Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK

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Abstract

Objective—To test for a significant association between air pollution and emergency hospital admissions for circulatory diseases (international classification of diseases-9 390-459) in London, England, that would be consistent with a causal effect of pollution on the previous day.

Methods—Long term concurrent trends, temperature, humidity, day of the week, influenza epidemic of 1989, and cyclical covariations with periodicity >20 days in daily measures of pollution and admissions for 1987-94 were allowed for.

Results—There were 373 556 admissions. No association was found between O_3 and circulatory diseases. Four other pollutants were associated with acute myocardial infarction and circulatory diseases combined. P values and attributable cases (95% confidence intervals) for acute myocardial infarction were: black smoke $P=0.003$, 2.5% (0.8% to 4.3%); NO_2 $P=0.002$, 2.7% (0.8% to 4.6%); CO $P=0.001$, 2.1% (0.7% to 3.5%); and SO_2 $P=0.0006$, 1.7% (0.7% to 2.6%). There were also associations between black smoke and angina ($P=0.02$), NO_2 and arrhythmia ($P=0.04$), and CO and other circulatory diseases ($P=0.004$), but none with heart failure. Acute myocardial infarction was the only diagnosis for which there were significant associations with and without adjustment for cyclical terms. The associations with acute myocardial infarction were significant only in the cool season.

Conclusion—Population data were consistent with 1 in 50 heart attacks currently presenting at London hospitals being triggered by outdoor air pollution. Further research is now needed to investigate whether background concentrations of black smoke, NO_2 , CO, and SO_2 are a preventable cause of myocardial infarction. These results, if applied to all myocardial infarctions in the United Kingdom, indicate a potential saving of 6000 heart attacks a year.

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Biological mechanisms by which air pollution at current levels might cause morbidity and death are speculative, but inflammation of the airways may be a common pathway for several pollutants including particulate matter,¹ and carbon monoxide (CO) reduces the oxygen transport capability of haemoglobin. Statistical associations between levels of air pollution currently experienced in cities and daily counts of mortality have been explored in more than 30 studies, and reviewers agree that the data indicate a consistent positive association.²⁻⁴ Hospital admissions for respiratory and cardiovascular diagnoses not restricted to emergencies, and also emergency room use, show consistent associations with air pollution as reviewed by Lipfert.⁵ The diagnosis of the cause of emergency admission to hospital, as confirmed at discharge, is likely to be more closely related to a mechanism by which pollution might act than the underlying cause of death from the death certificate, and will relate to more serious conditions than non-emergency admissions or emergency room visits. We therefore studied emergency admissions to London hospitals 1987-94 for circulatory diseases, which is the largest category of deaths. Associations of admissions were sought with daily measurements of ozone (O_3), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), CO, and black smoke. Although time series data of this type cannot establish causality, close and specific significant associations would encourage controlled experimentation and the search for biological mechanisms.

Previous workers have considered a range of lags from the same day up to 10 days previously for the effect of pollution and meteorology on health. With cumulative combinations these choices generated from three to 20 different lags, from which the most significant associations were presented in the published results. On the other hand, a single choice of lag reduces the multiplicity of tests and hence makes P values more meaningful. Based on published results, our previous work, and physical considerations, but before examining the data on circulatory admissions, we selected a lag of one day. The present investigation looks for significant associations between outdoor metered concentrations of five pollutants from 1987-94 and emergency hospital admissions for circulatory diseases on the next day.

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Methods

ENVIRONMENTAL MEASUREMENTS

Measurements of O₃, NO₂, SO₂, and CO were from a background monitoring site in central London near a mainline railway station with bus and taxi ranks. O₃ was taken as the mean of eight hourly assessments between 0900 and 1700 Greenwich mean time, but was not used if more than two hourly assessments were missing. The NO₂, SO₂, and CO measurements were means of 24 hourly measurements, but were not used if there were less than 18 values. Black smoke was a mean of concentrations from one central site and four suburban sites. Missing values of black smoke at individual sites were interpolated by the method of Buck.⁶ The CO was measured by gas filter correlation.⁷ Measurement techniques for the other pollutants have been described.⁸

Temperature and humidity data were obtained from the Meteorological Office monitoring site in central London. Temperature was taken as the mean of minimum and maximum daily temperatures, or was not used if there were less than 18 hourly values. Humidity was taken as the mean of values taken at 0600 and 1500, but was not used if either of these values were missing.

CIRCULATORY DISEASES

Hospital episode statistics data from 1 April 1987 to 31 March 1994 for diseases of the circulatory system, international classification of diseases ninth revision (ICD-9) 390–459, were used. Analyses were performed for the combined group and the following subgroups: acute myocardial infarction (ICD-9 410), angina pectoris (ICD-9 413), other ischaemic heart disease (ICD-9 414), arrhythmia (ICD-9 427), heart failure (ICD-9 428), cerebrovascular disease (ICD-9 430–8), and all remaining codes. The groupings were chosen before the time series data were analysed to give mean daily counts in excess of 10 for London, according to previously published data.⁹ The same definition of London and type of admissions were used as previously.¹⁰

METHOD OF ANALYSIS

The objective of the analysis was to find whether there was a significant association between a pollutant and hospital admissions, after allowing for meteorology and calendar variables. The definition and selection of model components was based on the scheme used by APHEA.¹¹

A starting date for a six week period around the influenza epidemic at the end of 1989 was identified between 25 October and 4 Decem-

ber 1989 so that a variable equal to one on each day of the 42 day period and zero at other times gave the lowest error sum of squares in a simple linear regression model for admissions using the flu indicator variable alone.

Six indicator variables for the days of the week and one for public holidays were used. Long term linear and quadratic functions of time for the whole seven year period and constant, linear, and quadratic trend terms for six individual years starting 1 April added a further 20 terms. To remove associations due to covariation over periods greater than 20 days, cyclical trends made up of pairs of sine and cosine terms for all integer frequencies over the seven years from two to 127—that is, with wave lengths from 3.5 years down to 20.1 days—were included in the model.

The 126 pairs of cyclical terms, but not the other terms, were eligible for elimination from a multiple linear least squares regression model without autoregressive terms by an automated step down variable selection procedure. The least significant pair was eliminated iteratively unless the step down *F* test gave a *P* value <0.01. The cyclical components were determined once only for each diagnostic group, and were not dependent on meteorological or pollution data. There were no missing data associated with modelling calendar terms.

Temperature, humidity, and pollutant were included in the models as untransformed linear terms lagged by one day as suggested by earlier work,^{8 10 12} and six autoregressive terms were included to reduce the effect of correlated errors within weeks.

Single pollutant Poisson models were analysed for each of the eight groupings of emergency admissions. In keeping with other reports a pollutant was said to be significantly associated with admissions if *P*<0.05, without adjustment for the number of tests, which remained large even though we used a specific lag.

When there was a significant association in the full model, we were concerned that this might have arisen as an artefact of the complex modelling. We tested for the presence of an association in the same direction in a partial model. Temperature, humidity, and long term trends on the scale of one year were retained in the partial model, but the cyclical components were not, thus testing for the presence of a significant association before allowing for covariation in admissions and pollution over 20 days to one year. For the partial model, the significance test for pollutant was also simplified from a test for a marginal explanatory term in multiple non-linear Poisson regression with overdispersion to a simple test for univariate Pearson correlation of the pollutant with the residuals from a least squares regression of log count.

Further analyses were performed only for the diagnostic grouping, acute myocardial infarction, which had the most consistent associations with pollution. The effect of a warm season, April to September inclusive, and cool season on the relation of admissions to pollution was studied in single and in two pol-

Table 1 Number of admissions per day for cardiovascular diagnoses in London 1987–94 (selected percentiles and mean)

ICD-9 Code	Diagnosis	Median	10%	90%	Max	Mean
410	Acute myocardial infarction	26	17	36	60	26.7
413	Angina pectoris	15	7	24	36	15.4
414	Other ischaemic heart disease	10	5	15	26	10.0
427	Cardiac dysrhythmia	11	5	19	36	11.9
428	Heart failure	24	13	38	64	24.6
430–8	Cerebrovascular	28	17	39	59	28.2
Other	Other circulatory	28	14	46	70	29.5
390–459	Circulatory system	144	90	200	253	145.1

Table 2 Daily environmental measurements in London, England 1987-94 (selected percentiles)

	Days	Min	10%	Median	90%	Max
Temperature (°C, midrange)	2537	-5	9	12	19	29
Relative humidity (%; average of 0600 and 1500)	2540	40	58	72	86	97
O ₃ (ppb, hourly mean 0900 to 1700)	2378	0	3	13	28	94
NO ₂ (ppb, 24 hourly mean)	2494	8	23	35	53	198
SO ₂ (ppb, 24 hourly mean)	2316	0	2	6	21	114
CO (ppm, 24 hourly mean)	1931	0.2	0.6	0.9	1.8	10
Black smoke (µg/m ³ , 24 hourly mean)	2553	1	7	12	22	62

lutant models. Attributable risk was calculated by the method of Morris *et al.*¹³

Results

In London there were 373 556 emergency admissions with circulatory diagnoses reported from 1 April 1987 to 31 March 1994 with a daily median of 145. For the diagnostic subgroups the medians ranged from 10 to 28 a day (table 1). The modelling of long term fluctuations resulted in a variable number of pairs of cyclical terms from two (ICD-9 414) to 83 (other circulatory). These differences were small compared with the residual degrees of freedom, which was usually over 2000 (minimum 1756).

Table 3 Poisson model results for significance of association of emergency admissions with previous day's air pollutant (% increase in admissions/unit of pollutant, and relative risk for an increase in concentration from the 10th to 90th percentile (RR80))

	P Value	Admissions %/Unit	Relative risk (RR80) (95% CI)
Acute myocardial infarction ICD-9 410:			
O ₃	0.2638	-0.07	0.9825 (0.9534 to 1.0142)
NO ₂	0.0024*	0.09	1.0274 (1.0084 to 1.0479)
SO ₂	0.0006*	0.17	1.0326 (1.0133 to 1.0511)
CO	0.0010*	2.26	1.0275 (1.0096 to 1.0457)
Black smoke	0.0033*	0.19	1.0303 (1.0092 to 1.0528)
Angina pectoris ICD-9 413:			
O ₃	0.4887	-0.06	0.9850 (0.9469 to 1.0256)
NO ₂	0.0948	0.07	1.0212 (0.9950 to 1.0457)
SO ₂	0.2291	0.07	1.0133 (0.9907 to 1.0383)
CO	0.2202	1.15	1.0140 (0.9904 to 1.0381)
Black smoke	0.0220*	0.19	1.0303 (1.0030 to 1.0593)
Other ischaemic heart disease ICD-9 414:			
O ₃	0.2230	-0.11	0.9726 (0.9270 to 1.0214)
NO ₂	0.8019	-0.01	0.9970 (0.9667 to 1.0289)
SO ₂	0.6495	-0.03	0.9944 (0.9651 to 1.0239)
CO	0.2676	-1.07	0.9870 (0.9588 to 1.0158)
Black smoke	0.1074	-0.16	0.9752 (0.9418 to 1.0090)
Cardiac arrhythmia ICD-9 427:			
O ₃	0.5562	0.06	1.0153 (0.9704 to 1.0598)
NO ₂	0.0382*	0.09	1.0274 (1.0006 to 1.0984)
SO ₂	0.2024	0.10	1.0181 (1.0000 to 1.0448)
CO	0.0755	0.16	1.0020 (0.9997 to 1.0043)
Black smoke	0.0755	0.16	1.0254 (0.9958 to 1.0570)
Heart failure ICD-9 428:			
O ₃	0.6708	-0.02	0.9940 (0.9604 to 1.0287)
NO ₂	0.8311	-0.01	0.9970 (0.9769 to 1.0194)
SO ₂	0.5194	0.03	1.0057 (0.9846 to 1.0258)
CO	0.2780	0.69	1.0084 (0.9885 to 1.0287)
Black smoke	0.4463	0.05	1.0079 (0.9846 to 1.0309)
Cerebrovascular diseases ICD-9 430-8:			
O ₃	0.3055	-0.06	0.9850 (0.9560 to 1.0127)
NO ₂	0.1234	-0.05	0.9851 (0.9684 to 1.0045)
SO ₂	0.9146	0.01	1.0019 (0.9837 to 1.0189)
CO	0.3667	-0.66	0.9920 (0.9735 to 1.0091)
Black smoke	0.7972	-0.02	0.9969 (0.9772 to 1.0176)
Other circulatory diseases:			
O ₃	0.4496	-0.04	0.9900 (0.9614 to 1.0220)
NO ₂	0.1763	0.06	1.0182 (1.0000 to 1.0398)
SO ₂	0.0621	0.10	1.0190 (0.9994 to 1.0383)
CO	0.0040*	2.56	1.0313 (1.0126 to 1.0503)
Black smoke	0.1130	0.10	1.0158 (0.9942 to 1.0386)
Combined circulatory diseases ICD-9 390-459:			
O ₃	0.0686	-0.11	0.9726 (0.9436 to 1.0046)
NO ₂	0.0034*	0.08	1.0243 (1.0054 to 1.0448)
SO ₂	0.0031*	0.13	1.0248 (1.0062 to 1.0444)
CO	0.0012*	2.31	1.0281 (1.0100 to 1.0466)
Black smoke	0.0083*	0.15	1.0238 (1.0028 to 1.0464)

*P<0.05. See table 2 for units.

There were fewer data for CO than for the other pollutants (table 2). Correlations between pollutants (not shown) were all significant at the P<0.0001 level, except for the correlation of O₃ and NO₂ which was not significant. O₃ was negatively correlated with the other pollutants, although all other pairwise correlations of pollutants were positive. The largest correlation was between black smoke and CO, 0.73, despite these being measured at different sites.

SINGLE POLLUTANT POISSON MODELS

Carbon monoxide was significantly associated with combined circulatory diseases, and for the diagnostic subgroups other circulatory and acute myocardial infarction (table 3). Sulphur dioxide was significantly related to all circulatory diseases, and to acute myocardial infarction. Nitrogen dioxide was significantly associated with all circulatory diseases, arrhythmia, and acute myocardial infarction. Black smoke was significantly related to all circulatory diseases, angina, and acute myocardial infarction. O₃ was negatively associated with hospital admission in all subgroups except for arrhythmia, but not significantly so (P>0.22). All

circulatory diseases approached a negative association with O_3 , but it was not significant ($P>0.06$).

The results were not significant (range of P values from table 3) in any of the five single pollutant models for heart failure ($0.27<P<0.84$), cerebrovascular disease ($0.12<P<0.92$), and other ischaemic heart disease ($0.10<P<0.81$). Arrhythmias were associated only with NO_2 ($P=0.04$), angina was associated only with black smoke ($P=0.02$), and other circulatory disease was associated only with CO ($P=0.004$).

Acute myocardial infarction alone among the diagnostic subgroups was associated with more than one pollutant.

Table 4 Acute myocardial infarction: one and two pollutant models with cool and warm season (diagonal elements (bold) are single pollutant models, off diagonal elements are test pollutant modelled with a second pollutant)

Test pollutant	Second pollutant				
	O_3	NO_2	SO_2	CO	Black smoke
O_3 (ppb):					
Cool season:					
P value	0.22	0.72	0.91	0.93	0.95
Coefficient	-0.0013	0.0004	0.0001	-0.0001	0.0001
Lower 95%	-0.0033	-0.0019	-0.0021	-0.0027	-0.0021
Upper 95%	0.0008	0.0027	0.0024	0.0024	0.0023
Warm season:					
P value	0.48	0.39	0.26	0.34	0.34
Coefficient	-0.0005	-0.0006	-0.0009	-0.0009	-0.0007
Lower 95%	-0.0018	-0.0020	-0.0024	-0.0025	-0.0020
Upper 95%	0.0009	0.0008	0.0006	0.0009	0.0007
NO_2 (ppb):					
Cool season:					
P value	0.0004*	0.0020*	0.15	0.84	0.23
Coefficient	0.0022	0.0013	0.0008	0.0002	0.0008
Lower 95%	0.0010	0.0005	-0.0003	-0.0015	-0.0005
Upper 95%	0.0034	0.0022	0.0018	0.0019	0.0021
Warm season:					
P value	0.49	0.53	0.65	0.90	0.63
Coefficient	0.0004	0.0003	-0.0003	0.0001	0.0003
Lower 95%	-0.0007	-0.0007	-0.0018	-0.0016	-0.0009
Upper 95%	0.0014	0.0014	0.0011	0.0018	0.0016
SO_2 (ppb):					
Cool season:					
P value	0.0005*	0.03*	0.0004*	0.02*	0.03*
Coefficient	0.0025	0.0015	0.0021	0.0020	0.0015
Lower 95%	0.0011	0.0001	0.0009	0.0003	0.0001
Upper 95%	0.0039	0.0030	0.0033	0.0037	0.0029
Warm season:					
P value	0.70	0.49	0.53	0.32	0.58
Coefficient	0.0004	0.0009	0.0006	0.0013	0.0006
Lower 95%	-0.0016	-0.0016	-0.0013	-0.0013	-0.0014
Upper 95%	0.0024	0.0034	0.0025	0.0039	0.0026
CO (ppb):					
Cool season:					
P value	0.001*	0.15	0.39	0.02*	0.38
Coefficient	0.0324	0.0205	0.0083	0.0227	0.0100
Lower 95%	0.0127	-0.0074	-0.0107	0.0074	-0.0121
Upper 95%	0.0520	0.0483	0.0273	0.0380	0.0321
Warm season:					
P value	0.63	0.63	0.55	0.40	0.39
Coefficient	0.0112	0.0154	-0.0160	0.0187	0.0243
Lower 95%	-0.0348	-0.0477	-0.0683	-0.0250	-0.0308
Upper 95%	0.0572	0.0786	0.0363	0.0625	0.0794
Black smoke ($\mu\text{g}/\text{m}^3$):					
Cool season:					
P value	0.0006*	0.23	0.10	0.13	0.002*
Coefficient	0.0033	0.0014	0.0015	0.0019	0.0024
Lower 95%	0.0014	-0.0009	-0.0003	-0.0006	0.0009
Upper 95%	0.0051	0.0037	0.0033	0.0043	0.0039
Warm season:					
P value	0.84	0.96	0.86	0.79	0.82
Coefficient	-0.0003	0.0001	0.0003	-0.0006	0.0003
Lower 95%	-0.0033	-0.0036	-0.0031	-0.0050	-0.0026
Upper 95%	0.0027	0.0037	0.0037	0.0038	0.0033

* $P<0.05$.

Cool season is October to March, warm season is April to September. Coefficient = Poisson regression coefficient—for example, % admissions per unit of pollutant = $(\exp(\text{coeff})-1) \times 100$.

SINGLE POLLUTANT PARTIAL MODELS

Significant associations found with a Poisson model were tested also in the partial model—that is, without sine and cosine terms and without the Poisson assumption. Only the associations with acute myocardial infarction were significant in both models. For CO the associations became undetectable with all circulatory diseases, $P=0.61$, and with other circulatory disease, $P=0.77$ (the direction of this association was reversed) whereas the association with acute myocardial infarction was consistent ($P=0.0005$). For SO_2 the association with all circulatory diseases was reversed ($P=0.54$), whereas an association with acute myocardial infarction in the same direction was detected ($P=0.01$). For NO_2 the associations with all circulatory diseases ($P=0.22$) and arrhythmia ($P=0.12$) were not detectable, but the association with acute myocardial infarction was evident ($P=0.002$). For black smoke the associations with all circulatory diseases ($P=0.23$) and angina ($P=0.37$) were not detectable, but for acute myocardial infarction the association was again evident ($P=0.002$).

The attributable numbers of cases of acute myocardial infarction were calculated for each day from the full Poisson model's fitted number of admissions under all the observed conditions including the level of pollutant for that day, and then changing pollutant to the minimum concentration (table 2).¹³ The sum of the differences converted to a percentage of admissions (95% confidence limits (95% CIs)) were: O_3 -1.0% (-2.8% to 0.8%), SO_2 1.7% (0.6 to 2.6), CO 2.1% (0.7 to 3.5), black smoke 2.5% (0.8 to 4.3), NO_2 2.7% (0.8 to 4.6).

ACUTE MYOCARDIAL INFARCTION, SEASON, AND TWO POLLUTANT MODELS

As acute myocardial infarction was the only diagnosis for which the results were consistent between full and partial models, we examined for acute myocardial infarction only the influence of a cool (October to November) and warm season (April to September). Single pollutant and then two pollutant Poisson models were used. In the single pollutant models, as before, all four pollutants other than O_3 were significant; however, they were only significant in the cool season and not in the warm season (table 4). In two pollutant models, again O_3 was not significant. All the other pollutants were significant in combination with O_3 , but in the cool season only. Only SO_2 was significant with a pollutant other than O_3 . SO_2 was significant with all the other pollutants (NO_2 $P=0.033$, CO $P=0.021$, black smoke $P=0.031$), but again only in the cool season.

Discussion

The evidence from previous studies for a consistent association between O_3 and circulatory disease is weak, and for a positive association it is weaker still. In the present data, there is a complete absence of an association with O_3 , compatible with cardiovascular toxicity, and there is even a suggestion of a negative associ-

ation. These findings are in agreement with those of Schwartz and Morris¹⁴ who did not find an association with hospital admissions for people over 64 years old for ischaemic heart disease, heart failure, or arrhythmia, despite looking for an association with concentrations of O_3 on the same day and up to two days before, adjusting for a warm season, and extensive modelling of temperature and humidity. No association of O_3 on the same day and lagged by one and two days with all year circulatory deaths in London was found for 1987–92: for the warm season alone there was a significant positive association at lag 0, whereas the results for the few observations in the cool season were consistent with an effect of equal magnitude but in the opposite direction.⁸ Cardiovascular deaths in Lyon, France, were also not associated with O_3 (estimated relative risk of 1).¹⁵ Admissions for heart failure under Medicare and O_3 have been analysed from seven cities in the United States: two cities had positive associations, four had negative, and one had neutral; only the associations for New York and Los Angeles were significant ($P < 0.05$), and these two results were in opposite directions.¹³

The balance of evidence for an association of black smoke with circulatory deaths is not strong, particularly in London, and this weakens the case for an association with acute myocardial infarction, which has a high immediate mortality. Black smoke and circulatory deaths were not associated in London for all year, cool season, or warm season.⁸ In Lyon there was also no significant association with particulate matter with an aerodiameter of $< 13 \mu m$ (PM13),¹⁵ and in Athens none for black smoke.¹⁶ On the other hand there were significant positive associations of deaths in Philadelphia and Cincinnati with total suspended particles^{17,18} and in Birmingham, United Kingdom with PM10.¹⁹ Cardiovascular morbidity, rather than mortality, has perhaps more consistent associations with particles. In Detroit, PM10 was associated with ischaemic heart disease in the > 64 year age group, and in our London data with acute myocardial infarc-

tion, angina, and all circulatory diseases, but in Birmingham there was no association with admissions for ischaemic heart disease.¹⁹ An association with heart failure has been reported for elderly people,¹⁴ but there was none in our data for all ages combined. In Beijing there was no association between mortality from pulmonary heart disease and total suspended particles.²⁰

We found an association between SO_2 and acute myocardial infarction and all circulatory diseases. The partial model association with all circulatory diseases was, however, not significant and in the opposite direction, whereas it was significant and in the same direction for acute myocardial infarction. Again mortality from circulatory disease in London did not reflect this association either for all year, or cool, or warm seasons.⁸ In Lyon¹⁵ and Beijing,²⁰ however, associations have been found; but not in Athens,¹⁶ or two other French cities despite examining at least 10 different lags.²¹

Associations of CO with heart failure in elderly people were found in several cities in the United States,¹³ and with ischaemic heart disease in Detroit,¹⁴ but were not apparent in our data. There is one previous report of associated mortality, which is based on one year's data in elderly people of Sao Paulo.²²

In the present data, only associations with acute myocardial infarction were supported by significant associations in the partial model: black smoke $P = 0.002$, NO_2 $P = 0.002$, SO_2 $P = 0.01$, CO $P = 0.0005$. There are published accounts from many cities of a lack of association of air pollution with cardiovascular deaths. Our data on all circulatory diseases and diagnostic subgroups, other than acute myocardial infarction, are consistent with those conclusions. For acute myocardial infarction, however, our positive associations for black smoke, NO_2 , and SO_2 with emergency admissions are hard to reconcile with a lack of association with circulatory deaths,⁸ although for acute myocardial infarction the time to emergency hospital admission compared with the time to death is likely to be less variable in relation to a provocation—such as air pollution—and is probably therefore a more sensitive outcome.

Significant associations might, however, arise solely because factors that affect the clearance and creation of air pollution—such as the weather and driving motor vehicles—also have a direct effect on human behaviour and on acute health outcomes (figure). Although we have modelled temperature and humidity, there are other factors—for example, wind and rain—which affect air pollution and also affect the amount of physical exercise taken and the duration of time spent outdoors, where levels of air pollution are generally different from indoors. With so much potential for confounding, and with a good many conflicting results to be found in the scientific literature, we chose to place the statistical emphasis on the direction and consistency of the relations (P values) rather than on estimat-

Short term relation between outdoor metered air pollution and health.

ing the size of effects from the models which produce the largest effects, as has usually been done.

The present results for CO taken with the dearth of published studies relating it to cardiovascular disease in general and acute myocardial infarction in particular make a case for further study of CO. Average concentrations over one hour at a kerbside monitor can reach 25 ppm.²³ Breathing CO at 50 ppm for one hour increased carboxyhaemoglobin by a factor of two, and reduced symptom limited exercise capacity in patients with stable angina pectoris as reviewed by Strom *et al.*²⁴ Thus the risk of myocardial ischaemia when pushing a lawn mower or shovelling snow²⁵ is increased by the proximity of an internal combustion engine. We have shown that fluctuations in background concentrations of CO are an additional risk factor for myocardial infarction in the population, and this effect could be due to an increased risk of infarction during exercise when environmental concentrations of CO have risen within the preceding 24 hours.

All the results for O₃ were not significant. For the other pollutants the only consistent associations were with acute myocardial infarction. Biologically plausible mechanisms have been advanced by which emissions, mainly from motor vehicles, might trigger circulatory disorders. Our results strengthen the case for the existence of a causal link with myocardial infarction.

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