Effect of Air Pollution on Daily Mortality in Hong Kong
Chit-Ming Wong, Stefan Ma, Anthony Johnson Hedley, and Tai-Hing Lam

Department of Community Medicine, The University of Hong Kong, Hong Kong, China

In different weather conditions, constituents and concentrations of pollutants, personal exposure, and biologic responses to air pollution may vary. In this study we assessed the effects of four air pollutants on mortality in both cool and warm seasons in Hong Kong, a subtropical city. Daily counts of mortality, due to all nonaccidental causes, and cardiorespiratory respiratory diseases were modeled with daily pollutant concentrations [24-hr means for nitrogen dioxide, sulfur dioxide, and particulate matter < 10 µm in aerodynamic diameter (PM10); 8-hr mean for ozone]. Using Poisson regression, we controlled for confounding factors by fitting the terms in models, in line with those recommended by the APHEA (Air Pollution and Health: a European Approach) protocol. Exposure-response relationships in warm and cool seasons were examined using generalized additive modeling. During the cool season, for a linear extrapolation of 10th–90th percentiles in the pollutant concentrations of all oxidant pollutants, NO2, SO2, and O3, we found significant effects on all the mortality outcomes under study, with relative risks (RR) of 1.04–1.10 (p < 0.038, except p = 0.079 for SO2 on respiratory mortality). We observed consistent positive exposure-response relationships during the cool season but not during the warm season. The effects of PM10 were marginally significant (RR = 1.06; p = 0.054) for respiratory mortality but not for the other outcomes (p > 0.135). In this subtropical city, local air quality objectives should take into account that air pollution has stronger health effects during the cool rather than warm seasons and that oxidant pollutants are more important indicators of health effects than particulates. Key words: air pollution concentrations, daily mortality, exposure–response, offset, stratification by seasons. Environ Health Perspect 109:335–340 (2001). [Online 8 March 2001] http://ehpnet1.niehs.nih.gov/docs/2001/109p335-340wong/abstract.html

Time-series methods are widely used for assessment of short-term health effects of air pollution (1). Although limitations arise from ecologic fallacy (2) and the harvesting effect (3–5), time-series methods are more powerful and better able to characterize the population exposure effects than those based on geographic aggregations in cross-sectional studies (6). Also, methods to control for time-related confounding factors are well established (7). Daily time-series analysis is not applicable to the estimation of longer-term chronic exposure effects of air pollution (8), which are public health concerns. Daily time-series analysis may be better estimated from longitudinal studies, but it can be used to assess the potential health benefits of air quality intervention in terms of the number of hospital admissions and deaths avoidable if days with high concentrations (according to a chosen reference value) were eliminated, thus providing information to support the setting of air quality objectives (9,10).

To date, there is coherent evidence that air pollution has short-term effects on mortality (9,11–15), but the questions whether there are independent effects of a single pollutant to account for a health outcome under study and whether there are thresholds and linear or non-linear relationships are still not settled.

In the United States, particulates are regarded as the pollutants that account for most excess mortality due to air pollution (16), but in Europe several studies indicated a stronger association with sulfur dioxide (17). Some showed that it might be the sulfuric acid (18), acid aerosol (19), and mass concentration (20) associated with particulates that are responsible for the effects. Other studies showed that independent effects of individual pollutants cannot be identified in light of the complexity and variability of the air pollution mixtures to which people are exposed (21,22).

In this study we assessed the effects of air pollution on mortality outcomes and identified which pollutants would contribute most to the effect in Hong Kong, a subtropical city in the Asian Pacific rim. Patterns of exposure–response relationships for four criteria pollutants, nitrogen dioxide, sulfur dioxide, particulate matter < 10 µm in aerodynamic diameter (PM10), and ozone were assessed during warm and cool seasons with a view to ascertaining their effects on the commonly used mortality outcomes.

Materials and Methods

Data. For the period 1995–1997, we obtained daily death counts for all nonaccidental causes [International Classification of Diseases, Revision 9 (ICD-9) < 800 (23)], respiratory disease (ICD-9 460–519), and cardiovascular disease (ICD-9 390–459) from the Census & Statistics Department (H Kong Special Administrative Region, People’s Republic of China); meteorologic data (daily mean temperature and relative humidity) from the H Hong Kong Observatory; and air pollutant concentrations (from two to seven monitoring stations) from the Environmental Protection Department. Daily means of 24-hr concentrations of NO2, SO2, and PM10 and 8-hr (900 hr–1700 hr) concentrations of O3 were derived if they were non-missing. Daily concentrations were defined as non-missing if more than 17/24 hr concentrations and more than 5/8 hr concentrations were valid. According to the second phase guidelines of APHEA (Air Pollution and Health: a European Approach), non-missing daily means were first centered for each station [i.e., individual daily concentrations (Xi) were subtracted by an annual station mean (X) for each day]. The centered data from all centers were then combined and added into the annual mean of all stations (X) to form X = (Xi – X, + X). The daily (mean) concentrations of individual pollutants were computed for analysis by taking the mean of X over all stations (24).

Statistical methods. We used Poisson regression with daily mortality counts as the dependent variable. To obtain a core model for each of the mortality outcomes for all ages, nonparametric smoothing (by means of the Loess function) terms for trends on days (1–1,096), seasonality, temperature, and humidity; and dummy variables for days of the week, holidays, and influenza epidemics (weeks with number of hospital admissions for influenza [ICD-9 487] in the upper quartile, which was on average over 8/week in 1995, 1996, and 1997, respectively) were fitted as the independent variables. In addition, we also considered the lag effects of temperature and humidity in building the core models. Residuals of each core model were examined to check whether there were discernible patterns and autocorrelation by means of residual plots and partial autocorrelation function plots, respectively (7). If necessary, both overdispersions and autocorrelations were further adjusted for the model using statistical procedures (7) implemented in S-Plus (MathSoft, Inc., Seattle, WA, USA). We paid special attention to ensure that there were no differences in the residuals

Address correspondence to A.J. Hedley, D Department of Community Medicine, The University of Hong Kong, Patrick Manson Building South Wing, 7 Sassoon Road, Hong Kong, China. Telephone: (852) 2819 9282 / 2819 9280. Fax: (852) 2855 9528. E-mail: hmrjrh@hkucu.hku.hk

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between warm (April–September) and cool (other months) seasons.

We estimated concentrations of current day up to the previous 5 days for O₃ and up to 3 days for other pollutants and identified the best lagged day by a modified version of Akaike’s Information Criterion (AIC) (25). The analysis was also performed using the Loess smoothing function to adjust for non-linear effect of a copollutant. Differences in pollutant effects between seasons were assessed by a season-by-pollutant concentration interaction term in each model, and the effect estimates for cool and warm seasons were derived from the model with the interaction terms.

To perform the stratified analyses, we first obtained expected mortality counts (E) from the core model for all seasons. Poisson regression for the mortality outcomes (Y) was then fitted on pollutant concentrations (X) to obtain the log relative risk (β) estimate with offset log(E) (26) separately for warm and cool seasons. Offset is a computation procedure to treat log(E) as a reference value and does not proceed to estimate a parameter for it in the Poisson regression log(E[Y]) = log(ε) + α + βx (where α is a parameter for the constant term). Exposure-response curves in warm and cool seasons were examined using generalized additive modelling (25).

**Results**

**Summary statistics.** Summary statistics of mortality counts, air pollution concentrations, and meteorologic measurements are presented in Table 1. There were more deaths, higher concentrations of pollutants (except for SO₂, which was about the same), and drier weather conditions in the cool season than in the warm season.

| Mortality counts | No. (day) | Mean | SD | Min | P₁₀ | Median | P₉₀ | Max |
|------------------|----------|------|----|-----|-----|--------|-----|-----|
| Nonaccident (ICD-9: <800) | | | | | | | | |
| Warm | 552 | 75.0 | 9.7 | 47 | 62 | 75 | 87 | 103 |
| Cool | 544 | 87.4 | 12.7 | 53 | 71 | 88 | 103 | 129 |
| Cardiovascular (ICD-9: 390-456) | | | | | | | | |
| Warm | 552 | 19.5 | 4.7 | 8 | 14 | 19 | 26 | 35 |
| Cool | 544 | 26.4 | 6.4 | 12 | 18 | 26 | 35 | 53 |
| Respiratory (ICD-9: 460-519) | | | | | | | | |
| Warm | 552 | 15.9 | 4.9 | 5 | 10 | 16 | 22 | 31 |
| Cool | 544 | 18.3 | 5.3 | 3 | 12 | 18 | 26 | 33 |

**Air pollution concentrations (µg/m³) NO₂ (24-hr) | | | | | | | | |
| Warm | 552 | 48.1 | 18.2 | 15.3 | 27.4 | 45.5 | 72.8 | 125.8 |
| Cool | 544 | 63.8 | 17.5 | 28.7 | 45.2 | 60.6 | 87.3 | 151.5 |
| SO₂ (24-hr) | | | | | | | | |
| Warm | 550 | 18.3 | 13.0 | 1.9 | 5.9 | 15.0 | 35.3 | 83.6 |
| Cool | 544 | 17.2 | 11.6 | 1.1 | 6.4 | 14.4 | 30.8 | 90.1 |
| PM₁₀ (24-hr) | | | | | | | | |
| Warm | 552 | 42.2 | 21.3 | 14.1 | 23.0 | 35.6 | 70.6 | 163.8 |
| Cool | 544 | 61.7 | 24.7 | 14.1 | 33.3 | 58.7 | 95.1 | 156.6 |
| O₃ (8-hr) | | | | | | | | |
| Warm | 548 | 32.0 | 24.5 | 0 | 8.1 | 23.9 | 64.7 | 168.9 |
| Cool | 538 | 35.1 | 21.3 | 0 | 7.9 | 33.2 | 62.8 | 101.6 |

**Meteorologic measurements:**

| Temperature (°C) | | | | | | | | |
| Warm | 552 | 27.3 | 1.9 | 21.0 | 24.5 | 27.4 | 29.6 | 30.9 |
| Cool | 544 | 19.0 | 3.6 | 6.9 | 14.5 | 18.9 | 23.8 | 27.4 |

| Humidity (%) | | | | | | | | |
| Warm | 552 | 80.7 | 7.4 | 46 | 73 | 80 | 91 | 97 |
| Cool | 544 | 74.7 | 12.4 | 31 | 58 | 76.5 | 89 | 95 |

**Abbreviations:** Max, maximum; Min, minimum; P₁₀, 10th percentile; P₉₀, 90th percentile.

**Table 2.** Relative risk (RR) and 95% confidence interval (CI) of the best single lagged-day effects by linear extrapolation for a 10th–90th percentile change in pollutant concentration (1995–1997): whole year.

| Causes of mortality | Lag day | Unadjusted RR (95% CI) | p-Value | Autocorrelation adjusted RR (95% CI) | p-Value | Copollutant | Adjusted for copollutant RR (95% CI) | p-Value |
|---------------------|---------|------------------------|---------|--------------------------------------|---------|-------------|--------------------------------------|---------|
| NO₂                 | 1       | 1.04 (1.01–1.05)       | 0.001   | 1.03 (1.01–1.05)                     | 0.003   | SO₂         | 1.00 (0.97–1.03)                     | 0.896   |
| Nonaccident         | 2       | 1.06 (1.03–1.10)       | 0.001   | 1.06 (1.02–1.10)                     | 0.003   | SO₂         | 1.04 (1.00–1.08)                     | 0.046   |
| Cardiovascular      | 0       | 1.08 (1.02–1.13)       | 0.003   | 1.07 (1.02–1.12)                     | 0.003   | SO₂         | 1.05 (0.98–1.12)                     | 0.168   |
| Respiratory         | 1       | 1.03 (1.02–1.05)       | 0.000   | 1.03 (1.01–1.05)                     | 0.000   | NO₂         | 1.03 (1.01–1.05)                     | 0.003   |
| SO₂                 | 1       | 1.05 (1.02–1.08)       | 0.001   | 1.05 (1.02–1.08)                     | 0.003   | NO₂         | 1.04 (1.00–1.07)                     | 0.023   |
| Nonaccident         | 0       | 1.04 (1.01–1.08)       | 0.010   | 1.04 (1.01–1.07)                     | 0.016   | NO₂         | 1.02 (0.97–1.06)                     | 0.450   |
| Cardiovascular      | 1       | 1.02 (1.00–1.04)       | 0.102   | 1.02 (1.00–1.04)                     | 0.132   | SO₂         | 0.99 (0.97–1.01)                     | 0.397   |
| Respiratory         | 2       | 1.03 (0.99–1.06)       | 0.165   | 1.02 (0.99–1.06)                     | 0.201   | NO₂         | 0.98 (0.92–1.03)                     | 0.363   |
| PM₁₀                | 1       | 1.06 (1.01–1.11)       | 0.024   | 1.05 (1.01–1.10)                     | 0.020   | NO₂         | 1.04 (0.99–1.10)                     | 0.093   |
| Nonaccident         | 1       | 1.01 (0.99–1.03)       | 0.224   | 1.01 (0.99–1.03)                     | 0.226   | NO₂         | 1.01 (0.99–1.03)                     | 0.288   |
| Cardiovascular      | 1       | 1.01 (0.98–1.05)       | 0.479   | 1.01 (0.98–1.05)                     | 0.426   | NO₂         | 1.00 (0.96–1.04)                     | 0.997   |
| Respiratory         | 1       | 1.04 (1.00–1.10)       | 0.078   | 1.03 (0.99–1.07)                     | 0.145   | NO₂         | 1.03 (0.99–1.07)                     | 0.163   |

*The copollutant that produced the least significant effect in the pollutant after adjustment.
outcomes (p < 0.015) for NO₂; b) they were significant in nonaccidental and cardiovascular mortality (p < 0.002) for SO₂; c) they were marginally significant in respiratory mortality (p = 0.054) for PM₁₀, and d) they were all significant (p < 0.038) for O₃. During the cool season with adjustment for copollutants, only the effects of NO₂ on cardiovascular mortality, SO₂ on nonaccidental and cardiovascular mortality, and O₃ on nonaccidental and respiratory mortality remained significant (p < 0.05). However, the between-season differences were statistically significant for NO₂ in cardiovascular mortality with and without adjustment for copollutants (p < 0.039) and for O₃ in all mortality outcomes (p < 0.044) without adjustment and in nonaccidental and respiratory mortality (p = 0.032) with adjustment for copollutant levels.

### Seasonal exposure–response relationships

Figures 1–4 show the exposure–response relationships for each pollutant for the three mortality outcomes at the best lagged day.

NO₂. During the warm season, we observed no clear exposure–response relationships for the three outcomes for NO₂. However, during the cool season there were observable linear exposure–response relationships throughout the concentration levels in nonaccidental mortality, but the curves showed positive and nonlinear relationships at concentrations higher than 80 µg/m³ in the other two outcomes.

SO₂. During the warm season, no exposure–response relationships were observed when SO₂ was < 30 µg/m³, but there were some linear or nonlinear relationships above that concentration. During the cool season, we observed positive exposure–response relationships for concentrations of 0–40 µg/m³ SO₂.

PM₁₀. For PM₁₀, no clear exposure–response relationships were observed for the three outcomes in warm seasons, but in the cool season there was a positive exposure–response relationship for respiratory mortality for concentrations up to 80 µg/m³.

O₃. There were no clear relationships for any of the three outcomes for O₃ during the warm season. However, during the cool season all of the mortality outcomes tended to increase with increasing concentrations.

### Discussion

All pollutant levels are high in Hong Kong. Although SO₂ has been reduced substantially due to government limits on the sulfur content of fuels in the early 1990s (27), the level of SO₂ in Hong Kong still ranks in the middle among more than 30 metropolitan cities in the world. The SO₂ level in Hong Kong is higher than those in Berlin, Germany; Boston, Massachusetts (US); Brisbane, Australia; Kuala Lumpur, Malaysia; London, United Kingdom; and Paris, France (28).

The levels of NO₂ and O₃ have been increasing along with increasing vehicular traffic volume. Levels of PM₁₀, which is primarily related to the use of diesel engines, in Hong Kong are among the highest in the world: they are only lower than those in the most polluted cities such as Barcelona, Spain; Guangzhou, China; Manila, Republic of the Philippines; Mexico City, Mexico; Philadelphia, Pennsylvania (US); Santiago, Chile; Shanghai, China; and Taipei, Taiwan.

In the present study the estimated effects of the pollutants on mortality reached a maximum at a lag of 1–2 days. These observations are consistent with those reported by Brenner et al. (29) in London: the effects increased from lag-day 0 to a maximum at lag-day 1 for NO₂, SO₂, and PM₁₀.

A major finding of this study is that O₃ had effects on all three mortality outcomes during the cool season, and the effects were greater than those in the warm season; this is unlike several other reports in which the effects were found in the warm season (9,12,29). This is consistent with our previous report on the effects of pollution on hospital admissions due to heart failure in subjects ≥65 years of age (30). The effects of the other oxidant pollutants (NO₂ and SO₂) were also significant for all of the mortality outcomes in the cool season but not in the warm season. In Athens, Greece, effects of SO₂ on all causes of nonaccidental mortality were also observed in the cool season (31), but in London, the effects for NO₂ and SO₂ were observed in the warm season (12).

Table 3. Relative risk (RR) and 95% confidence interval (CI) of best single lagged day effects by linear extrapolation for a 10th–90th percentile change in pollutant concentration (1995–1997), without and with adjustment for a copollutant.

### Table 3

| Causes of mortality | Warm season | Cool season | Between season |
|--------------------|-------------|-------------|---------------|
|                    | Copollutant | RR (95% CI) | p-Value | RR (95% CI) | p-Value | p-value |
| NO₂                |             |             |         |             |         |
| Nonaccidental      | -           | 1.02 (0.99–1.05) | 0.243 | 1.05 (1.02–1.08) | 0.003 | 0.193 |
| Cardiovascular     | SO₂         | 1.00 (0.97–1.04) | 0.927 | 1.01 (0.97–1.05) | 0.694 | 0.795 |
| Respiratory        | SO₂         | 1.05 (0.99–1.13) | 0.126 | 1.09 (1.02–1.16) | 0.015 | 0.509 |
|                    | SO₂         | 1.03 (0.95–1.12) | 0.529 | 1.06 (0.98–1.19) | 0.120 | 0.408 |
| SO₂                |             |             |         |             |         |
| Nonaccidental      | -           | 1.02 (0.99–1.04) | 0.170 | 1.04 (1.02–1.07) | 0.001 | 0.101 |
| Cardiovascular     | NO₂         | 1.02 (0.99–1.04) | 0.254 | 1.04 (1.00–1.07) | 0.030 | 0.292 |
| Respiratory        | NO₂         | 1.04 (0.99–1.09) | 0.101 | 1.04 (1.00–1.09) | 0.079 | 0.877 |
|                    | NO₂         | 1.03 (0.97–1.08) | 0.363 | 1.01 (0.94–1.08) | 0.890 | 0.625 |
| PM₁₀               |             |             |         |             |         |
| Nonaccidental      | -           | 1.01 (0.98–1.04) | 0.529 | 1.02 (0.99–1.05) | 0.168 | 0.659 |
| Cardiovascular     | SO₂         | 1.00 (0.96–1.03) | 0.802 | 0.99 (0.96–1.02) | 0.437 | 0.715 |
| Respiratory        | NO₂         | 1.01 (0.99–1.06) | 0.911 | 1.01 (1.00–1.09) | 0.135 | 0.306 |
|                    | NO₂         | 1.05 (0.98–1.12) | 0.194 | 1.06 (1.00–1.13) | 0.054 | 0.761 |
| O₃                 |             |             |         |             |         |
| Nonaccidental      | -           | 0.98 (0.97–1.02) | 0.609 | 1.04 (1.01–1.06) | 0.012 | 0.026 |
| Cardiovascular     | NO₂         | 0.98 (0.97–1.02) | 0.537 | 1.03 (1.01–1.06) | 0.021 | 0.032 |
| Respiratory        | NO₂         | 0.98 (0.94–1.03) | 0.485 | 1.05 (1.00–1.11) | 0.038 | 0.044 |
|                    | NO₂         | 0.98 (0.94–1.03) | 0.513 | 1.04 (0.99–1.09) | 0.150 | 0.132 |
|                    | NO₂         | 0.97 (0.94–1.04) | 0.750 | 1.00 (0.95–1.05) | 0.051 | 0.027 |
|                    | NO₂         | 0.99 (0.94–1.04) | 0.710 | 1.00 (1.02–1.15) | 0.013 | 0.030 |

*Warm season, April–September; cool season, October–March. *Estimated from <L> + copollutant + copollutant × season. *Estimated from <L> + copollutant × season.
reducing the risks of outdoor ambient air pollution exposure.

Another major finding in this study is the positive exposure–response relationships for NO$_2$ and SO$_2$ and all the outcomes during the cool season. There were no thresholds, and the effects showed an inverted “J” shape at higher concentrations. At very high concentrations, the risks of mortality could be reduced possibly because vulnerable subjects may have died before the concentration had reached the maximum levels (4). During the warm season, we observed no consistent positive or negative relationships for all the pollutants. In Hong Kong, there are greater variations in weather conditions in the warm season, when heavy rain, rain storms, and typhoons are common. These factors, in addition to the frequent use of air-conditioning, would prevent the actual exposure–response relationships between air pollution and mortality from being readily observable.

In the absence of an observed linear exposure–response relationship, generalized additive modelling (GAM) could be used to examine whether there are any other forms of relationships. Instead of obtaining a single parameter for the effect, GAM is fitted to obtain a parameter at each point of the independent variable after applying some smoothing function to the data. The fitted values (presented as deviation from an overall mean), along with values of the independent variable, produce an exposure–response plot. It is useful, as demonstrated in this study, in the interpretation of results of daily time-series studies for health effects of air pollution.

Morris and Naumova (33) reported synergistic effects of carbon monoxide and lower temperatures on hospital admissions due to congestive heart failure in Chicago, Illinois (USA). Both CO and cold temperature can increase the load on the heart and thus increase the effect on cardiovascular morbidity (33). For other pollutants, including SO$_2$, the production of synergistic effects was biologically plausible, as both lower temperatures and high air pollutant concentrations were related to increased blood viscosity. Changes in blood rheology may be caused by an inflammatory process in the lung induced by air pollution or by thermoregulatory adjustment to mild surface cooling in cold weather (34,35). This study in Hong Kong is the first to show that all of the oxidant pollutants under study increased effects ($p < 0.07$) on cardiovascular mortality in the cool season. It is also the first study to demonstrate the relationship between pollutant concentration and mortality stratified by cool and warm seasons, on the basis of statistical models with offset on expected counts from the same core model, thus ensuring comparability in the effect estimates between the two seasons. Overall, during the cool season there was a 5–10% ($p < 0.038$) increase in nonaccidental and cardiovascular mortality; this resulted in an increase from the 10th to the 90th percentile (from linear extrapolation) for each of the oxidant pollutants under study. The nonsignificant relative risk estimate for SO$_2$ on respiratory disease may be due to the small change in concentration from the warm season to the cool season. In a sensitivity analysis using the method with offset on expected counts, the estimated increases were consistent but lower, with increases of 2–7% ($p < 0.046$).
The larger p-values may be due to lower power in stratified analysis when the sample size was halved.

Except for respiratory mortality, no strong effects of particulate pollutants were observed in Hong Kong, unlike in other places, although the levels were high. This should be investigated further. The difference may arise from the use of a time-series study, in that the magnitude of the effect estimates depends on the day-to-day covariation of the daily health outcomes and pollutant concentrations instead of the absolute levels of the pollutant concentrations. However, PM$_{10}$ was found to have a significant effect on respiratory mortality (RR = 1.05; p = 0.028) in both seasons combined, but the effects remain approximately the same (RR = 1.05–1.06) although nonsignificant (p > 0.054) in the by-season estimates. The importance of PM$_{10}$ should not be diminished by this finding. NO$_2$ is important because of increasing volumes of vehicular traffic on the roads. SO$_2$ continues to have a strong effect, even though the concentrations have decreased and have been maintained at low levels. The formation of O$_3$ in the ambient air depends on a series of complicated photochemical reactions of oxygen, nitrogen oxides, and reactive hydrocarbons in the presence of sunlight. O$_3$ had been increasing until recently, and it is difficult and costly to control as a regional pollutant. However, for most of the pollutants, the effects were nonsignificant after adjusting for a copollutant; this may arise from a problem of multicollinearity, except for NO$_2$ in cardiovascular mortality and SO$_2$ in both nonaccidental and cardiovascular mortality. These observations, together with strong positive exposure-response relationships for NO$_2$ and SO$_2$, suggested that NO$_2$ and SO$_2$ have independent effects and may be better indicators of effects on mortality in this subtropical city. For O$_3$ all of the RR estimates were not significantly greater than unity; the RR estimates were insensitive to adjustment either for autocorrelation or copollutant (Tables 2 and 3) and were insensitive to the use of maximum, minimum, or mean temperature in the model (data not shown).

In setting air pollution control policy from a public health viewpoint, it is important to identify the health effects of air pollutants from local data. Because of the lack of data, there are few studies based on daily hospital admissions and mortality in the Asian Pacific region. For hospital admissions, there has been only one study in Australia (36) and two in Hong Kong (30,37). For mortality studies, there have been one in Beijing, China (38) based on 1-year daily data, two in Australia (36,39), and two in Korea (40,41). Our report should contribute to the understanding of the effects of air pollutants in this region and may clarify the differences in effects and mechanisms between Western and Eastern populations.

Local data on health effects of air pollution are required for setting standards and objectives for air pollution controls. When local data are not available, foreign data may be helpful, but they may not be relevant or applicable because of a difference in climate or other conditions. Our findings in this study provide information to support a review of air quality objectives with consideration of their effects on health (10).
1. Thurston GD. A critical review of PM\textsubscript{10} mortality time-series studies. J Expo Anal Environ Epidemiol 6:3-21 (1996).

2. Morgenstern H. Use of ecologic analysis in epidemiologic research. Am J Public Health 139:1336-1344 (1982).

3. Schimmel H, Murawski TJ. Evidence for possible acute health effects of ambient air pollution from time series analysis: methodological questions and some new results based on New York City daily mortality 1963-1976. Bull NY Acad Med 54:1052-1058 (1978).

4. Spix C. Daily time series of mortality counts: estimating the harvesting effect. Stat Med (in press).

5. Zeger SL, Dominici F, Samet J. Harvesting-resistant estimates of air pollution effects on mortality. Epidemiology 10:171-175 (1999).

6. Rossi G, Zanobetti A, Marchi M. Time series analysis in environmental epidemiology: short-term effects of air pollution on mortality and morbidity. Epidemiology Prev 19:90-98 (1996).

7. Schwartz J, Spix C, Touloumi G, Bacharova L, et al. Short-term effects of ambient sulphate dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. Am J Epidemiology 146:177-185 (1997).

8. McMichael AJ, Anderson HR, Brunekreef B, Cohen AJ. Air pollution and mortality: a review and meta-analysis. Environ Rev 64:36-52 (1994).

9. Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, Rossi G, Wofsykjan B, Sunyer J, Bacharova L, et al. Short-term effects of ambient sulphate dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. Air Pollution and Health: A European Approach. Br J Med J 314:1658-1660 (1997).

10. Hedley AJ, Wong CM, Wong TW, Tam CM, Yu ITS, Tam E. A critical review of PM\textsubscript{10} mortality time-series studies. J Expos Anal Environ Epidemiol 6:3-21 (1996).

11. McMichael AJ, Anderson HR, Brunekreef B, Cohen AJ. Inappropriate use of daily mortality analyses to estimate long-term mortality effects of air pollution. Int J Epidemiol 27:450-453 (1998).

12. Osto B, Sanchez J, Maranda C, Eskeland GS. Air pollution and mortality: results from a study of Santiago, Chile. J Expo Anal Environ Epidemiol 6:97-117 (1996).

13. Hedley A, Wong CM, Wong TW, Tam CM, Yu ITS, Tam E. Health Effects of Air Pollution. Report from the Sub-Working Group on the Review of Hong Kong's Air Quality Objectives. Hong Kong:Environmental Protection Department, Hong Kong Government, 1999.

14. Kinney P, Ozkaynak H. Associations of daily mortality and air pollution in Los Angeles County, California. Environ Res 54:99-120 (1991).

15. Anderson HR, Ponce de Leon A, Blued J, Bower J, S. Strachan DP. Air pollution and daily mortality in London: 1987-92. Br Med J 312:665-669 (1996).

16. Sunyer J, Castelletiag J, Szasz M, Tobias A, Anto J. Air pollution and mortality in Barcelona. Am J Epidemiol Community Health 50(suppl 1):S47-S51 (1996).

17. Wietlisbach V, Pinto Ackermann-Liebrich U. Air pollution and daily mortality in three Swiss urban areas. Soz Praventivmed 41:107-115 (1996).

18. Dockery DW, Schwartz J, Speizer FE. An association between air pollution and mortality in six US cities. N Engl J Med 328:1763-1769 (1993).

19. Thurston GD, Ito K, Lipmann M, Hayes C. Re-examination of London, England, mortality in relation to exposure to acid aerosols during 1963-1972 winters. Environ Health Perspect 79:73-82 (1990).

20. Breslow NE, Day NR. Statistical Methods in Cancer Research. London:Chapman and Hall, 1990.

21. Keatinge WR, Coleshaw SRK, Cotter F, Mattok M, Murphy M, Chelliah R. Increases in platelet and red cell counts, blood viscosity, and arterial pressure during mild surface cooling: factors in mortality from coronary and cerebral thrombosis in winter. Br Med J 289:1405-1408 (1984).

22. Morgan G, Corbett S, Wlodarczyk E, Lewis P, Lipsett D, Air pollution and daily mortality in Sydney, Australia, 1989 through 1991. Am J Public Health 83:755-764 (1993).

23. Hong Y-C, Leem J-H, Ha E-H, Christiani DC. PM\textsubscript{10} exposure and daily mortality in South Korea. Environ Health Perspect 107:149-154 (1999).

24. Simpson RW, Williams G, Petroeschevsky A, Morgan G, Rutherford S. Associations between outdoor air pollution and daily mortality in Brisbane, Australia. Arch Environ Health 52:442-454 (1997).

25. Lee J-T, Shin D, Chung Y. Air pollution and daily mortality in Inchon, South Korea. Arch Environ Health 52:679-683 (1997).

26. Xu X, Gao J, Dockery DW, Chen Y. Air pollution and daily mortality in residential areas of Beijing, China. Arch Environ Health 49:216-222 (1994).

27. Environmental Health Perspectives 107:149-154 (1999).

28. Hong Y-C, Leem J-H, Ha E-H, Christiani DC. PM\textsubscript{10} exposure, gaseous pollutants, and daily mortality in Inchon, South Korea. Environ Health Perspect 107:873-878 (1999).