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Atmospheric pollutants and mortalities in English local authority areas

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ABSTRACT

Objectives: To measure geographical co-relationships between disease-specific standardised mortality ratios (SMR) and different atmospheric emissions in 352 English local authorities. To link specific exposures with specific causes of death and to identify responsible polluting sources. To see whether long-term moderate exposures have the same lethal effects as short-term high-pollution (ie, smog) episodes.

Design: Geographical distributions of SMR, atmospheric emissions and social hazards, extracted from three different sources, were converted to a congruent format. Correlation coefficients were calculated within and between these different datasets. Mortality/pollutant correlations were recalculated after additionally standardising the SMR for social differences between local authorities.

Setting: The 352 English local authority areas, 1996–2004.

Main results: SMR for one group of diseases (including upper alimentary and respiratory cancers, ischaemic heart disease, peptic ulcer, pneumonia) were related to a range of combustion emissions and to multiple social deprivation, cigarette smoking, binge drinking and a northern location. Additional standardisation of all SMR for these social hazards left a small subgroup independently related to atmospheric pollution, mainly from oil combustion. Correlations with pneumonia deaths were exceptional.

Conclusions: High mortality rates were observed in areas with elevated ambient pollution levels. The strongest single effect was an increase in pneumonia deaths. Road transport was the chief source of the emissions responsible, although it was not possible to discriminate between the different chemical components. Many “pneumonia” deaths were probably caused by direct chemical injury, as in the 1952 London smog and are better regarded as “acute respiratory distress syndrome” or “acute lung injury”.

There are many records of high mortality rates during periods of severe atmospheric pollution. They include the disastrous London smog of 1952, which killed 4000 people and the central European episodes of the 1980s.^{1–3} Some major exposures have involved specific substances but most have arisen from exceptional meteorological conditions and have involved a wide mix of combustion-based or dust-carried materials. The most widely suspected agents have been sulphur dioxide, nitrogen oxides and particulates but specific agents have not been clearly identified.^{4–7} The modes of action are also uncertain and certified causes of death reported during major episodes were not limited to respiratory diseases. There were also problems in distinguishing between the immediately fatal illness

and pre-existing disease.¹ In ongoing exposure studies, daily variations of atmospheric pollution clearly result in immediate increases in sickness rates and impaired respiratory functions but have not successfully demonstrated the fatal mechanisms in the major episodes.⁸ Other long-term prospective studies of cohorts exposed to continuing specific pollutant levels have confirmed the increased total mortality experiences of the major pollution events. These investigations, which include the Harvard Six Cities study^{9 10} and others in Norway, The Netherlands and France,^{11–13} were able to note and to allow for variations in individual hazards, such as smoking and occupation but they still leave unanswered questions. As with all cohort studies, their results are subject to unrecognised confounding and their conclusions strictly relate only to those substances around which the cohorts were initially characterised.

The present investigation asks the following additional questions. Do long-term low-to-moderate exposures of total populations to atmospheric pollutants carry the same elevated mortality risks as in major episodes? What specific causes of death can be attributed to such pollution? What are the effective components among the mix of exposures? What are the sources of the effective agents?

These questions are addressed through examining mortality variations for many different diseases and for varying mean levels of exposure to different atmospheric emissions across 352 English local authority areas.

MATERIALS AND METHODS

Standardised mortality ratios (SMR) for a wide range of “underlying causes of death” between 1996 and 2004, for each of 352 local authorities, have been assembled and published by the Oxford Cancer Intelligence Unit.¹⁴ They are presented both as maps (which are readily accessible on the internet) and as tables. The latter provide the “outcome” basis for the present examination. Values based on small numbers were omitted from some tables but 45 disease-specific datasets were sufficiently complete and otherwise considered suitable for analysis. Some were examined for men and women separately.

The National Atmospheric Emissions Inventory (NAEI) assembles annual estimates of a range of different particulate and gaseous emissions within each 1 × 1 km square of the UK National Grid.¹⁵ They are grouped by local authority and within local authority by each of 11 main sources including: “domestic institutional and commercial”, “road transport”, “other transport”, “power production”, “industrial”, “natural”, etc. Grid-square estimates are

based upon the frequencies of emission-generating activities within each square, multiplied by the types and levels of emissions known to be generated by each.

Accumulated data for major emissions (2004) are also presented in the form of internet maps showing emission levels on a six-point colour scale. Local emissions are supplemented by estimates of materials diffusing from identified major sources from further afield. The coloured map pixels have a ground equivalence of 0.38 km, giving false resolution relative to the original grid data but the centres of grid squares can be related to single pixels within acceptable tolerances. This provides a practical method of scanning large and complex volumes of exposure data. (The map and colour scales used for these 2004 maps differ from previously available maps for 2001, as used in a previous study.¹⁶)

The main technical problem, apart from decoding the coordinates of the pixels, was to relate the grid square emissions data to the local populations that had generated the SMR. The local authority areas vary greatly in size and in their patterns and densities of habitation, so neither total local authority emissions nor mean grid square values adequately indicate personal exposures. This demanded identification of a population centre point for each local authority and the designation of a standard surrounding "emissions catchment" area for extracting an exposure score. Three potential centre points were: (1) the map reference of the central local authority post code (xxx 1AA); (2) a geographical centroid obtained from the NAEI local authority tabulations; (3) the location of maximum carbon dioxide emissions from "commercial institutional and residential combustion" (ie, space heating of schools, shops, hospitals, offices and homes). After initial investigations the last of these was adopted. It was based upon the greatest sequential north-south 3 square "domestic" carbon dioxide sum.

Alternative designations of standard surrounding areas were based upon map squares within different ranges of the centre points, allowing double or treble counting of inner zones. A narrower range improves specificity but a wider one allows for personal mobility, work day as well as domestic exposure and the effects of distant diffusing sources such as incinerators, refineries, large factories and tall chimneys. A broad range also caters for wider episodes of trapped pollution covering groups of

local authorities, such as occur seasonally or under anticyclonic conditions. In practice all the tested variations gave similar results. Those presented below used an outer square within ± 15 km of the centre point (ie, 31×31 grid squares) and inner zones of ± 5 km (double counting) and ± 1 km (treble counting). There was substantial overlap between adjacent catchments and many extended over adjacent local authority territory.

The NAEI mappings for different emissions (in weight/km² per year) used widely varying scales ranging from 1000s of tonnes (nitrogen oxides, volatile organic compounds (VOC), PM₁₀ (fraction of particulate matter in air of very small size (<10 μ m)), benzene, benzopyrene, 1,3-butadiene, sulphur dioxide, carbon monoxide) through some 10s or 100s of kilograms (arsenic and metals) to grams (dioxins). These different gravimetric scales were reduced to a common relative score based upon an interpretation of NAEI scaling practices. Again, several alternatives were examined and all gave similar results. The version used in the following tables allocated 20 points to the highest concentrations (red pixels) followed by 6, 3, 1, 0.1, 0.0, to the lesser values (orange, yellow, green, light blue, other).

Levels types and sources of emissions are strongly confounded with other local authority-specific health hazards such as poverty, social deprivation, local industrial risks, poor education, high traffic and housing densities and hazardous employments and lifestyles. To allow for this, social indicators for all local authorities were extracted from available sources. They included the 2004 revision of the Index of Multiple Deprivation (IMD) and a government-published list of local authority profiles giving the percentage of adult smokers and the percentage engaging in regular binge drinking.^{17 18} All are based upon social surveys. The National Grid eastings and northings of the population centre points, which also reflect variations in relative wealth, education and lifestyles, were likewise taken into account.

The different datasets were resequenced to correct different alphabetic conventions and to allow for omissions, bringing all into a congruent format. Comparisons were then based upon coefficients of correlation (r). Linear regression equations were also used in order to adjust SMR for social factors, beyond their intrinsic standardisations for age and sex. For example, the

Table 1 Significant ($p < 0.01$) correlations (r) between different diseases

	Cancers of								Multiple sclerosis
	Lung	Stomach	Rectum	Prostate	Brain	Melanoma	Breast	Ovary	
Cancers of									
Lung	–	–	–	–0.29	–0.24	–0.11	–0.24	–0.22	–0.44
Stomach	0.82	–	–	–0.21	–0.22	–0.52	–	–0.18	–0.19
Rectum	0.57	0.58	–	–	–	–0.31	–	–0.14	–0.31
Oesophagus	0.34	0.34	0.37	–	–	–	–	–	–
Pancreas	0.22	0.24	0.27	–	–	–	–	–	–0.31
Bladder	0.54	0.42	0.37	–	–	–0.19	–	–	–0.20
Non-cancers									
Ischaemic heart disease	0.74	0.78	0.58	–	–	–0.49	–	–0.21	–0.18
Stroke	0.29	0.24	0.17	–	–	–0.21	–	–0.21	–
Rheumatic heart disease	0.46	0.44	0.37	–0.16	–0.14	–0.27	–	–0.21	–0.18
Pepticulcer	0.48	0.39	0.30	–	–	–0.22	–	–0.15	–
Diabetes	0.32	0.40	0.28	–	–0.30	–0.34	–	–0.16	–0.23
Chronic obstructive pulmonary disease	0.77	0.69	0.48	–0.17	–0.26	–0.38	–	–0.24	–0.23
Asthma	0.40	0.41	0.27	–	–0.17	–0.23	–	–	–0.17
Pneumonia	0.41	0.30	–	–	–0.16	–0.27	0.16	–	–

One disease cluster shows strong positive mutual associations among its members (columns 1–3) whereas diseases in columns 4–9 show strong negative associations with the first group. Each disease cluster includes both cancers and non-cancers.

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linear dependency equation for lung cancer upon smokers per cent, was used to calculate new "expected" SMR for each local authority. The calculated value was divided into the current value to provide an additional correction and so on for all diseases and all standardising variables. Successive standardisation for several variables (as used here) can lead to mutual conflicts between variables but, in practice, no such problems arose.

The statistical significance (p) of an r value depends solely upon the number of variable pairs. For 352 pairs, p values of 0.05, 0.01, 0.001, 0.0001 correspond with r values of (\pm) 0.104, 0.137, 0.176, 0.208. In view of the large number of pairings examined, meaningful interpretations must be limited to especially high levels of significance. This requirement is reinforced by evident spatial autocorrelations of emissions catchments and of mortality experiences between adjacent local authorities. This issue is discussed later.

RESULTS

Wide coefficients of variation (CoV) (ie, SD/mean, %) showed major heterogeneities between local authorities in terms of the main social and emissions score variables: eg, IMD (48.6), alcohol bingers (21.8), benzene (67.2), 1,3-butadiene (81.3),

non-methane VOC (68.9), PM₁₀ (62.9), dioxins (41.8), arsenic (35.8), cadmium (51.2), carbon monoxide (64.1), chromium (52.9), copper (41.8), mercury (30.6), nickel (53.3), nitrogen oxides (58.0), lead (39.0), selenium (50.5), sulphur dioxide (23.6), vanadium (54.2), zinc (40.5). Calculated correlations between the different emissions, between the different social variables and between components within the two main datasets, showed high levels of mutual confounding. The main exception was for dioxins, which returned mainly negative (although significant) cross associations.

Pairings of many metallic and gaseous pollutants returned coefficients (r) greater than 0.8 ($p < 0.0001$), indicating that discrimination between their separate effects may not be possible on this scale.

The SMR showed varying distribution patterns with high CoV for some and much lower ones for others. High CoV were recorded for cancers of the lung (24.8), stomach (26.8) and pancreas (33.5), as well as for melanoma (25.6), asthma (32.6), pneumonia (19.2), chronic obstructive pulmonary disease (COPD) (28.2), diabetes (25.1), rheumatic heart disease (33.1), ischaemic heart disease (IHD) (15.5), myocardial infarction (20.1), peptic ulcer (21.6), peripheral vascular disease (24.5), multiple sclerosis (33.1), epilepsy (34.4) and motor neurone disease (25.3). Lower CoV were recorded for cancers of the

Table 2 Significant ($p < 0.01$) correlations (r) between hazards and diseases

	Social variables				Atmospheric emissions							
	IMD	Smoke %	Binge %	North	Benzene	Butadiene	PM ₁₀	CO	NO _x	SO ₂	Cr	Zn
Cancers of												
Lung	0.82	0.75	0.66	0.49	0.30	0.28	0.33	0.28	0.30	0.31	0.33	0.26
Stomach	0.72	0.64	0.59	0.53	0.22	0.20	0.26	0.20	0.22	0.30	0.31	0.20
Cancers of												
Oesophagus	0.35	0.30	0.36	0.32	-0.22	-0.21	-0.14	-0.20	-0.18	-	-	-0.18
Rectum	-	0.44	0.55	0.55	-	-	-	-	-	0.22	-	-
Bladder	0.45	0.45	0.31	0.21	-	-	-	-	-	0.13	-	-
Pancreas	0.20	0.15	0.13	0.25	-	-	-	-	-	-	-	-
Cancers of												
Colon	-	-	-	-	-0.18	-0.20	-0.16	-	-0.16	-	-0.14	-0.17
Uterus	-	-	-0.14	-	-	-	-	-	-	-	-	-
Breast	-	-	-	-	-	-	-	-	-	-	-	0.14
Ovary	-0.27	-0.21	-0.15	-0.17	-0.24	-0.26	-0.24	-0.25	-0.23	-	-0.21	-0.19
Prostate	-0.25	-0.27	-0.24	-0.15	-0.24	-0.25	-0.15	-0.24	-0.23	-	-0.21	-0.19
Brain	-0.34	-0.28	-	-	-0.23	-0.25	-0.26	-0.24	-0.22	-0.18	-0.22	-0.17
Melanoma	-0.39	-0.33	-0.31	-0.47	-0.17	-0.19	-0.21	-0.19	-0.17	-0.24	-0.22	-0.18
Hodgkin's	-0.16	-	-	0.14	-	-	-	-	-	-	-	-
Non-cancers (1)												
Ischaemic heart disease	0.68	0.57	0.60	0.63	-	-	-	-	-	0.22	-	-
Peripheral vascular disease	0.46	0.42	0.44	0.43	0.15	-	0.19	0.15	0.17	0.23	0.24	0.17
Stroke	0.29	0.27	0.24	0.26	0.14	0.16	0.15	0.16	-	-	-	-
Rheumatic heart disease	0.39	0.39	0.21	0.27	0.25	0.30	0.27	0.27	0.22	0.32	0.28	-
Peptic ulcer	0.46	0.43	0.21	-	0.30	0.27	0.32	0.28	0.31	0.24	0.34	0.29
Diabetes	0.49	0.40	-	0.17	0.18	0.19	0.21	0.17	0.16	0.24	0.21	0.15
Chronic obstructive pulmonary disease	0.68	0.62	0.48	0.37	0.26	0.25	0.29	0.25	0.27	0.27	0.31	0.26
Asthma	0.45	0.54	0.41	0.22	0.17	0.15	0.17	0.14	0.15	0.18	0.17	-0
Pneumonia	0.30	0.23	-	-	0.46	0.46	0.46	0.44	0.49	0.25	0.45	0.48
Non-cancers (2)												
Dementia	-	-	0.23	0.24	-0.19	-0.20	-0.16	-	-0.17	-	-	-
Multiple sclerosis	-0.23	-0.23	-	-	-0.17	-0.15	-0.18	-0.16	-0.18	-0.18	-0.20	-0.18
Motorneurone disease	-0.19	-	-	-	-0.25	-0.25	-0.25	-0.24	-0.26	-0.17	-0.24	-0.22
Parkinsons disease	-0.39	-0.31	-0.21	-0.16	-0.16	-0.15	-0.17	-0.15	-0.16	-	-0.19	-0.15

CO, Carbon monoxide; Cr, chromium; IMD, Index of Multiple Deprivation; NO_x, nitrogen oxides; PM₁₀, fraction of particulate matter in air of very small size (<10 μ m); SO₂, sulphur dioxide; Zn, zinc.

The five successive panels show disease subgroups with different patterns of association with the main social variables (columns 1–4) and the main emissions variables (columns 5–12).

oesophagus (14.6), colon (10.0), breast (8.6), ovary (12.5), prostate (10.0) and brain (15.3).

The high CoV diseases were positively correlated with each other suggesting joint susceptibility to a group of mutually correlated hazards. A selection of these associations is shown in the first three columns of table 1. A second disease group with lower CoV (shown in columns 4–9) returned neutral or negative associations with the first group, although they shared many positive associations with each other. Other causes of death including dementia, congenital defects, Crohn's disease, rheumatoid arthritis, renal failure, heart failure, thyroid cancer, cancer of the uterus, lymphoid leukaemia, diverticular disease and gallbladder disease, showed only non-significant or incoherent patterns of association.

Relationships between independent and dependent variables were even more intricate. They are shown in table 2, in which successive panels show two cancers (lung and stomach) jointly related to both social and combustion variables, a second cancer group with social but without major emissions correlations and a third showing null or negative associations with both. The fourth and fifth panels show nine non-cancers with jointly positive associations and four with mainly negative ones.

The question arises whether combustion emissions are a direct cause of the raised mortality rates in panels 1 and 4, or whether other socially correlated hazards are truly responsible. In addition, do the negative emissions correlations of panels 3 and 5 reflect specific hazards of a "non-IMD" lifestyle (eg, dietary or alcohol excesses, agricultural hazards or an ultraviolet-transparent atmosphere) or simply an escape from one kind of fatal illness to another?

To answer these questions, the SMR were additionally standardised using the social variables. Single-factor standardisations dispersed many but not all of the associations. For example, standardisation for IMD alone left intact a strong grouping of IHD, binge drinking and a more northerly location. For this reason, all the SMR sets were corrected successively for all five social parameters, namely grid easting, grid northing, IMD, smoking and binge drinking, using the method described earlier. This successfully dispersed almost all the positive associations between diseases and emissions. The exceptions were cancers of the lung and stomach, rheumatic heart disease, COPD, peptic ulcer and pneumonia. These standardised

correlations are shown in table 3. Deaths from pneumonia returned much the most powerful associations and, except for sulphur dioxide, all were significant at $p < 0.0001$. Notable absentees from the standardised SMR list were asthma and IHD.

The first seven emissions of table 3 together indicate the dominance of oil combustion. Coal combustion, as indicated by sulphur dioxide, played a relatively minor role. Engine exhaust accounts for the greater part of carbon monoxide, 1,3-butadiene, PM₁₀ and nitrogen oxides, whereas fuel evaporation and incomplete combustion account for much of the benzene and VOC. Copper, lead, selenium and zinc derive partly from brake and tyre wear and vanadium from oil combustion. The other metals come from a range of industrial activities that themselves are partly dependent on oil fuel and that also act as hubs for road and rail transport.

The dominant role of motorised transport was tested by a further stage of standardisation. When the socially standardised SMR in table 3 were additionally corrected for their dependency upon 1,3-butadiene, the most specific indicator of engine exhaust, all these correlations (not just 1,3-butadiene) disappeared.

DISCUSSION

Main findings

The main finding was a strong correlation between deaths from pneumonia and engine exhaust emissions, together with other transport-related substances. The pneumonia correlations far exceeded those of all other SMR in every class of exposure suggesting a direct lung contact injury. Excess deaths assigned to COPD and rheumatic heart disease, two diseases with chronic respiratory inadequacy, can also be interpreted as directly contact toxic. Asthma, with intermittent rather than entrenched respiratory impairment, did not show the same effect.

The full extent of environmentally determined fatal pneumonia in the population is indicated by the wide variation of its SMR, ranging from 60 in Berwick-upon-Tweed to 162 in Lewisham; this is in the context of 386 374 pneumonia deaths in the whole of England during these eight years. In the 35 (10%) highest SMR local authorities alone, there were 53 821

Table 3 Significant ($p < 0.01$) socially standardised correlations between diseases and exposures

	Lung cancer	Stomach cancer	RHD	Pneumonia	COPD	Peptic ulcer
Benzene	0.29	0.20	0.19	0.37	0.18	0.18
Benzpyrene	0.14	0.16	0.21	0.35	0.15	0.19
1,3-Butadiene	0.26	0.18	0.18	0.37	0.17	0.15
PM ₁₀	0.23	0.18	0.19	0.37	0.16	0.18
VOC	0.26	0.18	0.18	0.38	0.17	0.17
Carbon monoxide	0.25	0.18	0.19	0.37	0.17	0.16
Nitrogen oxides	0.31	0.21	0.21	0.42	0.21	0.20
Sulphur dioxide	—	—	—	0.18	—	—
Arsenic	0.14	0.19	0.17	0.30	0.15	0.17
Cadmium	0.30	0.23	0.22	0.42	0.22	0.21
Chromium	0.22	0.22	0.20	0.37	0.18	0.21
Copper	0.30	0.23	0.24	0.45	0.24	0.23
Lead	0.19	0.17	0.19	0.37	0.16	0.19
Selenium	0.30	0.22	0.22	0.42	0.22	0.20
Vanadium	0.31	0.23	0.22	0.44	0.23	0.22
Zinc	0.31	0.23	0.23	0.44	0.24	0.22

COPD, Chronic obstructive pulmonary disease; PM₁₀, fraction of particulate matter in air of very small size (<10 µm); RHD, rheumatic heart disease; VOC, volatile organic compounds.

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pneumonia deaths, an excess of 14 718 over the national rate expectation. Total annual losses as a result of air pollution, through pneumonia, probably approach those of the 1952 London smog.

The excess deaths from lung and stomach cancers and peptic ulcer are more difficult to interpret but the same pollutants may have acted as adjuvants to other agents, facilitating their access to sensitive tissues. The lung cancer and stomach cancer associations were identical in men and in women, reducing the likelihood of a specific occupational effect. Apart from these two diseases and the special case of childhood cancer,^{16 19} there was no evidence from these data that combustion emissions contributed to overall cancer mortality.

Strong correlations between different emissions, aggregated on this geographical scale, prevented a clear differentiation of individual effects. Strong correlations of metallic emissions with several diseases suggest possible direct toxicities but they probably arise mainly from confounding with transport effluents. In addition, more than one substance may be active; atmospheric chemical reactions, converting raw fumes to corrosive aerosols, are thought to be catalysed by metallic particles.⁶

Limitations

Conclusions drawn from ecological contrasts are rightly treated with caution. This study was not, however, a blind trawl. It was based upon specific questions arising from earlier studies and the results are compatible with the projected expectations. Four main technical problems must, however, be reviewed.

The first was the problem of bringing pollution data, mortality data and social data, all from different sources, into common geographical register. The analysis depended upon a set of ad hoc reconciliations. This demanded exploration of a range of alternative methods to ensure that the findings did not depend critically upon one particular choice of parameters.

The second problem was the coarse spatial resolution of the available mortality data. Earlier examinations of relationships between child cancers and perinatal exposures to nearby traffic effluents had shown a need for data of much higher spatial resolution.¹⁹ The same could be true for other diseases and toxicities operating only at short range that may not have been detected.

The third concern was a time shift between exposures and effects and long latent intervals between (some) disease initiations and subsequent deaths. The validity of the analysis supposes at least a moderate correspondence between current and earlier geographical patterns of exposures. Changes in their distributions, perhaps combined with population migrations, could have masked true associations. For all these reasons, negative conclusions drawn from these data and the weaker positive associations are less than reliable. For example, the ability of the lung cancer correlations to withstand social

standardisation and thus to appear among the select group in table 3 might have been the result of time-dependent inefficiencies of the standardisation process although the absence of a similar effect for IHD is against this.

Further problems arose from the intrinsic complexity of the relationships. This demanded a step-by-step dissection and a progressive differentiation of more eligible from less eligible associations and significance values had to be interpreted against the large numbers of possibilities thus examined. There was also the possibility (as always) of false effects from confounding factors outside the scope of the available data and the conclusions depended very much upon the strengths of the final correlations and upon the plausibility of suggested physical mechanisms. Spatial autocorrelations between adjacent local authorities were an additional concern here. Such correlations infringe the independence criterion on which assessments of significance depend. Fortunately, social standardisation restricted the possible causal relationships to a small group of diseases, at the same time eliminating major sources of geographical autocorrelation such as grid easting, grid northing and IMD. Finally, when additional standardisation for 1,3-butadiene completely neutralised all the remaining oil combustion correlations, it left no indication of external confounding or of an autocorrelation residue.

CONCLUSIONS

Acute lung injury (ALI) and acute respiratory distress syndrome are currently recognised as distinctive clinical entities (or sometimes as a combined entity) with high immediate fatality rates.^{20 21} Some cases result from therapeutic misadventures such as blood transfusions or drug sensitivities but there are also many known environmental causes including chemical injuries. A population incidence for ALI of 20–50/100 000 per year,^{22–25} means that large local authorities can expect 50–250 cases per year. The present findings therefore suggest that many “pneumonia” deaths in high-exposure zones may have been from ALI through direct chemical injury. This is consistent with observations in the 1952 London smog when respiratory deaths increased from 49 to 207 in successive four-day periods (in the absence of influenza) and in which autopsy data showed intense capillary engorgement and loss of epithelium.¹ Many died suddenly, at work or out of doors or were found dead in bed. In effect, these people were gassed and this mechanism could equally explain many of the current “pneumonia” deaths in high-exposure zones.

This new perspective could profitably guide treatment and prevention, the latter through better exhaust gas control, traffic restrictions, re-housing, domestic/workplace air filters, switching off idling engines and providing personal respirators to people with respiratory impairments. Environmental surveillance systems should pay special attention to pollution levels near important sources of exhaust gasses (motorways, city

What this paper adds

Relationships between ambient atmospheric pollutants and specific mortalities, in English local authority areas, are displayed for the first time. There was a clear relationship between oil combustion effluents, especially traffic effluents and an excess of deaths certified as caused by pneumonia. The actual causes of many of these excess deaths may have been a directly toxic acute lung injury, a finding concordant with deaths during the London smog of 1952.

Policy implications

Cases of pneumonia in high pollution zones and at high pollution times should be suspected and if necessary treated as acute lung injury. Preventive measures to control local population exposure to oil combustion pollutants, especially engine exhaust, should be put in place. Environmental control responsibilities should include regular map plotting of pneumonia deaths in order to detect and display the high-risk zones.

centres, ring-roads, bridge/tunnel approaches, airports, seaports, bus stations, railway stations, factories, incinerators, supermarkets, trading estates, hospitals). More directly, they could simply plot the work addresses and home addresses of the last few years' deaths from pneumonia.

Competing interests: None declared.

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