little compared to the baseline but the estimates of the impact of current pollution remain statistically significant.[18, 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.

(d) Other factors which could account for mortality rates

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 always 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.

One way of testing for this is to examine mortality from a cause that is unlikely to be affected by the within local authority time series variation in pollution. If the coefficients on air pollutants are similar to that found in our specification, then this suggests that some omitted factor may be driving the association we find between pollution and mortality rates. One candidate cause is suicide. The population at risk of suicide is somewhat different from those who die from cardiovascular or respiratory

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18 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 quite similar and of opposite sign to that of current O_3. This result might indicate that the impact of O_3 is simply 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.

19 We also allow for non-linear effects in the pollutants but find no evidence for these, perhaps because our measures are already averaged.

diseases. Suicide rates are higher in males than in females, and are highest in males aged 15 to 44 (Brock et al., 2006). Changes in pollution per se are unlikely to lead to suicide, though suicides may well be driven by changes in economic conditions or weather that are associated with changes in pollution. Row 11 reports the coefficients on pollution from the baseline specification with age-standardised mortality rates from suicide as the dependent variable. This specification (as all others in the table) includes the full set of controls to allow for the fact that suicide may be associated with the economic cycle and weather. The results show none of the coefficients on pollutants are statistically significant and the coefficients on PM_{10} and O_3 are in fact large and negative.

Ruhm (forthcoming) has argued that increases in the business cycle lead to more deaths: in upturns people work longer hours, have less leisure and are subject to more pressure. If this is the case, the higher mortality rates we observe may be due not to pollution, but to the impact of the business cycle. Our preferred specifications do include controls for employment for this reason. We also augmented the Row 1 specification with an additional control for average male pay (available for 1998 to 2001). This additional control was positive but not statistically significant. More importantly, it did not change our pollution estimates, which for this shorter period are all positive and significant for three of the pollution measures.[20] Again, this suggests our results are not driven by general economic activity.

We undertake two further tests to examine whether an association between the business cycle and mortality could be driving our results. First, we test directly the strength of the association between pollution levels and our measures of economic activity. Second, we test whether economic activity is positively associated with death rates. For the first test, we estimate separate within group regressions of each pollutant on the employment rate, controlling for trend, regional trends and local authority fixed effects. The upper panel of Table 7 gives the coefficient on employment rate for each pollutant. For England, pollution at local authority level is not positively associated with employment. The point estimates for CO, NO_2 and O_3 are very small and the only coefficient that is statistically significant is that for PM10, which is negative, suggesting that higher local employment is actually associated with lower levels of PM_{10}.

The lower panel of Table 7 presents the second test, directly testing the association between economic upturns and deaths at local authority level. It reports the coefficients on employment rate from within group regressions of the set of mortality rates used in Table 5 on employment rate, controlling for trend and regional trends. As might be expected from the results presented above, the results show no positive relationship between employment and mortality rates. Most of the coefficients are not significant; the only ones that are significant, which are for mortality from all circulatory diseases and mortality from coronary heart disease, are negative, indicating

[20] The coefficients are: CO 0.02 (s.e. = 0.01), NO_2 0.03 (s.e. = 0.016), PM_{10} 0.029 (s.e. = 0.021), O_3 0.062 (s.e. = 0.024).

that higher economic activity is associated with lower mortality rates during this period in England.[21]
We therefore find that pollution does not appear to be strongly associated with the economic cycle and nor do mortality rates appear to be pro-cyclical. This possible channel therefore does not explain the association we find between higher death rates and pollution.

Table 7: Within group estimates of the association between economic activity and air pollution and the association between economic activity and mortality

a) Association between economic activity and air pollution

	ln(CO)	ln(NO_2)	ln(PM_{10})	ln(O_3)
ln(employ-	-0.055	-0.087	-0.134**	-0.054
ment rate)	(0.108)	(0.066)	(0.052)	(0.038)
R-squared	0.87	0.86	0.75	0.79

b) Association between economic activity and mortality

	ln(all cause mortality)	ln(all circula-tory diseases mortality)	ln(coronary heart disease mortality)	ln(acute myocardial infarction mortality)	ln(bronchitis, emphysema and other COPD mortality)
ln(employ-	-0.022	-0.095***	-0.102**	-0.113	0.000
ment rate)	(0.026)	(0.035)	(0.048)	(0.088)	(0.099)
R-squared	0.94	0.91	0.91	0.85	0.83

Notes: Controls are time trend and region specific time trends. Sample is that used in Table 4 (2038 observations, 309 groups). Observations in Panel b) are weighted by the square root of mid-year population estimates for the local authorities.
Robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.

7. Conclusions

The current air quality limit values in England, as in the USA and much of Europe, are low by historical and international standards and are set to levels which are believed not to be harmful to health. We have sought to assess whether this view is correct

[21] We repeated this analysis using unemployment rates and male pay instead of employment rates. These results, for a shorter time period, also indicated that economic activity was - if anything - negatively associated with mortality rates. The coefficient on the unemployment rate for all cause mortality was 0.009 (s.e. = 0.01) and the coefficient on average male pay was 0.021 (s.e. = 0.044). The unemployment data are for 1998 to 2004, with 449 observations in 87 local authorities; the male average pay data are for 1998 to 2001, with 1416 observations in 354 local authorities.

using panel data at English local authority level to examine whether current levels of regularly measured airborne pollutants are associated with population deaths. We examine deaths from specific cardiovascular and respiratory causes that are known to be exacerbated by air pollution as well as all cause mortality, exploit the panel nature of our data to control for any unobserved time-invariant associations at local authority level between high levels of pollution and poor population health and estimate multi-pollutant models to allow for the fact that three of the pollutants are closely correlated.

Our results suggest that higher levels of PM_{10} and O_3 are associated with higher mortality rates. Cross-sectional analyses would not have identified these effects, as O_3 is higher in more rural areas with lower death rates and PM_{10} is heavily correlated with other pollutants produced from traffic emissions. The robustness of our findings to omission of outliers and the finding that pollution is positively associated with causes of death which are known to be associated with airborne pollutants, but not associated with a non-pollution related cause of death, suggest this approach identifies a true association between pollutants and death rates.

Our estimates can be used to give a back-of-the-envelope calculation of the benefits of the proposed reduction in the limit values for PM_{10} by 2010 to 20.0 $\mu g/m^3$. We estimate a 10% increase in PM_{10}, holding all other pollutants fixed, is associated with a 0.7% increase in the all cause mortality rate. A 0.7% increase in the all cause mortality rate equals 4.6 more deaths at the sample mean of all cause mortality. Therefore reducing PM_{10} pollution from our sample mean of 24.8 to 20.0 $\mu g/m^3$ (a fall of just under 20%) would be associated with 9.2 fewer deaths per 100,000 population. The population of England is just over 50m, so this translates into around 4,600 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. Devlin and Parkin (2004) estimate a value of around £30,000. If we assumed that those who died had another 10 years to live and were healthy, the value of the 46,000 life years gained is around £1,380 million. If they were less healthy, then this figure is too high. But as we do not take into account any of the non-mortality costs associated with pollution, this figure is much more likely to be a lower bound.[22, 23]

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[22] It could be argued that the short run effect of pollution is to kill the frail, so our estimates are an upper bound. However, what we estimate here is not the immediate effect of pollution but the longer term. This follows the general recognition that while an immediate effect of a short run increase in pollution is to kill the frail, long term exposure is more important. For example, the UK government group which examined costs and benefits of pollution concluded that "Evidence indicates that long term exposure to background levels of $PM_{2.5}$ is the most important effect of air quality on public health" and it included as health effects life expectancy losses from those assumed to be in poor health and in normal health (http://www.defra.gov.uk/environment/airquality/publications/stratreview-analysis/index.htm).

Finally, whatever valuation of the benefits is derived, 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. In fact, they are considerably closer to those derived from the much less common – and far more expensive – cohort studies. So our study suggests that reliance on results from UK, which mainly come from time series studies, will seriously underestimate the adverse health consequences of current levels of UK airborne pollution.

[23] In addition, our pollution measures are averaged over a year and over space, so inducing measurement error which will bias our estimates downwards.

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Appendix A: Current air quality standards

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

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

Appendix B: 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 μg/m^3
O_3: 1 ppb = 2.00 μg/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	Clinical and Health Outcomes Knowledge Base (www.nchod.nhs.uk)	1998-2004
Mortality from all circulatory diseases		
Mortality from coronary heart disease		
Mortality from acute myocardial infarction		
Mortality from bronchitis, emphysema and other chronic obstructive pulmonary diseases		
Mortality from accidents		
Mortality from suicide		
Mortality from lung cancer		
Covariates		
Smoking rate, regional level	Clinical and Health Outcomes Knowledge Base	1998, 2000-2003
Employment rate	Labour Force Survey (www.nomisweb.co.uk)	1998-2004
NVQ 4+ level rate		
Annual mean of summer daily max. temperature	Met Office – MIDAS Land and Surface Station Data	1998-2004
Annual mean of precipitation		
Household food energy derived from saturated fatty acids	Clinical and Health Outcomes Knowledge Base	1998-2004
Smoking rate, Health Authority level	Health Survey for England	1998-2003

Table A.1: Correlation between annual levels of pollution

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 A.1: Quantile plot of weekly CO concentrations at local authority level