Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China

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Objective: To investigate the association between ambient concentrations of air pollutants and respiratory and cardiovascular mortalities in Hong Kong.

Methods: Retrospective ecological study. A Poisson regression of concentrations of daily air pollutants on daily mortalities for respiratory and cardiovascular diseases in Hong Kong from 1995 to the end of 1998 was performed using the air pollution and health: the European approach (APHEA) protocol. The effects of time trend, seasonal variations, temperature, and humidity were adjusted. Autocorrelation and overdispersion were corrected. Daily concentrations of nitrogen dioxide (NO₂), sulphur dioxide (SO₂), ozone (O₃), and particulate matter <10 µm in aerodynamic diameter (PM₁₀) were averaged from eight monitoring stations in Hong Kong. Relative risks (RRs) of respiratory and cardiovascular mortalities (per 10 µg/m³ increase in air pollutant concentration) were calculated.

Results: Significant associations were found between mortalities for all respiratory diseases and ischaemic heart diseases (IHD) and the concentrations of all pollutants when analysed singly. The RRs for all respiratory mortalities (for a 10 µg/m³ increase in the concentration of a pollutant) ranged from 1.008 (for PM₁₀) to 1.015 (for SO₂) and were higher for chronic obstructive pulmonary diseases (COPD) with all pollutants except SO₂, ranging from 1.017 (for PM₁₀) to 1.034 (for O₃). RRs for IHD ranged from 1.009 (for O₃) to 1.028 (for SO₂). In a multipollutant model, O₃ and SO₂ were significantly associated with all respiratory mortalities, whereas NO₂ was associated with mortality from IHD. No interactions were detected between any of the pollutants or with the winter season. A dose-response effect was evident for all air pollutants. Harvesting was not found in the short term.

Conclusions: Mortality risks were detected at current ambient concentrations of air pollutants. The associations with the particulates and some gaseous pollutants when analysed singly were consistent with many reported in temperate countries. PM₁₀ was not associated with respiratory or cardiovascular mortalities in multipollutant analyses.

Many time series studies on the effects of variations in daily air pollutants on mortalities and morbidities have been reported in the United States and in Europe.^{1–6} The role of particulates has been the focus of the research, and the associations have been found between total suspended particulates and particulates less than 10 µm in aerodynamic diameter (PM₁₀) and all mortalities and those caused by respiratory and cardiovascular diseases.^{2–3, 5–11} Associations have also been reported with gaseous air pollutants—namely, ozone (O₃),^{4, 12} nitrogen dioxide (NO₂),⁴ sulphur dioxide (SO₂),^{5, 10, 13, 14} and carbon monoxide (CO).^{14, 15} Compared with the particulates, the relations between gaseous pollutants and mortalities are less consistent. Mortality has been shown to be associated with climate,¹⁶ and its interaction with air pollutants have been reported in several European countries.^{15, 17} In Asia, few such studies have been conducted except in northern China (Beijing) and Korea.^{7, 18, 19} The relations between seasons and air pollutants are likely to differ in tropical climates. Studies from tropical countries are scarce, possibly due to the lack of credible data. Hong Kong is a densely populated coastal city in southern China with 6.8 million people in a land area of about 1000 km². Summers are hot and humid, whereas winters are typically mild and dry. A large proportion of the population live in high rise buildings in close proximity to road traffic, a major source of air pollutants. Diesel vehicles contribute a substantial proportion of respirable particulates. Concentrations of PM₁₀, NO₂, and O₃ are higher than those in major cities in the United States, Western Europe, and some Asian countries such as Singapore and Japan, whereas SO₂ and CO concentrations are comparatively low.²⁰ Air

pollutants are monitored systematically over most districts. The methods and quality of data are comparable with standards in many developed countries. A vigorous validation procedure of the air pollutant measurements is followed. Mortality statistics are comprehensive and systematically validated by the health authority. Owing to the relatively high concentrations of particulates and the oxidant pollutants (NO₂ and O₃) in Hong Kong and the proximity of the residences to the pollution source compared with western cities, an investigation into the association between the air pollutants and health outcomes is warranted. We have reported an association between daily hospital admissions and air pollutants.²¹ To investigate the association between air pollution and mortality, and to compare the results with our previous findings and those reported elsewhere, we performed a time series analysis on concentrations of air pollutants and daily mortality data for respiratory and cardiovascular diseases over a 4 year period from 1995–8.

MATERIALS AND METHODS

Mortality data

Daily mortality data between 1995 and 1998 were obtained from the Census and Statistics Department. Mortality data covered all deaths reported in Hong Kong, and were coded

Abbreviations: APHEA, air pollution and health; the European approach; PM₁₀, particulate matter <10 µm in aerodynamic diameter; IHD, ischaemic heart diseases; COPD, chronic obstructive pulmonary diseases

according to the 9th revision of the international classification of diseases (ICD). Daily time series datasets were constructed for mortalities from "all diseases of the respiratory system" (ICD 461-519) and its subsets, chronic obstructive pulmonary diseases (COPD, ICD 490-496) and pneumonia and influenza (ICD 480-7). Daily datasets were also constructed for mortalities from "all diseases of the cardiovascular system" (ICD 390-459) and its subsets, ischaemic heart disease (IHD, ICD 410-414) and cerebrovascular disease (ICD 430-8).

Air quality and weather data

Air pollution data between 1995 and 1998 were obtained from the Environmental Protection Department. Hourly concentrations of four air pollutants: SO_2 , NO_2 , PM_{10} , and O_3 were monitored in eight monitoring stations interspersed in different districts of Hong Kong using pulsed fluorescence, gas phase chemiluminescence, tapered element oscillating microbalance, and ultraviolet absorption, respectively.²⁰ Twenty four hour mean concentrations of NO_2 , PM_{10} , and SO_2 were calculated. As the formation of O_3 is dependent on sunlight, a daytime (0900–1700) 8 hour mean concentration of O_3 was used for analysis.²² In view of the low ambient concentration of CO in Hong Kong, monitoring of CO has been confined to only one station in recent years. We have therefore excluded CO from our study. Daily mean temperatures and relative humidity for the same period were obtained from the Hong Kong Observatory.

Statistical analysis

A Poisson regression model was constructed in accordance with the air pollution and health: the European approach (APHEA) protocol.²² The following terms were included to construct the core model: day of the time series (t), days of the week, trigonometric functions to control for seasonal variations ($\sin 2k\pi t/365$ and $\cos 2k\pi t/365$, where $k=1, 2, 3, 4$, and 6 , represent cycles of 12, 6, 4, 3, and 2 months respectively), temperature and humidity.^{21–23} To control for overdispersion, the covariance matrix was modified by multiplying the dispersion parameter ϕ , and the scaled deviance and log likelihoods used in likelihood ratio tests were divided by ϕ . The function obtained by dividing a log likelihood for the Poisson distribution by a dispersion parameter is an example of a quasi-likelihood function.^{24–25} To control for autocorrelation, the autocorrelation functions plot of the residuals was examined and significant terms, up to lag day 7, were retained in the model.^{23–26} Different pollutants may affect mortality with variable time lags. To test the influence of an individual pollutant on mortality, a single pollutant model was constructed for each of the four pollutants by adding its daily concentration to the core model. The best fitting lag period of each pollutant was found by testing its concentration on single

lag days and on cumulative lag days (moving averages). Lag days from 0 (same day) to 5 (5 days before) were tested for O_3 ; lag days from 0 to 3 were tested for the other three pollutants. The lag period used to construct the model that gave the smallest Akaike's information criterion value was then selected for each pollutant.²⁷ The relative risk (RR) and its 95% confidence intervals (95% CIs) for a $10 \mu\text{g}/\text{m}^3$ increase in the concentration of each pollutant were then calculated. To study the combined effects of the pollutants, multipollutant models were constructed. Firstly, all four pollutants (as continuous variables) were entered into the core model irrespective of significance. Non-significant ($p \geq 0.05$) terms were then successively removed from the model by a process of backward elimination, until only the significant terms remained. Owing to the non-trivial correlation of some pollutants, a pairwise approach was adopted as in the APHEA protocol if more than one pollutant seemed to be associated with the outcome, the associations with one pollutant stratified by the level of the other pollutant was sought.²² With this approach, we explored the interaction between different pollutants by analysing each pair of pollutants using one as a continuous variable and the other as high and low concentrations dichotomised by the median, and their interaction term (between a pollutant and a high concentration of another pollutant). The influence of the concentration of a pollutant (expressed as a continuous variable) on a high and low concentration of another pollutant was then studied.^{22–23} Interaction was considered to be present when the p value of the interaction term was less than 0.05. As an ambient temperature has a significant influence on mortality,¹⁶ we also explored the possible interaction of cold seasons (December to February, when the mean monthly temperature was below 20°C) with the effects of the air pollutants by introducing a dummy variable for season in the model.²² To investigate dose-response relations, we derived from the single pollutant models the respective RRs of daily deaths by pollutant concentrations categorised into deciles with the lowest decile as reference.²

Harvesting, a hypothesis that a high death toll on one day due to air pollution would deplete the pool of vulnerable people and result in fewer deaths on succeeding days, was investigated according to the procedure described by Spix *et al.*²⁸ In short, the coefficients of the interaction between the mean daily mortalities on the previous k days (where k ranged from 2 to 21) and each of the pollutants were checked. Harvesting was considered to be present if any of the coefficients was significantly negative.

RESULTS

During the 4 years, there were 128 229 deaths of which 58 347 (46%) were caused by respiratory and circulatory diseases. Table 1 shows the daily number of deaths by cause and the

Table 1 Summary statistics of daily numbers of deaths, concentrations of pollutants ($\mu\text{g}/\text{m}^3$), and weather data

	Mean	SD	Min	25%	Median	75%	Max
Causes of death (n):							
All respiratory diseases	17	5	3	13	17	20	33
Chronic obstructive pulmonary diseases	6	32	0	4	5	7	19
Pneumonia and influenza	10	4	1	8	10	13	24
All cardiovascular diseases	23	7	7	19	22	27	53
Ischaemic heart disease	9	3	1	6	9	11	29
Cerebrovascular disease	9	3	0	6	8	11	22
Air pollutants ($\mu\text{g}/\text{m}^3$):							
NO_2	56.40	19.24	15.29	43.29	54.19	68.10	151.5
PM_{10}	51.53	24.79	14.05	31.58	45.90	66.51	163.79
O_3	33.93	23.15	0.3	15.92	29.29	49.67	168.93
SO_2	16.68	11.59	1.05	8.58	13.97	21.27	90.06
Weather variables							
Temperature	23.4	5.0	6.9	19.2	24.6	27.6	31.3
Humidity	78.1	10.4	31	74	79	85	97

Table 2 Correlation between pollutants and weather variables

	SO ₂	NO ₂	PM ₁₀	O ₃	Temperature	Humidity
SO ₂	1	0.438	0.344	-0.073	0.186	-0.089
NO ₂		1	0.780	0.413	-0.346	-0.341
PM ₁₀			1	0.538	-0.319	-0.524
O ₃				1	-0.045	-0.530
Temperature					1	0.227
Humidity						1

pollutant concentrations. The Pearson's correlation coefficients between air pollutants and meteorological variables are presented in table 2. After fitting the core model, residuals were plotted against the predicted values for diagnostic checking. No cyclical pattern could be discerned in the residual plot. Table 3 summarises the RRs of daily deaths for different disease categories. The lag days for the pollutants that fitted the model best for all respiratory diseases varied for different pollutants, ranging from 1 to 2 single lag days and 0 to 1 cumulative lag days. For all cardiovascular diseases, the "best lag" ranged from 0 to 2 single lag days and up to 0 to 2 cumulative lag days. The overdispersion parameter (ϕ) was 1.14 and 1.09 respectively for respiratory and cardiovascular diseases. The autocorrelation coefficients (r) of the models ranged from 0.005 to 0.052. For all four pollutants, there was a significant increase in mortality for respiratory diseases that ranged from 0.8% to 1.5% per 10 $\mu\text{g}/\text{m}^3$ increase in concentration of pollutant. For COPD, the risks were higher and significant for all pollutants except SO₂. For pneumonia and influenza, the RRs were significant for NO₂ and SO₂ only. For all cardiovascular diseases and cerebrovascular diseases, the RRs for the best fitting lag days of all four pollutants were non-significant. A significant increase in mortality from IHD, ranging from 0.9% to 2.8%, was associated with a 10 $\mu\text{g}/\text{m}^3$ increase in the concentration of all four pollutants.

In the multipollutant model for all respiratory mortalities, SO₂ (RR=1.015) and O₃ (RR=1.010) remained after eliminating the non-significant pollutants (table 4). In the three and four pollutant models, O₃ was the only significant pollutant. The RR of O₃ was stable in all three models, and similar in magnitude to that in the single pollutant model. For COPD, O₃ was the only significant pollutant in the two, three and four pollutant models, with RRs slightly lower than in the single pollutant model. For pneumonia and influenza, SO₂ was significant after eliminating non-significant terms from the model. For IHD mortalities, NO₂ was the only significant pollutant in the four, three, and two pollutant models, its RRs being smaller than that in the single pollutant model.

No significant interaction between any pair of pollutants was found in the pairwise analyses, or between any pollutant and the cold season. None of the coefficients of the interaction terms between the pollution concentration and the mean mortalities of the previous days from lag day 2 up to day 21 were significantly negative, suggesting the absence of a harvesting effect. When RRs of deciles of the pollutant concentrations were derived from each single pollutant model, with the lowest decile as reference, an upward trend in RRs was found for all pollutants from the lowest to the highest deciles (fig 1).

Table 3 RRs (95% CIs)/10 $\mu\text{g}/\text{m}^3$ increase in concentration of pollutant for daily numbers of deaths from different diseases by pollutants

	SO ₂	O ₃	NO ₂	PM ₁₀
Respiratory diseases	Lag 0–1 days	Lag 2 days	Lag 0–1 days	Lag 1 day
RR	1.015	1.010	1.013	1.008
95% CI	(1.001 to 1.029)	(1.004 to 1.016)	(1.004 to 1.022)	(1.001 to 1.014)
Chronic obstructive pulmonary diseases	Lag 2 days	Lag 0–4 days	Lag 0–2 days	Lag 0–3 days
RR	1.010	1.034	1.023	1.017
95% CI	(0.990 to 1.029)	(1.017 to 1.052)	(1.006 to 1.041)	(1.002 to 1.033)
Pneumonia and influenza	Lag 0–1 days	Lag 2 days	Lag 0–3 days	Lag 2 days
RR	1.021	1.007	1.016	1.007
95% CI	(1.003 to 1.039)	(0.999 to 1.015)	(1.002 to 1.030)	(0.999 to 1.015)
Cardiovascular diseases	Lag 0–1 days	Lag 0 day	Lag 0–2 days	Lag 2 days
RR	1.007	0.997	1.008	1.003
95% CI	(0.994 to 1.020)	(0.991 to 1.003)	(0.999 to 1.016)	(0.998 to 1.008)
Ischaemic heart disease	Lag 1 day	Lag 3 days	Lag 1 day	Lag 0–3 days
RR	1.028	1.009	1.024	1.013
95% CI	(1.012 to 1.044)	(1.000 to 1.018)	(1.012 to 1.036)	(1.001 to 1.025)
Cerebrovascular disease	Lag 2 days	Lag 0 day	Lag 1 day	Lag 2 days
RR	0.9881	0.999	0.996	1.007
95% CI	(0.965 to 1.011)	(0.998 to 1.000)	(0.985 to 1.007)	(0.998 to 1.016)

Table 4 RRs (95% CIs) for daily numbers of deaths/10 $\mu\text{g}/\text{m}^3$ increase in concentration of pollutant in multipollutant models*

Model	SO ₂	O ₃	NO ₂	PM ₁₀
All respiratory mortalities:				
4 Pollutant model	1.009 (0.990 to 1.029)	1.009 (1.002 to 1.016)	1.001 (0.991 to 1.020)	1.005 (0.992 to 1.010)
3 Pollutant model	1.010 (0.990 to 1.030)	1.009 (1.002 to 1.016)	1.006 (0.993 to 1.019)	
2 Pollutant model	1.015 (1.001 to 1.031)	1.010 (1.003 to 1.017)		
Mortality from COPD:				
4 Pollutant model	1.007 (0.9848 to 1.0296)	1.033 (1.0126 to 1.0548)	1.014 (0.989 to 1.041)	0.991 (0.968 to 1.015)
3 Pollutant model		1.032 (1.012 to 1.053)	1.016 (0.991 to 1.042)	0.993 (0.970 to 1.016)
2 Pollutant model*		1.029 (1.011 to 1.049)	1.011 (0.992 to 1.031)	
Mortality from pneumonia and influenza:				
4 Pollutant model	1.018 (0.997 to 1.040)	1.006 (0.997 to 1.015)	1.004 (1.017 to 1.025)	1.002 (0.991 to 1.013)
3 Pollutant model	1.017 (0.996 to 1.039)	1.006 (0.997 to 1.016)	1.006 (0.989 to 1.024)	
2 Pollutant model	1.021 (1.003 to 1.039)	1.008 (0.999 to 1.016)		
Mortality from ischaemic heart diseases:				
4 Pollutant model	1.011 (0.990 to 1.033)	1.007 (0.998 to 1.017)	1.020 (1.003 to 1.035)	0.9940 (0.978 to 1.009)
3 Pollutant model	1.012 (0.991 to 1.033)	1.006 (0.998 to 1.015)	1.016 (1.002 to 1.032)	
2 Pollutant model		1.006 (0.997 to 1.014)	1.022 (1.011 to 1.033)	

*Backward elimination of non-significant terms ($p>0.05$) starting with four pollutants.

DISCUSSION

This study provides additional information for our previous study on hospital admissions,²¹ and the many time series studies on air pollution and mortality in temperate countries.^{1-11 13 15 17-19 28 29 33-35 38 39} Although hospital admissions are influenced by socioeconomic and personal factors, mortality is the ultimate and most serious of all health outcomes. As explained earlier, both the mortality and air pollutant datasets were of reasonable quality. Our statistical methods followed the APHEA protocol, which facilitated the comparison of results. Significant associations were found with respiratory mortalities for all four pollutants in the single pollutant models. For the different respiratory diseases, the best lags ranged from 1 to 2 single lag days and 0 to 4 cumulative lag days. These lag periods were statistically chosen by model fitness. The durations of the lag periods, however, seemed reasonable, if we assume a short interval (in terms of

several days) between exposure and death, which varies with different diseases. It is also reasonable to assume that mortality might be associated with cumulative exposure over several days. The risk estimates for respiratory mortalities, at 0.8% to 1.5% per $10\mu\text{g}/\text{m}^3$ increase in pollutant concentration were similar in magnitude to results reported elsewhere.³⁰ These mortality risks were, however, lower than risks of hospital admissions we reported previously (1.3% to 2.2% per $10\mu\text{g}/\text{m}^3$ increase).²¹ Of the respiratory diseases, the RRs for mortalities from COPD, the most sensitive mortality to air pollution, were higher for O₃ and NO₂ (at increases of 3.4% and 2.3% respectively). The RRs of cardiovascular mortalities were non-significant for all four pollutants, by contrast with our previous findings for hospital admissions for cardiovascular diseases.²¹ Mortalities for IHD were significantly associated with all four pollutants, suggesting that the associations of air pollution were disease specific. Of cardiovascular diseases, the RRs of deaths from IHD (at 1.028 and 1.024 respectively) were

Figure 1 A regression line is fitted to show the upward trend of the RRs. RRs were derived from the Poisson regression models in which the concentrations of the individual pollutants were replaced by their respective deciles. See statistical analysis in the methods section.

the highest for NO₂ and SO₂. However, a direct comparison of the magnitude of the RRs among the air pollutants can be misleading, because of differences in molecular weight. By contrast with our findings, a pooled analysis among European countries showed equivocal RRs of cardiovascular and respiratory deaths for NO₂ but significant RRs for black smoke, SO₂, and O₃.²⁹ The summary RRs for SO₂ and O₃ in western European cities are similar in magnitude to ours. In Hong Kong, the mean concentrations of O₃, NO₂, and PM₁₀ are generally higher, but the concentrations of SO₂ are much lower than in European cities. Our findings of a significant RR of IHD for SO₂ suggest that the threshold has not been reached at the current SO₂ concentrations.

Owing to the high correlation between pollutants, it is not clear whether the observed effect of one pollutant in the single pollutant model represents that of an underlying pollutant. Much research focus has been given to the respirable fraction of particulates, and more recently, fine particulates (PM_{2.5}).^{31,32} In the multipollutant model for both respiratory diseases and IHD, no significant effect for PM₁₀ could be discerned. It should be noted that in some studies in the United States and Europe where the effects of particulates were significant, single pollutant models were used.^{2-6,12-15,17,29} These results agreed with that of our single pollutant model for PM₁₀. Our findings of significant associations of SO₂ and O₃ with respiratory deaths are in accord with those in many European cities and elsewhere.^{4,5,8,12,15,17,33-36} The problems of collinearity among pollutants in a multipollutant model are well recognised. In our data, the concentrations of NO₂ and PM₁₀ were highly correlated on the same days (table 2), and on the best lag days that were used in the multipollutant model ($r=0.73$). The correlation between all the other pairs of pollutants ranged from trivial to moderate (Pearson's r ranged from -0.07 to 0.45 for the best lag days). Hence, it is statistically valid to include SO₂, O₃, and either of NO₂ and PM₁₀ in the models. For all respiratory mortalities and COPD, the RR for O₃ was remarkably stable in all the models. A significant association with O₃ for respiratory mortality in our multipollutant model is biologically plausible. Firstly, O₃ is a highly toxic oxidant pollutant with known adverse effects on the respiratory system.³⁷ Secondly, the concentration of O₃ in Hong Kong is higher than in many cities in Europe and the United States, possibly related to the abundance of sunshine, even in winter months. In our earlier study on hospital admissions, O₃ was significantly associated with admissions for both respiratory and cardiovascular diseases.²¹

The high ambient concentration of NO₂ in Hong Kong is a possible explanation of our finding of a significant association with mortalities from IHD. Significant associations between NO₂ and all cardiovascular mortalities have been reported in some studies.^{4,10,13} However, the relations between NO₂ and cardiovascular mortalities were inconsistent in a meta-analysis of pooled data from four European cities.²⁹ The reasons for the lack of association of PM₁₀ with any respiratory or cardiovascular mortalities in a multipollutant model are not clear. The effects of PM₁₀ might have been masked by those of NO₂ and O₃—the concentrations of both were fairly high. Neither could we explain why SO₂, rather than PM₁₀, was associated with deaths from pneumonia and influenza, especially when the mean concentration of SO₂ was relatively low and that of PM₁₀ was fairly high. The chemical composition of particulates might be related to the effects on health. In Hong Kong, diesel vehicle exhaust is a major source of PM₁₀, although crustal dust and marine aerosols are important sources as well.²⁰

No interaction was detected in this study between any two pollutants, whether analysed in pairs or when all four pollutants were considered together. Significant interactions between particulates and SO₂ have been reported in Athens and Lyon in the APHEA mortality studies.^{15,33} The discrepancies might be due to differences in concentrations of pollutant

Key messages

- Significantly increased risks of mortality from all respiratory diseases and ischaemic heart diseases were shown at current ambient concentrations of particulates, sulphur dioxide, nitrogen dioxide, and ozone.
- The associations between mortality and particulates and some gaseous pollutants concurred with many studies in temperate countries.
- The associations were not explained by "harvesting" of vulnerable populations and the effect of air pollution was likely to be real.
- A dose-response relation between the relative risks and the concentrations of the pollutants was found for all four air pollutants.
- When all pollutants were included in the models, ozone and sulphur dioxide remained significantly associated with respiratory mortalities, whereas nitrogen dioxide was associated with mortality from ischaemic heart disease.

Policy implications

- Current air quality standards are not sufficient to protect the public from adverse health effects.
- Further control of air pollution is likely to result in health benefits.

between Hong Kong and these cities. The absence of interaction with the cold season could be related to the relatively mild winters in Hong Kong, where the mean monthly temperatures from December to February ranged from 15° to 19°C and the mean daily temperature rarely drops below 10°C. When analyzed by deciles of pollutant concentrations, a dose-response relation was found for all pollutants and respiratory mortalities, with no evidence of a threshold effect.

The role of CO on cardiovascular mortalities has been investigated elsewhere. Touloumi *et al* reported a positive association between ambient CO concentrations and daily mortality in Athens,³⁸ but this association was reduced considerably after including CO and black smoke or SO₂ in the same model. Carbon monoxide was not considered as a copollutant in other time series analyses.³⁹ Owing to the insufficiency of data, we did not evaluate the effect of CO, which might be relevant to cardiovascular mortalities. The ambient concentration of CO in Hong Kong is low. The mean 24 hour concentration was 800 µg/m³, less than one tenth of the local air quality objective of 10 000 µg/m³.²⁰

The absence of a harvesting effect of respiratory and cardiovascular mortalities within a 3 week period suggests that the shortening of life is likely to be substantial. Schwartz suggested that deaths from chronic obstructive airway diseases were mostly brought forward by a few months, whereas some (but not most) deaths from pneumonia might be brought forward by a few days.⁴⁰ We have not investigated the harvesting effect of specific causes of death on different time scales.

In conclusion, we found significant associations of all four air pollutants with mortalities from respiratory disease and IHD, the effect size being strongest for COPD. Although the multipollutant model selected SO₂ and O₃ as significant pollutants for respiratory deaths and NO₂ for deaths from ischaemic heart disease, other pollutants such as PM₁₀ might play an important part. The joint effects of the air pollutants are still poorly understood and comparative studies in cities with different pollutant profiles are warranted. The ecological design of the study precludes the inference of cause and effect.

As the current ambient concentrations of these pollutants seem to be above the threshold, some health benefits should be noticeable if air pollution is reduced. A reduction in morbidity and mortality after the implementation of an intervention programme will add evidence to the hypothesis of a causal link between air pollution and ill health.

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PostScript

LETTERS

Neurobehavioural testing in workers occupationally exposed to lead

Whether or not low to modest levels of exposure to lead have a detrimental effect on cognition is an important issue given the growing attention, for example, in the United States, that has recently been paid towards potentially revising downward the levels of lead exposure allowed in the workplace. Thus, we read with interest the meta-analysis of studies on this topic that appeared in this journal by Goodman and colleagues.¹ Unfortunately, we believe that the authors' conclusions are not valid. Specifically, the authors state that "the data available to date are inconsistent and are unable to provide adequate information on the neurobehavioural effects of exposure to moderate blood concentrations of lead". We found no direct support for this conclusion in the publication. Moreover, numerous flaws in their method limit any specific inferences that can be made. In general, we found that the meta-analysis combined evidence from studies of widely varying quality and did not account for significant confounding within and between studies. Given these and other flaws, it was predictable that the authors did not find an association between blood lead levels and neurobehavioural test scores.

Specific concerns that we had with the methods include: (1) The authors offer no evaluation of the quality of the evidence from available studies based on study design and analytical method. (2) The authors combine data from poorly done studies with data from well done studies, clouding any effects that are observable from the better conducted studies. (3) Although age and education adjustment within studies is assessed, six studies were included that did not adjust for age, and another three studies did not adjust for education. These are the two most well established predictors of neurobehavioural test scores and the most important potentially confounding variables. (4) Even among the remaining studies that did adjust for age and education, the authors do not address the confounding in the meta-analysis that is caused by variation in age and education across study populations. (5) The authors' main effect measure is an exposed versus control comparison. Among the options that could have been pursued, this is the effect measure with the lowest power. It is unable to assess a dose-effect relation, and it is also the one most prone to selection bias. (6) Relatively few of the 22 studies listed in table 2 contribute to the estimate of the effect size for each neurobehavioural outcome. Moreover, the authors do not state which studies contributed to the effect estimate.

It is important to note that several recent studies, all published before this article was accepted for publication, reported that blood lead was associated with neurobehavioural test scores in multiple cognitive domains. One study of 803 Korean lead workers is the largest study reported to date and observed consistent associations of blood lead with test scores in the domains of executive abilities,

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manual dexterity, and peripheral motor strength at blood lead levels as low as 18 µg/dl.² In another study of former organolead manufacturing workers, tibia lead was associated with test scores at cross section and with longitudinal declines in test scores.³ These findings suggest that lead may have both short term and progressive influences on neurobehavioural performance.

We elaborate on our main concerns, below.

(1) No evaluation of the quality of the evidence available from studies, and (2) Data from poorly done studies were combined with data from well done studies. It is traditional in meta-analysis to establish a priori criteria for what defines acceptable evidence from studies. The authors only had three inclusion criteria, none of which refer to the quality of the study designs, analytical method, adjustment for confounding, evaluation of bias in selection of exposed and non-exposed subjects, and other such methodological factors. There is apparently no consideration for this arguably single most important step in meta-analysis. The meta-analytical results could simply reflect wide heterogeneity in the quality of the evidence that was combined. This factor alone could account for the overall conclusion of no association.

(3) Inclusion of studies that did not control for age and education. Age and education are the two most important predictors of neurobehavioural test scores in working populations. In the absence of adjustment for these confounders there should be convincing evidence that the two groups being compared were equivalent in age and education. Eight of the included studies did not adjust for age and/or education. The authors offer no explanation for why these studies should be included in the meta-analysis.

(4) No adjustment for age, education, or lead dose differences across studies. By not adjusting for age and education differences across studies, the authors make an implicit assumption that age and education do not modify the relation between blood lead and neurobehavioural test scores. This may or may not be true. In the meta-analysis, the authors also implicitly assume a fixed difference in blood lead levels between exposed and non-exposed groups. Table 1 clearly indicates that this assumption does not hold.

(5) Reliance on exposed versus control comparisons. This is a weak test and a test

that is not germane to the conclusions that the authors make. The authors conclude that blood lead levels, that are described as "moderate" in one location in the manuscript and "low" in another, are not associated with neurobehavioural test scores. All studies included exposed workers with a range of blood lead levels, from very low to high. More appropriate approaches could have been considered, for example, only including studies that reported beta coefficients for the blood lead versus test score relation, or adjusting for mean blood lead levels in exposed and non-exposed groups.

(6) Reliance on a small number of unspecified studies for effect estimates. Table 2 of the meta-analysis reports the number of studies that were combined to derive effect estimates, but does not specify which studies were combined. This omission does not allow the reader to determine whether solid evidence was combined with more questionable evidence, or to evaluate whether any of the issues described above were germane to the effect estimates reported.

Two more concerns exist regarding the authors' treatment of the issue of cumulative versus ongoing lead exposure, as well as the identification of the source of funding for this study. In their introduction, the authors quote the review by Balbus-Kornfeld *et al*, which noted that "the current scientific evidence is flawed because of inadequate estimation of cumulative exposure to or absorption of lead ..." but fail to acknowledge this issue in the interpretation of their own meta-analysis. In fact, as has been widely reported in the literature, methods are now available to non-invasively measure bone lead levels as a reliable and accurate measure of cumulative lead dose. Several studies⁴⁻⁵ suggest that cumulative lead dose, as measured by tibia lead levels, is a very important biological marker that may be related to cognitive decrements not predicted by blood lead levels. With regard to funding, the authors note that they are mainly from the Exponent Health Group in Alexandria, Virginia, and Menlo Park, California; however, they fail to describe what motivated the study or sources of funding for the study. We believe this information would be of interest to scientists and policy makers engaged in work on this topic.

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Suicide mortality among electricians

Järholm and Stenberg¹ evaluated suicide mortality rates among electricians ("exposed to electromagnetic fields (EMFs)") and glass and wood workers ("unexposed to EMFs") in the Swedish construction industry. Standard mortality rates were lower for the two job groups compared to the Swedish general population. This is likely to be due to the healthy worker effect. The internal cohort analysis showed that electricians had a lower suicide mortality rate than glass and wood workers.

As the authors rightfully point out, these results should not be seen as evidence against the association between exposure to EMFs and suicide, in particular because no quantitative estimates of exposure were obtained to directly evaluate this association. Järholm and Stenberg cited a small measurement survey in the Swedish construction industry, which indicated that exposure levels were low and comparable between the two occupational groups. Therefore, one would not expect to see an EMF mediated increase in suicide risk among electricians compared to glass and wood workers, if an association between EMF exposure and suicide truly exists.

Järholm and Stenberg suggested that the difference in suicide rate between the two job groups was unlikely to be due to differences in socioeconomic factors, but they did not provide an alternative explanation. One possible explanation may be a healthy worker survivor effect related to employment status (for example, at time of death) within this cohort. That is, active workers may be more physically and mentally fit than those who left the industry or are unemployed, and may therefore be at lower risk of committing suicide.² A large body of literature suggests that unemployment and suicide are positively related,^{3,4} and being out of work was positively associated with suicide in the electric utility industry.⁵ Since cessation of work also leads to cessation of work related exposures, employment status may be an important potential confounder (or perhaps effect modifier) for the association between work related exposures and suicide. The lower suicide rate among electricians compared to glass and wood workers may be explained by a larger proportion of glass and wood workers with an inactive employment status at the time of death.

Although it is unlikely that consideration of employment status, if possible, would greatly alter the conclusions reached by Järholm

and Stenberg,¹ it would be informative to see its influence on the rate ratio.

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Authors' reply

We appreciate Dr Wijngaarden's interest in our report and his suggestion for understanding the differences in risk. Dr Wijngaarden suggests that difference in unemployment rate between electricians and glass worker and wood workers could be an explanation.

We have no data on employment status at time of death and can therefore not test this hypothesis. However, if employment status is an important predictor, this could explain some of the difference, as the wood workers had a different employment structure to the other groups. Electricians and glass workers have had permanent positions for a long time, while wood workers were employed for a certain project, for example, building a house, before the 1990s. When the project was finished they had to find another employer. Today, most construction workers have permanent positions in Sweden.

In our search of the literature in an attempt to understand differences in suicide rates between occupations, we found little information. This might be an important area of research in the future.

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Are incinerator workers exposed to PCDDs and PCDFs?

Kumagai and his colleagues¹ have reported that incinerator workers employed at intermittently burning incineration plants were not necessarily exposed to high concentrations of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The authors' conclusions were based on concentrations of PCDDs and PCDFs in serum samples of the workers.

I have deep concerns regarding the study methodology and results which do not consider the accumulation of PCDDs and PCDFs in the adipose tissue. PCDDs and PCDFs are organochlorines with different degrees and positions of chlorination, which determine their persistence and toxicity. They are lipophilic and difficult to metabolise, and any environmental exposure of living organisms to them results in their accumulation and persistence in fat tissues.² Meanwhile, it is feasible to use blood sera to obtain and

analyse PCDDs and PCDFs. Adipose tissue organochlorines levels have been regarded as a preferred indicator of human exposure. Levels in adipose tissue are known to be higher and more representative of the cumulative internal exposure.^{2,3} Previously, Archibeque-Engle and colleagues⁴ did not find a significant relation between serum concentration and tissue residues for organochlorine compounds. Based on the lack of correlation between adipose tissue and serum, as well as an absence of some compound residues in serum, the authors emphasised that adipose tissue should be analysed in addition to serum.

Finally, I would like to acknowledge the authors for such original subject study, which enables us to raise the profile of and discuss new hypotheses in environmental and occupational health.

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Importance of work intensity on respiratory problems in hairdressers

We read the report by Hollund *et al* with great interest.¹ We agree that there is limited information about the prevalence of airway symptoms caused by highly reactive chemicals in hairdressing salons. In this well designed study, authors focused on age as a risk factor and observed an increased prevalence of respiratory symptoms among the oldest and youngest hairdressers and observed more symptoms among hairdressers over 40 years of age.

Work intensity, work duration, working conditions, and job titles (master, and fellow hairdresser) should also be considered as risk factors for occupational asthma and respiratory symptoms. With the exception of work intensity, these features have been reported as risk factors in previous studies.^{2–4} Work intensity is an objective parameter for evaluating occupational exposures. In our study, we calculated work intensity from the average number of chemical applications per week (bleaching, dye, and permanent wave) and observed a 3.6 times higher risk of occupational asthma among hairdressers with high work intensity (95% CI 1.2 to 10.9) with a significant trend (χ^2_{trend} 4.9, $p = 0.027$).⁵ However, we did not observe any excess by work duration, which probably is a result of the healthy worker effect. Hollund *et al* stated that the older hairdressers had more customers than the younger ones, which may be

evidence of higher occupational exposures. If they had used work intensity as a more objective criterion than age, they might have prevented possible misclassifications by age. Working conditions of hairdressers and exposures depend on country and regional variability, which might also affect study results. In the United States and United Kingdom, the term "hairdressers" is inclusive, denoting women's hairdressers and barbers for men.⁶ In Turkey, however, the term addresses women's hair salons only. Most of the studies on hairdressers have been published from Nordic and industrialised countries.⁷⁻¹¹ Studies from developing countries will help to describe the extent of occupational health problems among hairdressers and to identify aetiological factors.

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NOTICES

27th International Congress on Occupational Health: The Challenge of Equity in Safety and Health at Work, Iguassu Falls, Brazil, 23-28 February 2003

The Congress will have about nine keynote conferences, approaching different angles of the Central Theme; those themes will then be discussed in depth by Panels (60), where different opinions will be debated. There will be about 60 mini-symposia organised by the ICOH Scientific Committees and Work Groups; facilities for the presentation of 1000 posters; and about 500 free papers. Interest groups may schedule meetings in Congress areas.

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CORRECTIONS

We apologise for the following errors in table 4 of the paper by Wong *et al* (Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China. *Occup Environ Med* 2002;**59**:30-5).

- Mortality from pneumonia and influenza: 4 Pollutant model, under NO₂: "1.004 (1.017 to 1.025)" should read: "1.004 (0.984 to 1.025)".
- Mortality from ischaemic heart diseases: 2 Pollutant model, also under NO₂: "1.022 (1.011 to 1.003)" should read: "1.022 (1.011 to 1.033)".

We apologise for the following error in the paper by Yassin *et al* (Knowledge, attitude, practice, and toxicity symptoms associated with pesticide use among farm workers in the Gaza Strip. *Occup Environ Med* 2002;**59**:387-393).

The page reference at the start of the paper should be 387-393, and not 387-394.