Air pollution and hospital admissions for respiratory and cardiovascular diseases in Hong Kong

Tze Wai Wong, Tai Shing Lau, Tak Sun Yu, Anne Neller, Siu Lan Wong, Wilson Tam, Sik Wing Pang

Abstract

Objective—To investigate short term effects of concentrations of pollutants in ambient air on hospital admissions for cardiovascular and respiratory diseases in Hong Kong.

Methods—Retrospective ecological study. A Poisson regression was performed of concentrations of daily air pollutant on daily counts of emergency hospital admissions in 12 major hospitals. The effects of time trend, season, and other cyclical factors, temperature, and humidity were accounted for. Autocorrelation and over-dispersion were corrected. Daily concentrations of nitrogen dioxide (NO$_2$), sulphur dioxide (SO$_2$), ozone (O$_3$), and particulate matter <10 µm in aerodynamic diameter (PM$_{10}$) were obtained from seven air monitoring stations in Hong Kong in 1994 and 1995. Relative risks (RR) of respiratory and cardiovascular disease admissions for an increase of 10 µg/m$^3$ in concentration of air pollutant were calculated.

Results—Significant associations were found between hospital admissions for all respiratory diseases, all cardiovascular diseases, chronic obstructive pulmonary diseases, and heart failure and the concentrations of all four pollutants. Admissions for asthma, pneumonia, and influenza were significantly associated with NO$_2$, O$_3$, and PM$_{10}$. Relative risk (RR) for admissions for respiratory disease for the four pollutants ranged from 1.013 (for SO$_2$) to 1.022 (for O$_3$), and for admissions for cardiovascular disease, from 1.006 (for PM$_{10}$) to 1.016 (for SO$_2$). Those aged >65 years were at higher risk. Significant positive interactions were detected between NO$_2$, O$_3$, and PM$_{10}$, and between O$_3$ and winter months.

Conclusions—Adverse health effects are evident at current ambient concentrations of air pollutants. Further reduction in air pollution is necessary to protect the health of the community, especially that of the high risk group.

Keywords: air pollution; respiratory diseases; cardiovascular diseases

In recent years, time series studies have been used extensively in the study of air pollution and health outcomes. Positive associations between individual air pollutants and mortalities or morbidities have been found in many American and European studies. Pooled estimates in 12 European cities of increase in daily mortality for an increase of 50 µg/m$^3$ in concentrations of sulphur dioxide (SO$_2$) and particulates were 3% and 2% respectively. Few time series studies have been reported in Asian cities except Beijing. Hong Kong is a densely populated city in Southern China with hot, humid summers and mild, dry winters. Motor vehicles are the main source of air pollutants. In 1994 and 1995, the mean daily concentrations of nitrogen dioxide (NO$_2$) and particulates <10 µm in aerodynamic diameter (PM$_{10}$) were quite high, at 53.7 µg/m$^3$ and 50.1 µg/m$^3$ respectively. Compared with western European cities, Hong Kong had a low concentration of SO$_2$ (daily mean 20.2 µg/m$^3$), whereas ozone (O$_3$) concentrations were comparable (8 hour mean 28.7 µg/m$^3$). To elucidate the association between air pollutants and acute health effects, we performed a time series study on daily hospital admissions from 12 major hospitals and air pollutant concentrations from 7 air quality monitoring stations. The aim of the study was to examine the relation between concentrations of air pollutants and health effects from local data.

Materials and methods

HOSPITAL DATA

Emergency hospital admissions for respiratory and cardiovascular diseases in all 12 major hospitals for 1994 and 1995 were collected. A computerised format of patient data captured age, date of admission, and diagnosis on discharge from the ninth revision of the international classification of diseases (ICD-9). Two groups of diseases were chosen: diseases of the respiratory system (ICD 460–466, 471–478, 480–487, and 490–496) and diseases of the cardiovascular system (ICD 410–417, 420–438, and 440–444). Also, the following diseases were analysed separately: asthma (ICD 493), chronic obstructive pulmo-
nary disease (ICD 490–496), pneumonia and influenza (ICD 480–487), heart failure (ICD 428), ischaemic heart disease (ICD 410–414), and cerebrovascular disease (ICD 430–438).

DATA ON AIR QUALITY
Hourly concentrations of SO2, NO2, PM10, and O3 are monitored by a comprehensive network of stations with pulsed fluorescence, gas phase chemiluminescence, tapered element oscillating microbalance (TEOM), and ultraviolet absorption, respectively.31 Mean daily temperature and humidity were also measured. A rigorous quality control programme was implemented.

STATISTICAL MODELLING
We adopted the method described in the Air Pollution on Health: a European Approach (APHEA) protocol.4 3 This allowed flexibility in variations of local climate and levels of pollution. The daily number of hospital admissions was used as the dependent variable in a Poisson regression model. The following terms were included in the core model: linear and quadratic time trends; year; trigonometric terms to control for seasonality (sin (2kπt/365) and cos (2kπt/365), where k = 1, 2, 3, 4, and 6 represented cycles of 12, 6, 4, 3, and 2 months respectively); days of the week; holiday effects; mean temperature; and humidity. To control for autocorrelation, autoregressive terms up to an order of five were tested. After the core model was fitted, different lags of concentrations of air pollutants were added. The best lag was chosen based on the Akaike’s information criterion value of the model. Brännäs and Johansson’s method (which accounts for over-dispersion by modifying the covariance matrix*) was used in calculating the 95% confidence intervals (95% CIs) of the relative risks (RRs).34

SINGLE POLLUTANT MODEL
Daily concentrations of each air pollutant were added separately into the core model to obtain the respective partial regression coefficients (β) and RR. Delayed effects were investigated with single day lags and cumulative lags up to 5 days for O3 and 3 days for the other air pollutants. Relative risks were calculated for admissions with respiratory and cardiovascular diseases and for the specific diseases already mentioned.

INTERACTIONS BETWEEN POLLUTANTS
In some studies, a multipollutant approach was adopted, either by including all air pollutants into a model, or with stepwise procedures.31 0 In the APHEA protocol, concerns of collinearity between air pollutants preclude the inclusion of all pollutants into a multiple pollutant model.4 To explore interactions between pollutants, we performed pairwise analyses by entering two pollutants and their interaction term into the core model. Each pollutant was analysed as a continuous variable with the other pollutant as a dichotomous variable (high and low concentrations, with the median as the cut off point). Interaction of each pollutant with the cold season (December to March, months with a mean temperature below 20°C) was similarly examined.

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* A consistent estimator of the asymptotic covariance matrix is:

\[
\text{Cov}(\hat{\beta}) = A^{-1}B(A^{-1})^T
\]

where B = X' V X, with V an estimator of Cov (y), y being the vector of time series observations on the count variable, and A = covariance matrix of the ML estimator of \( \beta \).
Table 2  Relative risks (95% CIs)/10 µg/m³ increase in air pollutant for respiratory and cardiovascular admissions by age group (single pollutant model)

<table>
<thead>
<tr>
<th>Pollutant:</th>
<th>Respiratory admissions</th>
<th>Cardiovascular admissions</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO₂</td>
<td>Lag 0–3 days</td>
<td>Lag 0–1 day</td>
</tr>
<tr>
<td>0–4</td>
<td>1.030***</td>
<td>1.010 to 1.030</td>
</tr>
<tr>
<td>5–64</td>
<td>1.023***</td>
<td>1.011 to 1.034</td>
</tr>
<tr>
<td>≥65</td>
<td>1.024***</td>
<td>1.014 to 1.035</td>
</tr>
<tr>
<td>Overall</td>
<td>1.020***</td>
<td>1.013 to 1.026</td>
</tr>
<tr>
<td>SO₂</td>
<td>Lag 0 day</td>
<td>Lag 0–1 day</td>
</tr>
<tr>
<td>0–4</td>
<td>1.005</td>
<td>(0.991 to 1.018)</td>
</tr>
<tr>
<td>5–64</td>
<td>1.008</td>
<td>(0.996 to 1.021)</td>
</tr>
<tr>
<td>≥65</td>
<td>1.023***</td>
<td>(1.012 to 1.036)</td>
</tr>
<tr>
<td>Overall</td>
<td>1.013**</td>
<td>(1.004 to 1.021)</td>
</tr>
<tr>
<td>PM₁₀</td>
<td>Lag 0–3 days</td>
<td>Lag 0–2 days</td>
</tr>
<tr>
<td>0–4</td>
<td>1.010**</td>
<td>(1.011 to 1.028)</td>
</tr>
<tr>
<td>5–64</td>
<td>1.017**</td>
<td>(1.009 to 1.026)</td>
</tr>
<tr>
<td>≥65</td>
<td>1.016**</td>
<td>(1.010 to 1.022)</td>
</tr>
<tr>
<td>Overall</td>
<td>1.016**</td>
<td>(1.004 to 1.023)</td>
</tr>
<tr>
<td>O₃</td>
<td>Lag 0–3 days</td>
<td>Lag 0–5 days</td>
</tr>
<tr>
<td>0–4</td>
<td>1.019***</td>
<td>(1.009 to 1.030)</td>
</tr>
<tr>
<td>5–64</td>
<td>1.022***</td>
<td>(1.011 to 1.034)</td>
</tr>
<tr>
<td>≥65</td>
<td>1.020***</td>
<td>(1.018 to 1.039)</td>
</tr>
<tr>
<td>Overall</td>
<td>1.022***</td>
<td>(1.015 to 1.029)</td>
</tr>
</tbody>
</table>

*p<0.05; **p<0.01; ***p<0.001.
†† 1 ppm of NO₂=1880 µg/m³; 1 ppm of SO₂=2860 µg/m³; 1 ppm of O₃=1960 µg/m³.

Results
Table 1 shows the pollutant concentrations, weather variables, and hospital admissions during the study period. Pollutant concentrations between different monitoring stations were strongly correlated (Pearson’s correlation coefficient $r$ ranged from 0.88 to 0.99 for O$_3$ and PM$_{10}$, 0.68 to 0.89 for NO$_2$, and 0.41 to 0.80 for SO$_2$). PM$_{10}$ was strongly correlated with NO$_2$ ($p=0.79$). For the other pollutants, $r$ ranged from -0.12 to 0.51. Those aged ≥65 accounted for 68% and 38% of admissions for cardiovascular and respiratory diseases respectively. Children under 5 accounted for 31% of respiratory diseases but only 0.5% of cardiovascular diseases. The mean numbers of admissions were slightly higher on days with a mean temperature below 20°C and lower on days above 25°C compared with the 20–25°C range. Time series plots of the daily numbers of hospital admissions for respiratory and cardiovascular diseases and their residuals are shown in the figure. No seasonal or other long term pattern was apparent in the residuals.

We found significant associations between admissions for respiratory and cardiovascular diseases and an increase of 10 µg/m³ for all four pollutants (table 2). A lag effect (cumulative from day 0 to ≥1 days) was found for all pollutants except SO$_2$ for respiratory admissions. The overdispersion parameter ($\phi$) was 1.63 for respiratory diseases and 1.40 for cardiovascular diseases, and the respective autocorrelation coefficients ($r$) were -0.003 and 0.028. Relative risks among those aged ≥65 were higher than in other age groups for all pollutants except PM$_{10}$. Table 3 shows RRs for individual diseases. Significant RRs for chronic obstructive pulmonary disease (COPD) and heart failure were found for all four pollutants. Significant RRs for asthma, pneumonia, and influenza were found for all pollutants except SO$_2$. Ischaemic heart disease and cerebrovascular diseases were not significantly associated with any of the pollutants. Interactions between pollutants and climate are shown in table 4. For respiratory admissions, significant positive interactions were found for O$_3$ with high PM$_{10}$ concentrations. For cardiovascular admissions, significant interactions were found for both PM$_{10}$ and NO$_2$ with high O$_3$ concentration, and for O$_3$ with high PM$_{10}$ concentrations. Ozone had a significant positive interaction with cold season for both respiratory and cardiovascular admissions.

Discussion
This study represents one of few time series studies on health effects of air pollution reported in Asia. The study period was short but the mean daily admissions for respiratory and cardiovascular illnesses were quite large. Confounding by severely cold weather (which has been associated with increased mortalities and morbidities), a concern in some studies, but does not pose a problem here because of the mild winters.

Our findings were broadly consistent with those in European countries and the United States. The effect size for PM$_{10}$, a 1.6% increase in respiratory admissions for an increase of 10 µg/m³, falls within the range (0.8%–3.4%) reported by Dockery and Pope.²⁷ It should be noted that PM$_{10}$ was measured by tapered element oscillating microbalance (TEOM) which may underestimate the true concentration of particulates. The association between O$_3$ and respiratory admissions was in

Table 3  Relative risks (95% CIs) of hospital admissions for individual diseases per 10 µg/m³ increase in the concentrations of air pollutants

<table>
<thead>
<tr>
<th>Pollutant:</th>
<th>Disease category</th>
<th>Lag 0–3 days</th>
<th>Lag 0 day</th>
<th>Lag 0–1 day</th>
<th>Lag 0–2 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO₂</td>
<td>Asthma</td>
<td>1.026**</td>
<td>1.017</td>
<td>1.015*</td>
<td>1.031***</td>
</tr>
<tr>
<td></td>
<td>Chronic obstructive pulmonary diseases</td>
<td>1.029***</td>
<td>1.023**</td>
<td>1.010***</td>
<td>1.032***</td>
</tr>
<tr>
<td></td>
<td>Pneumonia and influenza</td>
<td>1.028***</td>
<td>0.990</td>
<td>1.025**</td>
<td>1.022***</td>
</tr>
<tr>
<td></td>
<td>Heart failure</td>
<td>1.044***</td>
<td>1.036**</td>
<td>1.043**</td>
<td>1.038***</td>
</tr>
<tr>
<td></td>
<td>Ischaemic heart disease</td>
<td>1.025 to 1.063</td>
<td>1.013 to 1.059</td>
<td>1.032 to 1.064</td>
<td>1.018 to 1.059</td>
</tr>
<tr>
<td></td>
<td>Cerebrovascular diseases</td>
<td>1.000 to 1.020</td>
<td>0.995 to 1.025</td>
<td>0.997 to 1.013</td>
<td>0.983 to 1.001</td>
</tr>
</tbody>
</table>

*p<0.05; **p<0.01; ***p<0.001.
agreement with findings elsewhere. A significant association between NO2 and respiratory admissions, not found in other studies, might be due to correlations with PM10. Adverse respiratory effects of SO2 were detectable even at relatively low concentrations. Significant associations with cardiovascular morbidities have not been as widely reported elsewhere as respiratory illnesses.

Compared with respiratory and cardiovascular admissions, the RR for certain disease codings such as asthma, COPD, and heart failure were higher and seemed to be more sensitive indicators of health effect, whereas other diseases (ischaemic heart disease and cardiovascular disease) showed no association. A positive association was found between concentrations of carbon monoxide (CO) and heart failure in United States cities. However, CO concentrations were unavailable in this study. The absence of significant association between the pollutants and ischaemic heart disease or cardiovascular disease contrasts with findings by Pönkä et al and Wordley, who reported a significant association between admissions for cardiovascular disease and PM10 (at lower concentrations than ours) on the same day.

Elderly people had higher RRs for respiratory and cardiovascular admissions for all four pollutants than other age groups except children aged 0–4 years for respiratory admissions and PM10. The identification of target diseases and high risk groups would be useful in finding suitable air quality guidelines in environmental health.

Although the best lag of each pollutant was chosen by statistical criteria, the chemical and toxicological properties of the pollutants might offer plausible explanations. Sulphur dioxide is very soluble in the upper respiratory tract and exerts an immediate irritant effect on the respiratory mucosa. This might explain the absence of lag effect for respiratory admissions. A cumulative lag was found for the less soluble O3 and NO2. Both are highly reactive oxidants which can cause inflammation of the pulmonary epithelium. Low concentrations of O3 cause pulmonary function decrement, biochemical changes, and respiratory symptoms. NO2 forms nitrous and nitric acid in the respiratory epithelium and alters host defence in animal studies. Both gases at high concentrations have caused delayed pulmonary oedema. The pathophysiology of the chemically heterogeneous particulates is less clear. Acid aerosols have been incriminated as a possible cause of ill health. In Hong Kong, the main constituents of PM10 are carbon and sulphates. The acidity of particulates has not been assessed here. The reason for the different lag periods between respiratory and cardiovascular diseases remains unclear.

Collinearity between pollutants was a common problem in time series studies, especially when a multipollutant model is attempted. To study interactions between pollutants, we performed the more conservative pairwise analysis. Significant interactions were found between PM10, SO2, and O3. The NO2 and O3 are strong oxidants, which may explain their synergistic effect. Mechanisms for the interaction between particulates and the oxidants are complex. Gilmour et al hypothesised that the pathogenicity of PM10 particles involved the generation of hydroxyl radicals which led to oxidative stress at the cellular level. A multiple pollutant analysis on mortality in Philadelphia, however, found no interaction between particulates and the gaseous pollutants. Synergistic effects between SO2, O3, and particulates have been reported in Athens but were not found here, possibly because of low SO2 concentrations. In Spokane, where SO2 concentrations were even lower, Schwartz reported similar findings of a significant health effect of particulates independent of SO2. Climatic effects on risk estimates were more obvious in some studies other than ours, illustrating the importance of local pollution mix and meteorological characteristics. The underlying reason for a significant positive interaction between O3 and the cold season (December to March) was unclear.

As with all ecological studies, this study is limited by the lack of precise exposure estimates, and caution should be exercised in inferring cause-effect relations. As more and more epidemiological studies in different parts of the world provide independent and consistent observations of adverse health outcomes at current concentrations of pollutants in ambient air, the need to re-examine national environmental health policies and standards is evident. Collinearity issues and limitations of the time series design preclude the identification of the underlying pollutant causing the health effects, be it ultrafine particles or acid aerosols. Interactions among pollutants and their patho-

Table 4  Relative risks (95% CI) of pollutants at high level † of another pollutant and cold season‡

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>High NO2</th>
<th>High PM10</th>
<th>High O3</th>
<th>Cold season</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory admissions:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO2</td>
<td>—</td>
<td>1.009 (0.993–1.025)</td>
<td>1.013 (0.999–1.026)</td>
<td>1.004 (0.988–1.020)</td>
</tr>
<tr>
<td>PM10</td>
<td>0.984 (0.968–1.001)</td>
<td>—</td>
<td>1.005 (0.995–1.016)</td>
<td>1.006 (0.994–1.017)</td>
</tr>
<tr>
<td>O3</td>
<td>1.005 (0.992–1.019)</td>
<td>1.016* (1.004–1.029)</td>
<td>—</td>
<td>1.018** (1.005–1.032)</td>
</tr>
<tr>
<td>Cardiovascular admissions:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO2</td>
<td>—</td>
<td>1.002 (0.988–1.016)</td>
<td>1.013* (1.002–1.024)</td>
<td>1.103 (0.999–1.027)</td>
</tr>
<tr>
<td>PM10</td>
<td>1.007 (0.995–1.020)</td>
<td>—</td>
<td>1.017*** (1.007–1.028)</td>
<td>1.000 (0.989–1.011)</td>
</tr>
<tr>
<td>O3</td>
<td>1.012 (0.998–1.026)</td>
<td>1.021* (1.005–1.038)</td>
<td>—</td>
<td>1.023** (1.007–1.039)</td>
</tr>
</tbody>
</table>

†High level: level above the median; RR were expressed using low level as reference.
‡December to March
*0.05<p<0.01
**p<0.001
***p<0.001
physiological mechanisms remain to be clarified. Despite our incomplete understanding, stricter control of air pollutants at source (whether by transport policies or regulatory changes) should lower the concentrations of the main ambient pollutants and thus reduce mortalities and morbidities.

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