Effect of Air Pollution on Daily Mortality in Hong Kong

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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 cardiovascular and respiratory diseases were modeled with daily pollutant concentrations [24-hr means for nitrogen dioxide, sulfur dioxide, and particulate matter < 10 μm in aerodynamic diameter (PM_{10}); 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, NO_2, SO_2, and O_3, 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 SO_2 on respiratory mortality). We observed consistent positive exposure–response relationships during the cool season but not during the warm season. The effects of PM_{10} 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 season and that oxidant pollutants are more important indicators of health effects than particulates. *Key words*: air pollutant concentrations, daily mortality, exposure–response, offset, stratification by seasons. *Environ Health Perspect* 109:335–340 (2001). [Online 8 March 2001]
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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 (PM_{10}), 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 (Hong Kong Special Administrative Region, People's Republic of China); meteorologic data (daily mean temperature and relative humidity) from the 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 NO_2, SO_2, and PM_{10} and 8-hr (900 hr–1700 hr) concentrations of O_3 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 [i.e., individual daily concentrations (X_{ij}) were subtracted by an annual station mean (X_i) for each day j]. The centered data from all centers were then combined and added into the annual mean of all stations (X) to form $X'_{ij} = (X_{ij} - X_i + X)$. The daily (mean) concentrations of individual pollutants were computed for analysis by taking the mean of X'_{ij} 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 trend 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

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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 O_3 and up to 3 days for other pollutants and identified the best lagged day by a modified version of Akaike's Information Criterion (ACI) (*25*). The analysis was also performed using the Loess smoothing function to adjust for nonlinear 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 (ξ) 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 on log(ξ) (*26*) separately for warm and cool seasons. Offset is a computation procedure to treat log(ξ) as a reference value and does not proceed to estimate a parameter for it in the Poisson regression $\log[E(Y)] = \log(\xi) + \alpha + \beta x$ (where α is a parameter for the constant term). Exposure–response curves in warm and cool seasons were examined using generalized additive modeling (*25*).

Results

Summary statistics. Summary statistics of mortality counts, air pollutant concentrations, and meteorologic measurements are presented in Table 1. There were more deaths, higher concentrations of pollutants (except for SO_2, which was about the same), and drier weather conditions in the cool season than in the warm season.

Lag effects. In whole-year analysis, NO_2, SO_2, and PM_{10} showed similar patterns for their effects on all the mortality outcomes in that the relative risks (RRs) increased from lag-day 0, were maximal at either lag-day 1 or lag-day 2, and declined to the lowest at lag-day 3. The RRs at the best lagged day (i.e., the day with minimum AIC) were significant in all three categories of deaths ($p < 0.01$) for NO_2 and SO_2, and was significant ($p = 0.024$) only for respiratory mortality for PM_{10}. For O_3, the RRs were not significant ($p > 0.05$) for any of the lagged days or for any mortality outcomes. With adjustment for autocorrelation, there was little change in the RRs and *p*-values. However, with adjustment for a copollutant, only the RR for NO_2 in cardiovascular mortality ($p = 0.046$) and for SO_2 in nonaccidental ($p = 0.003$) and cardiovascular ($p = 0.023$) mortality remained significant (Table 2).

Effects by seasons. During the warm season, there were no significant effects ($p > 0.1$) for all pollutants for all mortality outcomes (Table 3). During the cool season, without adjustment for copollutants, *a*) all of the RRs at the best lagged days were significantly greater than unity in all the mortality

Table 1. Summary statistics of mortality outcome, air pollution levels, and meteorologic measures by season.

	No. (day)	Mean	SD	Min	P_{10}	Median	P_{90}	Max
Mortality counts								
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	26.2	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/m^3)								
NO_2 (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
SO_2 (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
PM_{10} (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
O_3 (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; P_{10}, 10th percentile; P_{90}, 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.

		Unadjusted		Autocorrelation adjusted			Adjusted for copollutant	
Causes of mortality	Lag day	RR (95% CI)	*p*-Value	RR (95% CI)	*p*-Value	Copollutant[a]	RR (95% CI)	*p*-Value
NO_2								
Nonaccident	1	1.04 (1.01–1.05)	0.001	1.03 (1.01–1.05)	0.003	SO_2	1.00 (0.97–1.03)	0.896
Cardiovascular	2	1.06 (1.03–1.10)	0.001	1.06 (1.02–1.10)	0.003	SO_2	1.04 (1.00–1.08)	0.046
Respiratory	0	1.08 (1.02–1.13)	0.003	1.07 (1.02–1.12)	0.008	SO_2	1.05 (0.98–1.12)	0.168
SO_2								
Nonaccident	1	1.03 (1.02–1.05)	0.000	1.03 (1.01–1.05)	0.000	NO_2	1.03 (1.01–1.05)	0.003
Cardiovascular	1	1.05 (1.02–1.08)	0.001	1.05 (1.02–1.08)	0.003	NO_2	1.04 (1.00–1.07)	0.023
Respiratory	0	1.04 (1.01–1.08)	0.010	1.04 (1.01–1.07)	0.016	NO_2	1.02 (0.97–1.06)	0.450
PM_{10}								
Nonaccident	1	1.02 (1.00–1.04)	0.102	1.02 (1.00–1.04)	0.132	SO_2	0.99 (0.97–1.01)	0.397
Cardiovascular	2	1.03 (0.99–1.06)	0.165	1.02 (0.99–1.06)	0.201	NO_2	0.98 (0.92–1.03)	0.363
Respiratory	1	1.06 (1.01–1.11)	0.024	1.05 (1.01–1.10)	0.028	NO_2	1.04 (0.99–1.10)	0.093
O_3								
Nonaccident	5	1.01 (0.99–1.03)	0.224	1.01 (0.99–1.03)	0.226	NO_2	1.01 (0.99–1.03)	0.288
Cardiovascular	3	1.01 (0.98–1.05)	0.479	1.01 (0.98–1.05)	0.426	NO_2	1.00 (0.96–1.04)	0.997
Respiratory	4	1.04 (1.00–1.08)	0.078	1.03 (0.99–1.07)	0.145	NO_2	1.03 (0.99–1.07)	0.163

[a]The copollutant that produced the least significant effect in the pollutant after adjustment.

outcomes ($p < 0.015$) for NO_2; *b*) they were significant in nonaccidental and cardiovascular mortality ($p < 0.002$) for SO_2; *c*) they were marginally significant in respiratory mortality ($p = 0.054$) for PM_{10}; and *d*) they were all significant ($p < 0.038$) for O_3. During the cool season with adjustment for copollutants, only the effects of NO_2 on cardiovascular mortality, SO_2 on nonaccident and cardiovascular mortality, and O_3 on nonaccident and respiratory mortality remained significant ($p < 0.05$). However, the between-season differences were statistically significant for NO_2 in cardiovascular mortality with and without adjustment for copollutants ($p < 0.039$) and for O_3 in all mortality outcomes ($p < 0.044$) without adjustment and in nonaccident and respiratory mortality ($p < 0.032$) with adjustment for copollutant.

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.

NO_2. During the warm season, we observed no clear exposure–response relationships for the three outcomes for NO_2. 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/m^3 in the other two outcomes.

SO_2. During the warm season, no exposure–response relationships were observed when SO_2 was < 30 μg/m^3, 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/m^3 SO_2.

PM_{10}. For PM_{10}, 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/m^3.

O_3. There were no clear relationships for any of the three outcomes for O_3 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 SO_2 has been reduced substantially due to government limits on the sulfur content of fuels in the early 1990s (*27*), the level of SO_2 in Hong Kong still ranks in the middle among more than 30 metropolitan cities in the world. The SO_2 level in Hong Kong is higher than those in Berlin, Germany; Boston, Massachusetts (USA); Brisbane, Australia; Kuala Lumpur, Malaysia; London, United Kingdom; and Paris, France (*28*).

The levels of NO_2 and O_3 have been increasing along with increasing vehicular traffic volume. Levels of PM_{10}, 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 (USA); 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 Bremner et al. (*29*) in London: the effects increased from lag-day 0 to a maximum at lag-day 1 for NO_2, SO_2, and PM_{10}.

A major finding of this study is that O_3 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 (NO_2 and SO_2) were also significant for all of the mortality outcomes in the cool season but not in the warm season. In Athens, Greece, effects of SO_2 on all causes of nonaccidental mortality were also observed in the cool season (*31*), but in London, the effects for NO_2 and SO_2 were observed in the warm season (*12*). When the data from five western European cities and four central European cities were combined, SO_2 also showed slightly stronger effects during the warm season than during the cool season (*32*).

In Hong Kong in the cool season, air pollutant levels were higher (NO_2, 64 vs. 48; PM_{10}, 62 vs. 42; O_3, 35 vs. 32 μg/m^3) than those in the warm season, except SO_2, which was slightly lower (17 vs. 18). Because pollutants were correlated (r = 0.54–0.72 between NO_2, SO_2, and PM_{10} during the cool season), greater effects observed during cool weather may be due to other pollutants that were also at higher levels during the cool season. The cool season in Hong Kong is drier (humidity 75% vs. 81%), less cloudy (63% vs. 72%), and less variable, so people are more likely to go outdoors and open the windows, thus being exposed to higher levels of air pollution. In contrast, during the warm season (temperatures of 25°C–30°C and humidity of 73%–91% between 10th to 90th percentiles) people usually use air-conditioning, thus

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[a] and with[b] adjustment for a copollutant.

Causes of mortality	Copollutant	Warm season RR (95% CI)	Warm season *p*-Value	Cool season RR (95% CI)	Cool season *p*-Value	Between season *p*-value
NO_2						
Nonaccident	–	1.02 (0.99–1.05)	0.243	1.05 (1.02–1.08)	0.003	0.193
	SO_2	1.00 (0.97–1.04)	0.927	1.01 (0.97–1.05)	0.694	0.795
Cardiovascular	–	1.00 (0.94–1.06)	0.981	1.10 (1.05–1.16)	0.000	0.013
	SO_2	0.99 (0.94–1.05)	0.793	1.08 (1.02–1.14)	0.007	0.039
Respiratory	–	1.05 (0.99–1.13)	0.126	1.09 (1.02–1.16)	0.015	0.509
	SO_2	1.03 (0.95–1.12)	0.529	1.08 (0.98–1.19)	0.120	0.408
SO_2						
Nonaccident	–	1.02 (0.99–1.04)	0.170	1.04 (1.02–1.07)	0.001	0.101
	NO_2	1.02 (0.99–1.04)	0.252	1.04 (1.00–1.07)	0.030	0.292
Cardiovascular	–	1.01 (0.97–1.05)	0.546	1.07 (1.02–1.11)	0.002	0.070
	NO_2	1.01 (0.97–1.06)	0.520	1.05 (1.00–1.09)	0.045	0.310
Respiratory	–	1.04 (0.99–1.09)	0.101	1.04 (1.00–1.09)	0.079	0.877
	NO_2	1.03 (0.97–1.08)	0.363	1.01 (0.94–1.08)	0.890	0.625
PM_{10}						
Nonaccident	–	1.01 (0.98–1.04)	0.529	1.02 (0.99–1.05)	0.168	0.659
	SO_2	1.00 (0.96–1.03)	0.802	0.99 (0.96–1.02)	0.437	0.715
Cardiovascular	–	1.00 (0.94–1.06)	0.911	1.04 (0.99–1.09)	0.135	0.306
	NO_2	1.00 (0.91–1.10)	0.983	0.97 (0.91–1.03)	0.349	0.614
Respiratory	–	1.05 (0.98–1.12)	0.194	1.06 (1.00–1.13)	0.054	0.761
	NO_2	1.04 (0.96–1.12)	0.379	1.05 (0.99–1.12)	0.139	0.810
O_3						
Nonaccident	–	0.99 (0.97–1.02)	0.609	1.04 (1.01–1.06)	0.012	0.026
	NO_2	0.99 (0.97–1.02)	0.537	1.03 (1.01–1.06)	0.021	0.032
Cardiovascular	–	0.98 (0.94–1.03)	0.485	1.05 (1.00–1.11)	0.038	0.044
	NO_2	0.98 (0.94–1.03)	0.513	1.04 (0.99–1.09)	0.150	0.132
Respiratory	–	0.99 (0.94–1.05)	0.750	1.08 (1.02–1.15)	0.011	0.027
	NO_2	0.99 (0.94–1.04)	0.710	1.08 (1.02–1.15)	0.013	0.030

Warm season, April–September; cool season, October–March.
[a]<1> Estimated from core model + pollutant + season + pollutant × season. [b]Estimated from <1> + copollutant + 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 results 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% ($p < 0.046$)

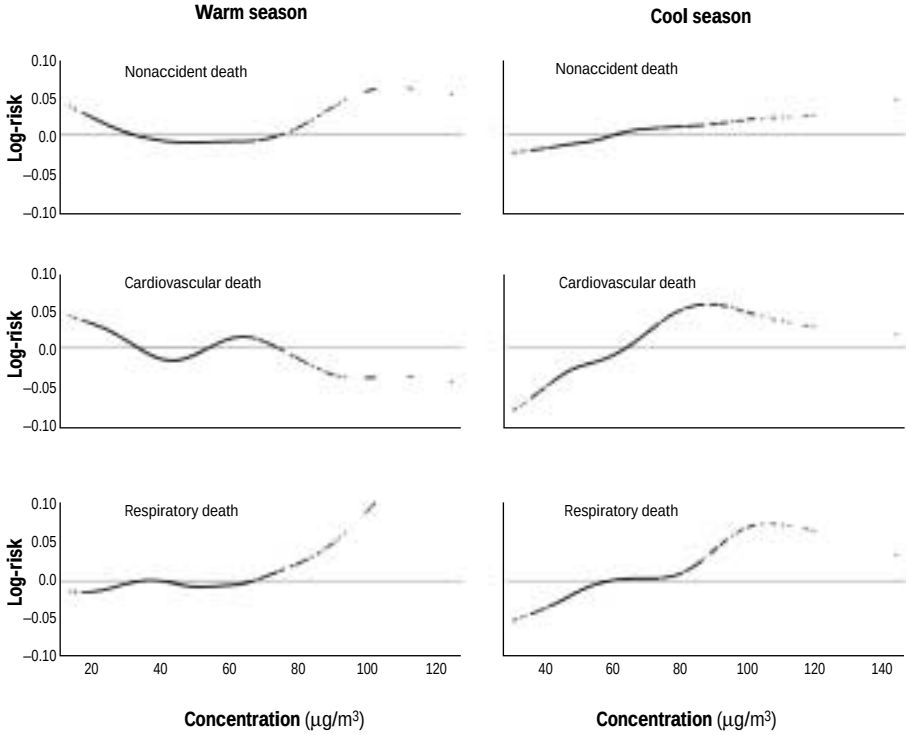

Figure 1. Smoothed plots of NO_2 against mortality risk in log scale (deviated from overall mean) at the best lagged day.

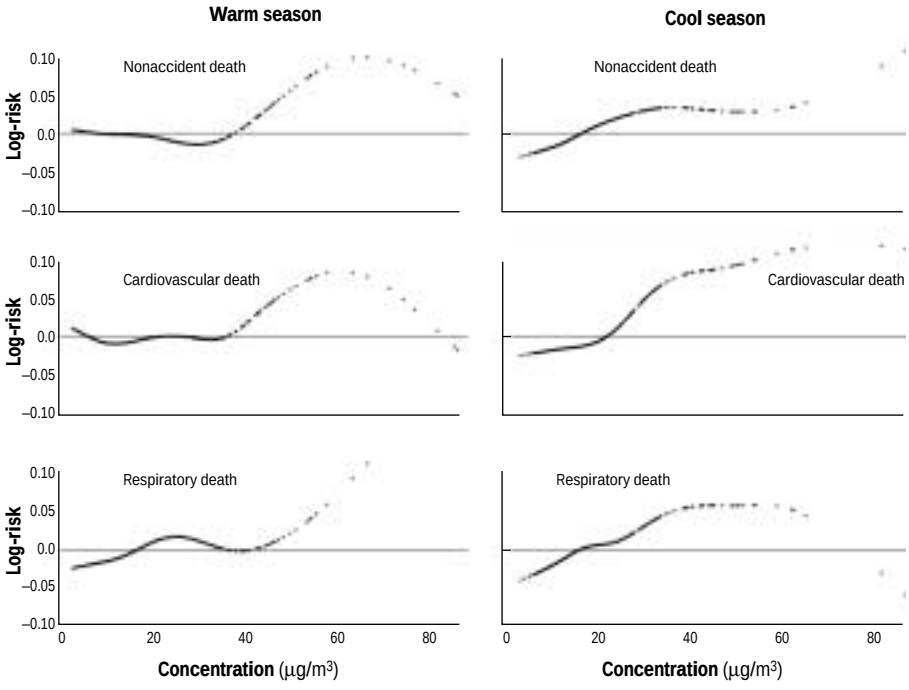

Figure 2. Smoothed plots of SO_2 against mortality risk in log scale (deviated from overall mean) at the best lagged day.

(data not shown). 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*).

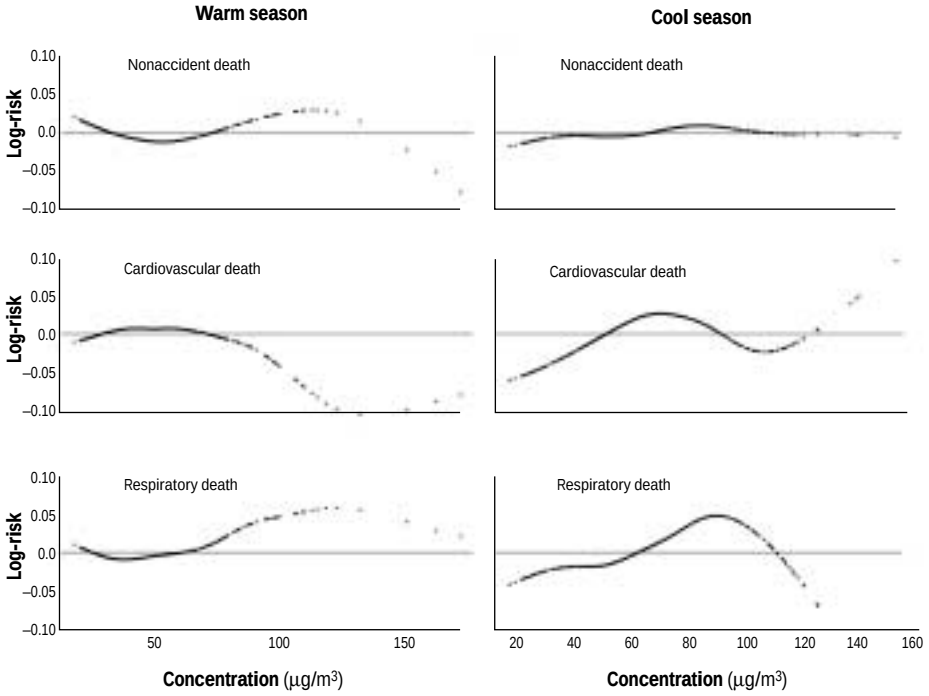

Figure 3. Smoothed plots of PM_{10} against mortality risk in log scale (deviated from overall mean) at the best lagged day.

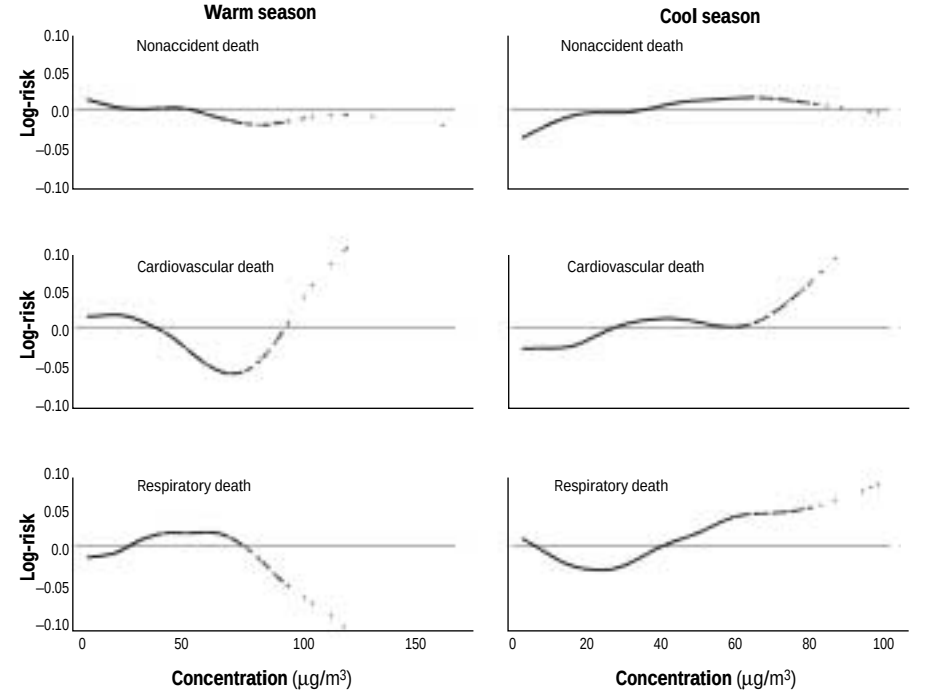

Figure 4. Smoothed plots of O_3 against mortality risk in log scale (deviated from overall mean) at the best lagged day.

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