

A Tale of Two Cities: Effects of Air Pollution on Hospital Admissions in Hong Kong and London Compared

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The causal interpretation of reported associations between daily air pollution and daily admissions requires consideration of residual confounding, correlation between pollutants, and effect modification. If results obtained in Hong Kong and London—which differ in climate, lifestyle, and many other respects—were similar, a causal association would be supported. We used identical statistical methods for the analysis in each city. Associations between daily admissions and pollutant levels were estimated using Poisson regression. Nonparametric smoothing methods were used to model seasonality and the nonlinear dependence of admissions on temperature, humidity, and influenza admissions. For respiratory admissions (≥ 65 years of age), significant positive associations were observed with particulate matter $< 10 \mu\text{m}$ in aerodynamic diameter (PM_{10}), nitrogen dioxide, sulfur dioxide, and ozone in both cities. These associations tended to be stronger at shorter lags in Hong Kong and at longer lags in London. Associations were stronger in the cool season in Hong Kong and in the warm season in London, periods during which levels of humidity are at their lowest in each city. For cardiac admissions (all ages) in both cities, significant positive associations were observed for PM_{10} , NO_2 , and SO_2 with similar lag patterns. Associations tended to be stronger in the cool season. The associations with NO_2 and SO_2 were the most robust in two-pollutant models. Patterns of association for pollutants with ischemic heart disease were similar in the two cities. The associations between O_3 and cardiac admissions were negative in London but positive in Hong Kong. We conclude that air pollution has remarkably similar associations with daily cardiorespiratory admissions in both cities, in spite of considerable differences between cities in social, lifestyle, and environmental factors. The results strengthen the argument that air pollution causes detrimental short-term health effects. **Key words:** air pollution, cardiac and respiratory hospital admissions, daily time-series, Hong Kong, London. *Environ Health Perspect* 110:67–77 (2002). [Online 18 December 2001] <http://ehpnet1.niehs.nih.gov/docs/2002/110p67-77/wong/abstract.html>

There is now considerable evidence that daily hospital admissions for cardiorespiratory diseases are linked to levels of particulate and gaseous ambient air pollution on the same or previous days (1–3). This is consistent with even more substantial evidence concerning daily mortality. In the formulation of public health policy it has been assumed that these associations have a causal basis, but at the scientific level there remain important questions concerning residual confounding, the effects of individual pollutants or mixtures, and other factors that may modify health effects. Further evidence on these issues will have an important bearing on conclusions about the cause and mechanisms of the health effects of air pollution.

Because populations are exposed to mixtures rather than to individual pollutants, multicity studies have the potential to create added insights into some of these issues. Those that have been established so far, using the approaches of APHEA (Air Pollution and Health: a European Approach) Phase I (4) and Phase II (5) and NMMAPS (National Mortality and Morbidity Air Pollution Study) (6,7), are confined to the temperate

climatic zones. Hong Kong is a large city in a subtropical region where there is evidence of adverse effects of air pollution (8,9). London, United Kingdom, is a city of similar size for which adverse health effects of air pollution have also been reported (10,11). On one hand, there are similarities between the two cities in terms of their main sources and levels of pollutants and patterns of the respiratory and cardiac diseases. On the other hand, there are differences between the two cities in terms of a number of factors that might influence confounding or effect modification; these include demography, climate, housing, lifestyle, patterns of disease, the health care system, and seasonal cycles of both weather and pollution variables.

We have conducted parallel analyses of the short-term associations between air pollution and daily hospital admissions in Hong Kong and London to compare and contrast the health effects of air pollution in the two cities. This comparison has relevance to the understanding of the short-term health effects of air pollution, their consistency, and the factors that may modify their effects.

Methods

Daily emergency hospital admissions for respiratory and cardiac diseases were obtained from routine hospital information systems for Hong Kong (1995–1997) and London (1992–1994). The data included in this study are from patients admitted to hospitals immediately either through the accident and emergency departments, general outpatient departments, or directly to the inpatient wards on the grounds of urgency. The series that we chose for comparison were those selected by the APHEA-2 collaboration; these included asthma [*International Classification of Diseases, Revision 9* (ICD-9) code 493] (12) for ages 15–64 years, respiratory disease (ICD-9 460–519) for ages 65 and over, cardiac diseases (ICD-9 396–429) for all ages, and ischemic heart disease (IHD; ICD-9 410–414) for all ages.

Daily average 24-hr concentrations of PM_{10} (particles with median aerodynamic diameter $< 10 \mu\text{m}$), nitrogen dioxide, and sulfur dioxide and average 8-hr concentrations of ozone were collected from background monitoring stations in each city. Only stations able to provide data for 75% or more days during the study periods were used. A daily concentration was accepted as valid if more than 17/24 or 5/8 (in the case of O_3) hourly measurements were made. When data were available from more than one monitoring station, we used a simple filling-in procedure to improve data completeness. Missing values were replaced with the mean of values from those stations with available data. The pollutant measures from

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Table 1. Comparison of environmental factors of Hong Kong and London.

Environmental factor	Hong Kong	London
Population (millions)	6.2 (1995) ^a	6.9 (1992) ^b
Area (km ²)	1,092	1,580
Climate	Subtropical, with rain and tropical cyclones in the summer months	Maritime, with mild winters and temperate summers
Mean January/July temperatures (°C)	16/29	3/23
Rainfall	224 cm, most falling in the summer months	58 cm, evenly distributed through the year
Topography	Peninsula with offshore islands	Estuarine river basin
Lifestyle		
Smoking rates (≥ 15 years of age)	Male 26.7%; female 3.1% ^c	Male 28%; female 27% ^d
Regular alcohol consumers	Male 20.0%; female 2.0% ^e	Male 27%; female 11% ^d
Health care system	Primary care services provided mainly by private sector (85%) Hospital services provided mainly by public sector (86%)	National Health Service
Median size of private dwellings	40.0–69.9 m ^{2a}	85 m ^{2f}
GDP per capita (with adjustment for purchasing power parity)	U.S. \$20,458 ^g	U.S. \$20,890 ^g
Leading causes of death	(1996 data) ^h 1. Malignant neoplasms, 31.3% 2. Heart diseases, 15.8% 3. Cerebrovascular disease, 10.7% 4. Pneumonia (all forms), 10.6% 5. Injury and poisoning, 5.1%	(1996 data) ⁱ 1. Circulatory diseases, 42.6% 2. Malignant neoplasms, 25.0% 3. Respiratory diseases, 15.9% 4. Digestive diseases, 3.6% 5. Injury and poisoning, 2.9%

GDP, gross domestic product.

^aData from the Hong Kong Annual Digest of Statistics (17). ^bData from the Office of Population and Censuses Surveys (18). ^cData from the Census and Statistics Department (19). ^dData from Statistics on Smoking: England, 1976 to 1996 (20). ^eData from Janus et al. (21); alcohol consumption at least once per week (25–74 years of age). ^fData from the Office of National Statistics (22). ^gData from Asia Week (23). ^hDepartment of Health. Department of Health Annual Report (24). ⁱData from the Office of National Statistics (25).

Table 2. Comparison of selected health and air pollution statistics between Hong Kong and London.

Health variable	Hong Kong	London
Population < 15/> 65 years of age (%)	18.9/10.0 (1996) ^a	18.8/13.9 (1992) ^b
Infant mortality rate (per 1,000 live births)	4.0	7.2
Age-standardized mortality ^c (per 1,000 population)		
From all causes	3.7	4.5
From respiratory diseases	0.7	0.5
From cardiovascular diseases	0.9	1.9
Emergency admissions for respiratory disease		
Respiratory (% of all causes)	10.0 (1996)	5.1 (1992/1993)
Age standardized rate ^c (per 1,000 population)	12.9	8.0 (1992–1994)
Age distribution (%)		
0–14 years	33	35
15–64 years	22	26
≥ 65 years	45	39
Subcategories (%)		
Lower respiratory infections (ICD-9 466, 480–487)	23	22
Asthma (ICD-9 493)	13	25
CPD (ICD-9 490–496, excluding 493)	24	15
Emergency admissions for cardiovascular disease		
Cardiovascular (% of all causes)	7.6 (1996)	5.9 (1992/1993)
Age standardized rate (per 1,000 population)	5.8	5.5 (1992–1994)
Age distribution (%)		
0–14 years	2	0
15–64 years	37	32
≥ 65 years	61	68
Subcategories (%)		
Stroke (ICD-9 430–438)	22	19
Cardiac (ICD-9 390–429)	63	70
[Ischemic heart disease (ICD-9 410–414)]	37	30
[Arrhythmias (ICD-9 427)]	20	9
[Cardiac failure (ICD-9 428)]	22	18
Sources of pollutant emissions	(1997) ^d (TSP including PM ₁₀)	(1997) ^e
PM ₁₀ (%)		
Traffic (vehicle, marine vessel, aircraft)	61	83
Industry	6	11
Power generation (and heating for London)	33	6
NO _x (%)		
Traffic (vehicle, marine vessel, aircraft)	41	83
Industry	8	5
Power generation (and heating for London)	45	13
SO ₂ (%)		
Traffic (vehicle, marine vessel, aircraft)	14	28
Industry	21	34
Power generation (and heating for London)	65	38

TSP, total suspended particulate.

^aData from the Hong Kong Department of Health (24). ^bData from the Office of Population and Censuses Surveys (18). ^cThe standard population was adopted from Segi (26). ^dData from the Planning, Environment and Lands Bureau (27). ^eData from the London Research Centre (28).

all stations providing data were then averaged to provide city-wide daily estimates.

We used a statistical approach that closely followed the one adopted by the APHEA-2 study. Poisson regression was used to model the associations between the dependent variable, daily admission counts, and independent variables including non-parametric smooth functions of time, temperature, humidity, and influenza. We used loess functions (13) of time with a minimum span of 60 days to model seasonal fluctuations in admission counts. Temperature and humidity recorded on the day of admission and up to 3 days before admission were investigated and modeled using nonparametric smooth functions, with the degree of smoothing determined by the exposure-response curves and the Akaike's Information Criteria (14). In addition, dummy variables for days of the week, holidays, and unusual events such as thunderstorms and influenza epidemics were included as other independent variables. Daily admission counts for influenza at the 4th quartile (for each week) were used as indicators of influenza epidemics. Models were fitted using a quasi-likelihood method assuming constant over-dispersion over time. This modeling procedure was carried out for each series studied, and the core models were assessed using plots of model residuals and fitted values and plots of the estimated partial autocorrelation functions. Pollution measures were then added in turn, and if necessary, both overdispersion and autocorrelation were further adjusted for using statistical procedures implemented in S-PLUS (Insightful Corporation, Seattle, WA, USA) (15). We examined concentrations on the day of admission and on the previous 3

days, and the means of the current day and the previous day (lag 0–1).

Any linear effect of the pollutant could be assessed by adding a pollutant measure into the model described above. A possible nonlinear effect of the pollutant was further assessed by an exposure–response relationship generated by generalized additive modeling (16). The procedure involved symmetrical application of a loess smoothing function on a number of pollutant measures around a specific point and estimation of the risk at that specific point. A plot of risk against all the specific points along the *x*-axis produced an exposure–response curve.

To investigate seasonal differences in the pollution effects, dummy variables were added to the models to indicate season and pollutant–season interaction terms. The warm season was defined as April–September and the cool season was October–March. We derived estimates of the pollutant effects in each season from the models together with *p*-values for the interaction terms, which indicated whether or not the observed seasonal differences were statistically significant.

We used two-pollutant models to estimate the effects of one pollutant at mean cumulative lag 0–1 days after controlling for another pollutant also at mean cumulative lag 0–1 days.

Results

Background demographic, health, and environmental data. The background characteristics of the two cities have been summarized (Table 1). Hong Kong has a population of over 6 million and occupies an area of approximately 1,000 km², comprising two major islands, some smaller outer islands, a peninsula, the lands adjacent to the mainland, and some reclaimed areas. It is situated at 22.5°N latitude at the mouth of the Pearl River, which opens into the South China sea. Hong Kong has a subtropical climate that tends toward the temperate for nearly half the year. The average annual rainfall is 224 cm, most of which falls in the summer months.

Greater London has a population of about 7 million people and occupies a roughly circular basin of 1,600 km², which is bisected east to west by the River Thames and bounded to the north and south by low hills. It lies at a latitude of 45°N and has a temperate maritime climate. The average annual rainfall is 58 cm, which falls throughout the year.

Table 2 shows a comparison of relevant demographic, health, and environmental characteristics for Hong Kong and London. The age distributions of the two cities are similar, but age-standardized annual mortality rates are lower in Hong Kong than in London for deaths from all causes and from

cardiovascular diseases. Standardized annual rates for admission to public hospitals are higher in Hong Kong than in London for respiratory disease (12.9 vs. 8.0 per 1,000) and for cardiovascular disease (5.8 vs. 5.5 per 1,000). Among respiratory hospital admissions, the proportions due to respiratory infections are similar, but admissions due to chronic obstructive pulmonary disease are greater in Hong Kong and those due to asthma are higher in London. The relative distributions of subcategories of cardiac diseases were similar except that arrhythmias were more common in Hong Kong.

PM₁₀ and NO₂ emissions in London were both predominantly from traffic (83% and 83%, respectively); in Hong Kong they were from both traffic (61% and 41%, respectively) and power generation (33% and 45%, respectively). SO₂ in London was almost equally derived from traffic, industry, and power generation (28%, 34%, and 38%, respectively), but in Hong Kong they were mainly from power generation (65%) and industry (21%).

Daily time-series data. Summary statistics for daily counts of admissions, by cause and age, pollutant concentrations, and meteorologic variables are shown in Table 3. Correlations between these variables are shown in Table 4. London had almost twice the median daily count of admissions for asthma as Hong Kong but only two-thirds the median number of admissions for respiratory disease. The numbers of cardiac admissions were more comparable, but London had almost 50% more emergency admissions for IHD than Hong Kong.

The concentrations of NO₂, SO₂, and O₃ were higher in London, whereas PM₁₀ levels in Hong Kong were almost double those in London (46.8 vs. 24.8 μg/m³) (Table 3). In Hong Kong, there was a marked seasonal variation in ambient concentrations of NO₂, O₃, and PM₁₀, all of which were lowest in the warm season and highest in the cool season. In contrast, SO₂ tended to show less seasonal variation and was highest in the warm season. In London, there was little seasonal variation in NO₂,

Table 3. Summary statistics for daily hospital admissions, pollutant concentrations, and meteorologic measurements in Hong Kong (1995–1997) and London (1992–1994); *n* = 1,096 days.

Variable, city	Mean (Warm/cool)	SD	Percentile					
			Min	10th	50th	90th	Max	
Emergency hospital admission (no./day)								
Asthma (ICD-9 493), 15–64 years								
Hong Kong	7.8	(7.0/8.6)	3.4	0.0	4.0	7.0	12.0	24.0
London	14.1	(13.0/15.1)	5.8	2.0	8.0	13.0	21.0	85.0
Respiratory (ICD-9 460–519), ≥ 65 years								
Hong Kong	91.3	(86.7/96.1)	22.5	45.0	64.0	88.0	122.0	174.0
London	58.3	(49.5/67.4)	19.4	13.0	37.0	55.0	82.0	150.0
Cardiac (ICD-9 390–429), all ages								
Hong Kong	98.7	(94.0/103.4)	23.3	40.0	67.0	101.0	127.5	176.0
London	121.1	(118.3/124.0)	23.4	50.0	89.0	121.0	152.0	196.0
IHD (ICD-9 410–414), all ages								
Hong Kong	36.0	(35.3/36.7)	10.3	8.0	23.0	35.0	49.0	76.0
London	51.3	(50.5/52.0)	10.0	22.0	39.0	51.0	64.0	86.0
Pollutant concentration (daily μg/m ³)								
NO ₂ (24 hr)								
Hong Kong ^a	55.9	(48.1/63.8)	19.4	15.3	31.8	53.5	81.8	151.5
London ^b	64.3	(62.6/66.1)	20.4	23.7	42.3	61.2	88.8	255.8
O ₃ (8 hr)								
Hong Kong ^c	33.5	(32.0/35.1)	23.0	0	7.9	28.3	64.0	168.9
London ^d	34.9	(45.3/24.0)	23.1	2.4	8.6	32.0	60.1	159.8
PM ₁₀ (24 hr)								
Hong Kong ^e	51.8	(42.2/61.6)	25.0	14.1	24.7	46.8	87.2	163.8
London ^f	28.5	(28.2/28.8)	13.7	6.8	15.8	24.8	46.4	99.8
SO ₂ (24 hr)								
Hong Kong ^g	17.7	(18.3/17.2)	12.3	1.1	6.2	14.5	32.8	90.0
London ^h	23.7	(22.2/25.3)	12.3	6.2	13.2	20.6	38.1	113.6
Meteorologic measurements (daily)								
Temperature (°C)								
Hong Kong	23.2	(27.2/19.0)	5.0	6.9	16.0	24.3	29.1	30.9
London	11.9	(15.5/8.3)	5.0	–0.8	5.6	11.8	18.6	25.5
Humidity (%)								
Hong Kong	77.7	(80.7/74.7)	10.6	31.0	64.0	79.0	90.0	97.0
London	70.6	(67.5/73.7)	10.9	41.0	56.0	70.0	85.0	97.0

Abbreviations: Max, maximum; Min, minimum.

^a*r* = 0.65–0.90 between seven stations. ^b*r* = around 0.8 between three stations. ^c*r* = 0.79 between two stations. ^d*r* = 0.95 between two stations. ^e*r* = 0.92–0.97 between five stations. ^fOnly one station involved. ^g*r* = 0.44–0.81 between five stations. ^h*r* = –0.1 to 0.8 (median 0.5) in six stations.

SO₂, or PM₁₀, but there was marked seasonal variation in O₃, which was highest in the warm season. Thus, the only pollutant with a similar seasonal pattern in both cities was SO₂. Mean daily temperature was twice as high in Hong Kong as in London (23°C vs. 12°C), and the mean relative humidity was also higher in Hong Kong. The two cities have similar cycles of temperature, but their seasonal patterns for humidity differ markedly; humidity in Hong Kong is highest in the warm season, but in London it is highest in the cool season (Table 3).

Single-pollutant models. The associations between pollutants (*a priori* mean lag 0–1 days) and the four admission outcomes are shown in Table 5. We found no statistically

significant associations between asthma admissions and any of the four pollutants in either of the cities. For respiratory admissions, we found small, positive, and statistically significant associations with all four pollutants in Hong Kong. By contrast, only O₃ was significantly associated with respiratory admissions in London. For cardiac diseases, both cities showed significant positive associations of comparable size with NO₂, PM₁₀, and SO₂. There were no significant positive associations with O₃ in Hong Kong, whereas in London it was significantly negative. The direction of effects for IHD was the same as for all cardiac diseases in both cities, but the estimates were lower in Hong Kong than in London (except O₃) and none

were significant. In London, the relative risks for IHD were similar to those for all cardiac diseases and all were significant; the association with O₃ was negative.

Results for the most significant single day lag from lags 0 to 3 are shown in Table 6 and illustrated along with the other single day lags in Figure 1. Generally, these results are similar in terms of direction and magnitude to the *a priori* choice of mean lags 0 and 1. One difference was that in London, the associations between admissions for asthmatic attacks in the 15–64 age group, as well as respiratory disease in the ≥ 65 age group and NO₂, PM₁₀, and SO₂ in the best single lag days, were larger than the *a priori* (lag 0–1) choice and are statistically significant.

Table 4. Matrix of Spearman’s rank correlation coefficient (*r*) between mean daily concentration of pollutants and meteorologic data (1995–1997).

	SO ₂	PM ₁₀	O ₃	Temperature	Humidity		SO ₂	PM ₁₀	O ₃	Temperature	Humidity
Hong Kong						London					
Whole year						Whole year					
NO ₂	0.37	0.82	0.43	-0.45	-0.35	NO ₂	0.71	0.68	-0.29	-0.16	0.01
SO ₂		0.30	-0.18	0.17	-0.16	SO ₂		0.64	-0.25	-0.13	-0.15
PM ₁₀			0.54	-0.42	-0.53	PM ₁₀			0.17	0.02	-0.05
O ₃				-0.14	-0.59	O ₃				0.47	-0.52
Temperature					0.19	Temperature					-0.27
Warm season						Warm season					
NO ₂	0.28	0.80	0.54	-0.43	-0.18	NO ₂	0.66	0.68	0.05	0.08	-0.09
SO ₂		0.22	-0.14	0.37	-0.16	SO ₂		0.56	0.14	0.26	-0.33
PM ₁₀			0.65	-0.25	-0.40	PM ₁₀			0.27	0.32	-0.14
O ₃				-0.17	-0.57	O ₃				0.26	-0.53
Temperature					-0.26	Temperature					0.26
Cool season						Cool season					
NO ₂	0.61	0.72	0.23	0.10	-0.36	NO ₂	0.76	0.68	-0.61	-0.36	0.01
SO ₂		0.53	-0.21	0.13	-0.20	SO ₂		0.70	-0.58	-0.46	-0.05
PM ₁₀			0.36	0.01	0.55	PM ₁₀			-0.56	0.23	0.01
O ₃				0.05	-0.60	O ₃				0.29	-0.37
Temperature					0.21	Temperature					0.05

Table 5. Summary of single-pollutant excess risk (ER) and 95% confidence interval (CI) for a 10 µg/m³ change in pollutant concentration for mean lag 0–1 day: comparison between Hong Kong and London.

Emergency admission complaints, age	Hong Kong ER (95% CI)	London ER (95% CI)
Asthma, 15–64 years		
NO ₂	-0.6 (-2.1–1.0)	1.0 (0.0–2.1)
O ₃	0.0 (-1.3–1.4)	-0.7 (-1.8–0.4)
PM ₁₀	-1.1 (-2.4–0.1)	1.4 (-0.1–3.0)
SO ₂	-0.1 (-2.4–2.2)	0.7 (-1.0–2.5)
Respiratory, ≥ 65 years		
NO ₂	1.8 (1.2–2.4)	-0.1 (-0.6–0.5)
O ₃	0.8 (0.3–1.3)	0.8 (0.2–1.4)
PM ₁₀	1.0 (0.5–1.5)	0.4 (-0.3–1.2)
SO ₂	1.8 (0.9–2.6)	0.2 (-0.6–1.1)
Cardiac, all ages		
NO ₂	1.4 (0.9–2.0)	0.7 (0.3–1.0)
O ₃	0.3 (-0.2–0.7)	-0.6 (-1.0–0.1)
PM ₁₀	0.7 (0.3–1.1)	0.8 (0.3–1.4)
SO ₂	2.1 (1.3–2.8)	1.6 (1.0–2.2)
IHD, all ages		
NO ₂	0.6 (-0.2–1.4)	0.7 (0.2–1.2)
O ₃	0.4 (-0.3–1.1)	-0.8 (-1.4–0.2)
PM ₁₀	0.5 (-0.1–1.1)	0.9 (0.1–1.6)
SO ₂	0.1 (-1.1–1.2)	1.7 (0.8–2.6)

Table 6. Summary of single-pollutant excess risk (ER) and 95% confidence interval (CI) for a 10 µg/m³ change in pollutant concentration for the best single lag day: comparison between Hong Kong and London.

Emergency admission complaints, age	Hong Kong		London	
	Lag	ER (95% CI)	Lag	ER (95% CI)
Asthma, 15–64 years				
NO ₂	1	-1.3 (-2.6–0.1)	2	1.1 (0.2–2.0)
O ₃	2	1.2 (0.0–2.4)	0	-0.7 (-1.7–0.3)
PM ₁₀	0	-1.1 (-2.1–0.0)	2	2.2 (0.8–3.6)
SO ₂	2	-1.5 (-3.4–0.5)	3	2.1 (0.7–3.6)
Respiratory, ≥ 65 years				
NO ₂	0	1.3 (0.8–1.8)	3	0.9 (0.5–1.3)
O ₃	1	0.6 (0.2–1.0)	0	0.6 (0.1–1.2)
PM ₁₀	0	0.7 (0.3–1.0)	3	1.5 (0.8–2.2)
SO ₂	0	1.7 (1.0–2.4)	3	1.2 (0.5–2.0)
Cardiac, all ages				
NO ₂	0	1.2 (0.7–1.7)	0	0.7 (0.4–1.0)
O ₃	2	0.5 (0.1–0.8)	0	-0.8 (-1.2–0.4)
PM ₁₀	0	0.5 (0.2–0.9)	0	1.1 (0.5–1.5)
SO ₂	0	1.6 (1.0–2.2)	0	1.4 (0.9–1.9)
IHD, all ages				
NO ₂	3	0.7 (0.1–1.4)	0	0.7 (0.2–1.1)
O ₃	3	0.5 (0.0–1.0)	0	-0.9 (-1.4–0.3)
PM ₁₀	2	0.5 (-0.1–1.0)	3	0.3 (-0.5–1.0)
SO ₂	2	0.4 (-0.5–1.4)	0	1.4 (0.7–2.2)

These stronger associations all occur with longer lag 3 except once with lag 2. Another clear difference between the two cities was for admissions for IHD. In Hong Kong the most significant associations occurred at lag 2 or 3 days for the four pollutants, whereas in London they were at lag 0 days for NO₂, O₃, and SO₂. PM₁₀ was the exception in London, with the most significant lag

occurring at lag 3 days. In both cities however the magnitude of the effects were similar whether at mean lag 0–1 days or the most significant day.

Estimates of pollution effects by season (Table 7) showed contrasting patterns between the two cities for respiratory disease and similar patterns for cardiac disease (Figure 2). In Hong Kong, pollution effects

on respiratory disease tended to be greater in the cool season and significantly so for NO₂ and SO₂ (Table 7). In London, the pattern was reversed with greater effects in the warm season, significantly so for NO₂ and PM₁₀ (Table 7). The two cities were similar in having larger estimates of cardiac admissions in the cool season (with the exception of O₃ for London); all of these seasonal interactions were significant for Hong Kong, but only one (PM₁₀) was significant for London (Table 7).

Two-pollutant models. In Hong Kong, associations between respiratory admissions and each of the four pollutants studied tended to be robust to inclusion of a second pollutant into the models (Table 8). There were two exceptions: the PM₁₀ and SO₂ associations were substantially reduced after NO₂ was added to the models. In London, associations between respiratory admissions and NO₂, PM₁₀, and SO₂ were nonsignificant and remained unchanged after the addition of a second pollutant. The significant O₃ associations found in London were robust to the inclusion of an additional pollutant.

For cardiac admissions in Hong Kong, the addition of NO₂ or SO₂ reduced the magnitude and statistical significance of NO₂, SO₂, and PM₁₀ associations (O₃ was not found to be significant in single-pollutant models). These results were largely replicated in the London analyses, although in a model containing NO₂ and SO₂, SO₂ was clearly the “most robust” pollutant, retaining both the magnitude and statistical significance of its association after the inclusion of NO₂.

Exposure–response relationships. For respiratory admissions in Hong Kong, a negative

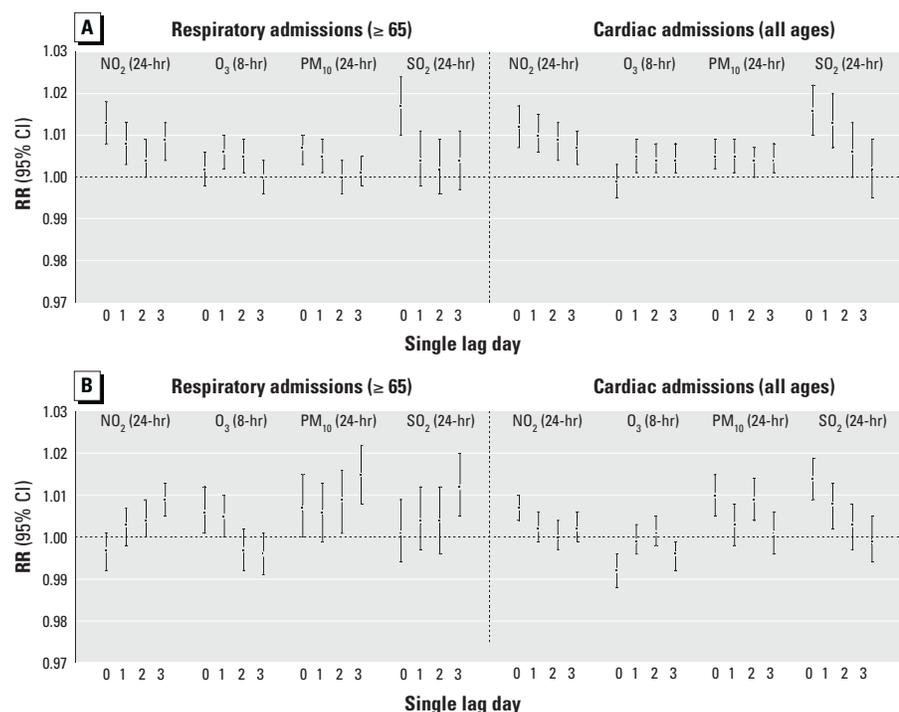


Figure 1. Effect of pollutants at a single lag day on hospital admissions due to cardiac and respiratory disease in (A) Hong Kong and (B) London. Values shown are relative risk (RR) and 95% confidence interval (CI) for a 10 $\mu\text{g}/\text{m}^3$ increase in concentration.

Table 7. Summary of single-pollutant results in excess risk (ER) and 95% confidence interval (CI) for a 10 $\mu\text{g}/\text{m}^3$ change in concentration at mean lag 0–1 day in warm and cool seasons.

Emergency admission complaints, age	Significance for pollutant by season interaction		Emergency admission complaints, age	Significance for pollutant by season interaction	
	Warm ER (95% CI)	Cool ER (95% CI)		Warm ER (95% CI)	Cool ER (95% CI)
Hong Kong					
Asthma, 15–64 years					
NO ₂	-0.5 (-2.7–1.6)	-0.6 (-2.8–1.6)	NO ₂	0.6 (-0.8–2.0)	1.3 (-0.1–2.8)
O ₃	-0.3 (-2.0–1.3)	0.6 (-1.4–2.6)	O ₃	-0.1 (-1.4–1.2)	-2.6 (-4.6–-0.5)
PM ₁₀	-1.0 (-2.8–0.8)	-1.2 (-2.8–0.4)	PM ₁₀	0.6 (-1.9–3.1)	1.6 (-0.3–3.6)
SO ₂	1.5 (-1.5–4.6)	-2.0 (-5.4–1.4)	SO ₂	-1.4 (-4.7–1.9)	1.6 (-0.5–3.8)
Respiratory, ≥ 65 years					
NO ₂	0.8 (0.1–1.6)	3.0 (2.1–3.9)	NO ₂	0.6 (-0.2–1.4)	-0.7 (-1.4–0.0)
O ₃	0.8 (0.2–1.4)	1.0 (0.2–1.7)	O ₃	1.0 (0.3–1.7)	0.2 (-0.7–1.2)
PM ₁₀	0.8 (0.1–1.4)	1.2 (0.6–1.9)	PM ₁₀	1.8 (0.5–3.1)	-0.5 (-1.5–0.5)
SO ₂	1.1 (0.0–2.2)	2.7 (1.4–4.0)	SO ₂	1.3 (-0.5–3.1)	-0.3 (-1.3–0.8)
Cardiac, all ages					
NO ₂	0.3 (-0.4–1.0)	2.6 (1.9–3.3)	NO ₂	0.4 (-0.1–0.9)	0.8 (0.3–1.4)
O ₃	0.0 (-0.5–0.6)	0.9 (0.2–1.6)	O ₃	-0.2 (-0.7–0.3)	-1.1 (-1.8–-0.4)
PM ₁₀	0.0 (-0.6–0.6)	1.3 (0.8–1.9)	PM ₁₀	0.1 (-0.7–1.0)	1.2 (0.5–2.0)
SO ₂	1.0 (0.0–2.0)	3.3 (2.1–4.4)	SO ₂	0.6 (-0.6–1.7)	1.9 (1.2–2.7)
IHD, all ages					
NO ₂	0.1 (-0.9–1.2)	1.2 (0.0–2.3)	NO ₂	0.4 (-0.3–1.1)	1.0 (0.2–1.7)
O ₃	0.4 (-0.4–1.2)	0.6 (-0.5–1.6)	O ₃	-0.5 (-1.2–0.2)	-1.3 (-2.3–-0.3)
PM ₁₀	0.2 (-0.7–1.0)	0.8 (-0.1–1.6)	PM ₁₀	0.1 (-1.1–1.4)	1.3 (0.3–2.3)
SO ₂	-0.6 (-2.0–0.8)	1.0 (-0.8–2.8)	SO ₂	1.0 (-0.6–2.6)	2.0 (0.9–3.1)

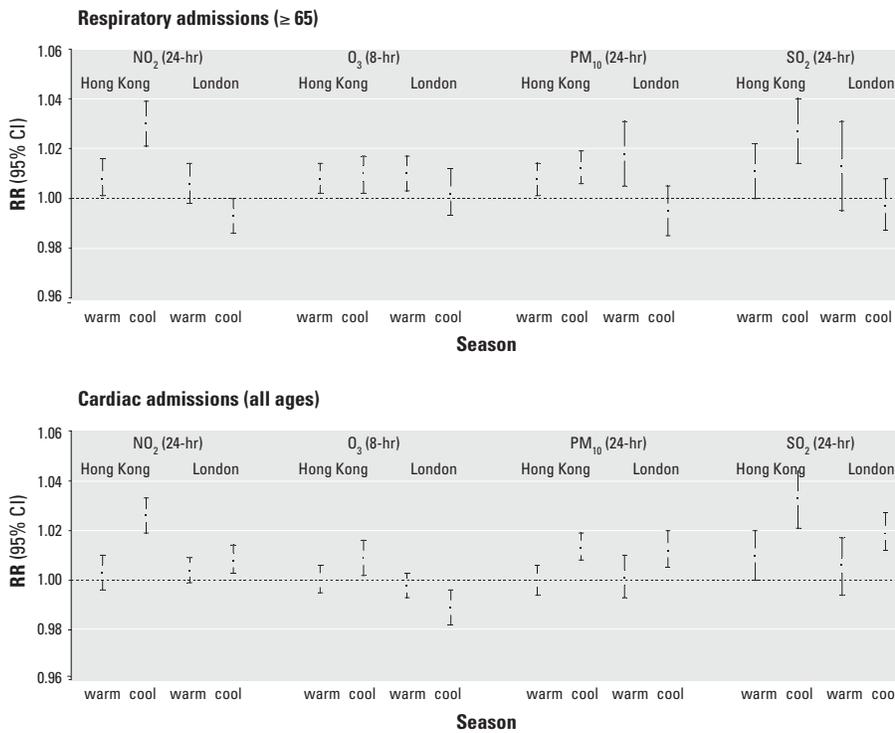


Figure 2. Comparison of pollutant effects in cool and warm seasons on hospital admissions due to respiratory and cardiac diseases. Values shown are relative risk (RR) and 95% confidence interval (CI) for a 10 µg/m³ increase in concentration in mean lag 0–1 days.

Table 8. Excess risk (ER) and 95% confidence interval (CI) for a 10 µg/m³ change in mean concentration of lag 0–1 day in each air pollutant from a single- and co-pollutant model.

Emergency admission	After adjusting for co-pollutant			
	NO ₂ ER (95% CI)	O ₃ ER (95% CI)	PM ₁₀ ER (95% CI)	SO ₂ ER (95% CI)
Respiratory				
NO ₂				
Hong Kong	1.8 (1.2–2.4) ^a	1.6 (1.0–2.3)	1.7 (0.8–2.7)	1.6 (0.8–2.4)
London	-0.1 (-0.6–0.5) ^a	0.1 (-0.5–0.6)	-0.4 (-1.2–0.4)	-0.2 (-0.9–0.5)
O ₃				
Hong Kong	0.5 (0.0–1.0)	0.8 (0.3–1.3) ^a	0.5 (0.0–1.1)	1.0 (0.5–1.5)
London	0.8 (0.2–1.4)	0.8 (0.2–1.4) ^a	1.1 (0.5–1.7)	0.9 (0.3–1.5)
PM ₁₀				
Hong Kong	0.0 (-0.7–0.7)	0.8 (0.3–1.3)	1.0 (0.5–1.5) ^a	0.6 (0.1–1.1)
London	0.9 (-0.3–2.0)	0.4 (-0.3–1.2)	0.4 (-0.3–1.2) ^a	0.7 (-0.5–1.8)
SO ₂				
Hong Kong	0.3 (-0.7–1.4)	1.9 (1.1–2.8)	1.2 (0.3–2.2)	1.8 (0.9–2.6) ^a
London	0.5 (-0.7–1.7)	0.5 (-0.4–1.5)	-0.4 (-1.8–1.0)	0.2 (-0.6–1.1) ^a
Cardiac				
NO ₂				
Hong Kong	1.4 (0.9–2.0) ^a	1.5 (0.9–2.0)	1.7 (0.9–2.5)	0.7 (0.1–1.4)
London	0.7 (0.3–1.0) ^a	0.7 (0.3–1.1)	0.6 (0.0–1.2)	0.1 (-0.3–0.6)
O ₃				
Hong Kong	-0.1 (-0.6–0.4)	0.3 (-0.2–0.7) ^a	0.0 (-0.5–0.5)	0.4 (-0.1–0.9)
London	-0.5 (-0.9–0.0)	-0.6 (-1.0–0.1) ^a	-0.6 (-1.0–0.1)	-0.3 (-0.8–0.1)
PM ₁₀				
Hong Kong	-0.3 (-0.9–0.4)	0.7 (0.3–1.2)	0.7 (0.3–1.1) ^a	0.1 (-0.4–0.6)
London	0.2 (-0.6–1.0)	0.8 (0.3–1.3)	0.8 (0.3–1.4) ^a	-0.3 (-1.1–0.4)
SO ₂				
Hong Kong	1.4 (0.4–2.3)	2.1 (1.4–2.9)	2.0 (1.1–2.8)	2.1 (1.3–2.8) ^a
London	1.4 (0.6–2.3)	1.6 (0.9–2.2)	2.2 (1.2–3.2)	1.6 (1.0–2.2) ^a

^aEstimates from the single-pollutant model.

exposure–response relationship was observed for concentrations of O₃ < 20 µg/m³ (mainly in the warm season); in London (mainly in the cool season), a neutral relationship was found. For levels of O₃ > 20 µg/m³, there were similar positive linear relationships in both cities (Figure 3).

For cardiac admissions and PM₁₀ between the 10th and 90th percentiles (i.e., 25–87 µg/m³ in Hong Kong and 16–46 µg/m³ in London), both cities showed positive exposure–response relationships (Figure 3). We observed a negative linear association for O₃ across the range of the pollutant in London, whereas in Hong Kong we observed a “J”-shaped exposure–response relationship, indicating a positive association between cardiac admissions and the higher levels of O₃ (data not shown).

For the other exposure–response relationship, there were similarities as well as dissimilarities between the two cities (Figures 4–7), which was quite in agreement with those results presented in Table 5 for the same lag 0–1 day effects.

Discussion

Validity of results. The analytic method was the same in each city and followed the approach adopted by the APHEA collaboration. One of the present authors (R.W.A.) was responsible for analyzing the APHEA 2 respiratory admissions data and worked closely with researchers in Hong Kong to ensure that the application of methods was the same in each city. One feature of this method of Poisson regression is that seasonal, long-term trends and weather factors were modeled using nonparametric methods. This method is widely accepted and has been found to yield comparable results to the earlier method, which uses sinusoidal models for seasonal control (29). It also gives similar results to methods that use a synoptic approach to control for weather factors (30). In a sensitivity analysis, the method of parametric seasonal control was applied to selected series in both London (11) and Hong Kong; results were similar to those observed using the current method, which used generalized additive models (data not shown). The data on air pollution, weather, and outcomes were defined in an identical manner. We did not validate the consistency of hospital diagnosis, but since medical practice and the death certification procedure in Hong Kong has been strongly influenced by British and Commonwealth medical education, it is unlikely that there were major differences. In any case, the adoption of some broad categories (lower respiratory disease and cardiac disease) should have absorbed diagnostic transfer within those groups. The lower level of asthma admissions corresponds to the

known lower prevalence of asthma in Hong Kong (31,32). The lack of statistically significant association in asthmatic admissions may be due to the small numbers, relative to the other categories, and low statistical power to detect a significant association.

Respiratory admissions. The results for respiratory admissions were similar when the best single day lag was chosen, with all pollutants showing significant effects in both cities. These results are in line with many other studies (33). However, when the 0–1 day lag was compared, the cities were similar only for O₃, with only Hong Kong showing significant effects of the other pollutants. This may be explained by the fact that in Hong Kong the effects of PM₁₀, NO₂, and SO₂ were greatest at early lags, whereas in London the effects were greater with later lags. We have considered whether this difference in lags could be explained by the different primary health care systems. In Hong Kong this is a combination of private practitioners (the great majority) and public out-patient clinics, and heavy use of hospital accident and emergency departments. Perhaps this results in more rapid referral to hospital of persons with severe lower respiratory disease than in London, where the state-provided primary care system takes more responsibility for treating moderately severe disease at home and for controlling access to hospital facilities. We observed that the lag patterns for respiratory mortality associated with NO₂, PM₁₀, and SO₂ (Figure 8) (34,35) also followed the respective patterns for respiratory admissions (Figure 1). The other difference in the effects of NO₂, PM₁₀, and SO₂ was that in Hong Kong, the effects were stronger in the cool season, whereas they were stronger in the warm season in London. One common factor here is that the humidity is lower in the season showing the largest effects. It may also be relevant that the average levels of all pollutants apart from SO₂ are highest during the cool season in Hong Kong.

The strong associations between respiratory admissions and NO₂ and PM₁₀ suggest that traffic may be an important source of toxic pollution. In both cities, it has been estimated that only a minority (about 20–40%) of PM₁₀ particles in the ambient air (not just for emissions from various sources) is derived from local traffic (36,37). This is consistent with the finding that NO₂ retains its strong association in two pollutant models while PM₁₀ does not.

O₃ showed consistent significant effects on respiratory admissions, irrespective of whether the mean lag 0–1 days or best single day lag was chosen. The exposure–response relationships with O₃ were linear in both cities when concentrations were

> 20 µg/m³, but in Hong Kong, a negative relationship was observed below this level. It could be postulated that the Hong Kong population would be more resistant to O₃ because the diet is higher in antioxidants and because air conditioning is used in most closed spaces, but our data suggest that both populations are equally susceptible. It is relevant to note that O₃ also shows associations with respiratory admissions in a range of European cities, with little heterogeneity (33,38). In both London and Hong Kong, the association with O₃ was very robust to the inclusion of other pollutants in the model.

Cardiac admissions. The results for cardiac admissions were similar for both cities in respect to NO₂, PM₁₀, and SO₂. This was irrespective of whether the mean lag 0–1 or the best lag was chosen, because in contrast to respiratory admissions, both cities displayed the same lag patterns, with lower risks at longer lags. These results add to the accu-

mulating evidence worldwide that air pollution has short-term effects on cardiac admissions. Our evidence indicates that within the cardiac group of diagnoses, there are also effects on ischemic heart disease, but we do not know from this study if the same applies to other diagnoses such as cardiac failure or cardiac arrhythmias. However, we previously demonstrated an effect of O₃ in the cool season on admissions for these cardiac events in the elderly in Hong Kong (8). The exposure–response relationships with NO₂, PM₁₀, and SO₂ were linear in both cities, and there were similar seasonal associations, with both cities having larger effects in the cool season. The two-pollutant models also showed considerable similarities, with NO₂ being robust to the inclusion of PM₁₀ in the models but affected to some extent by SO₂. In both cities, the effect of SO₂ retained its statistical significance in the presence of all the other pollutants.

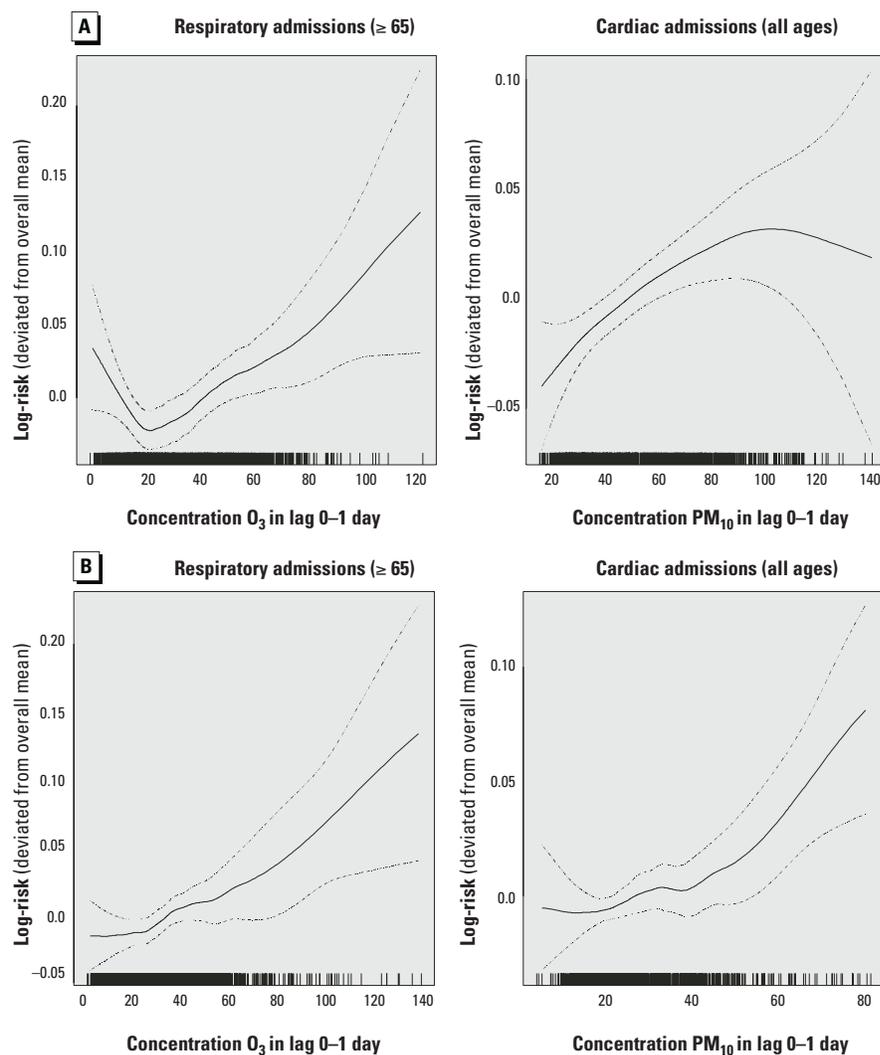


Figure 3. Exposure (µg/m³) and response relationships for O₃ and respiratory admissions and for PM₁₀ and cardiac admissions in (A) Hong Kong and (B) London. The density of the vertical bars on the x-axis shows the distribution of the pollutant concentration data.

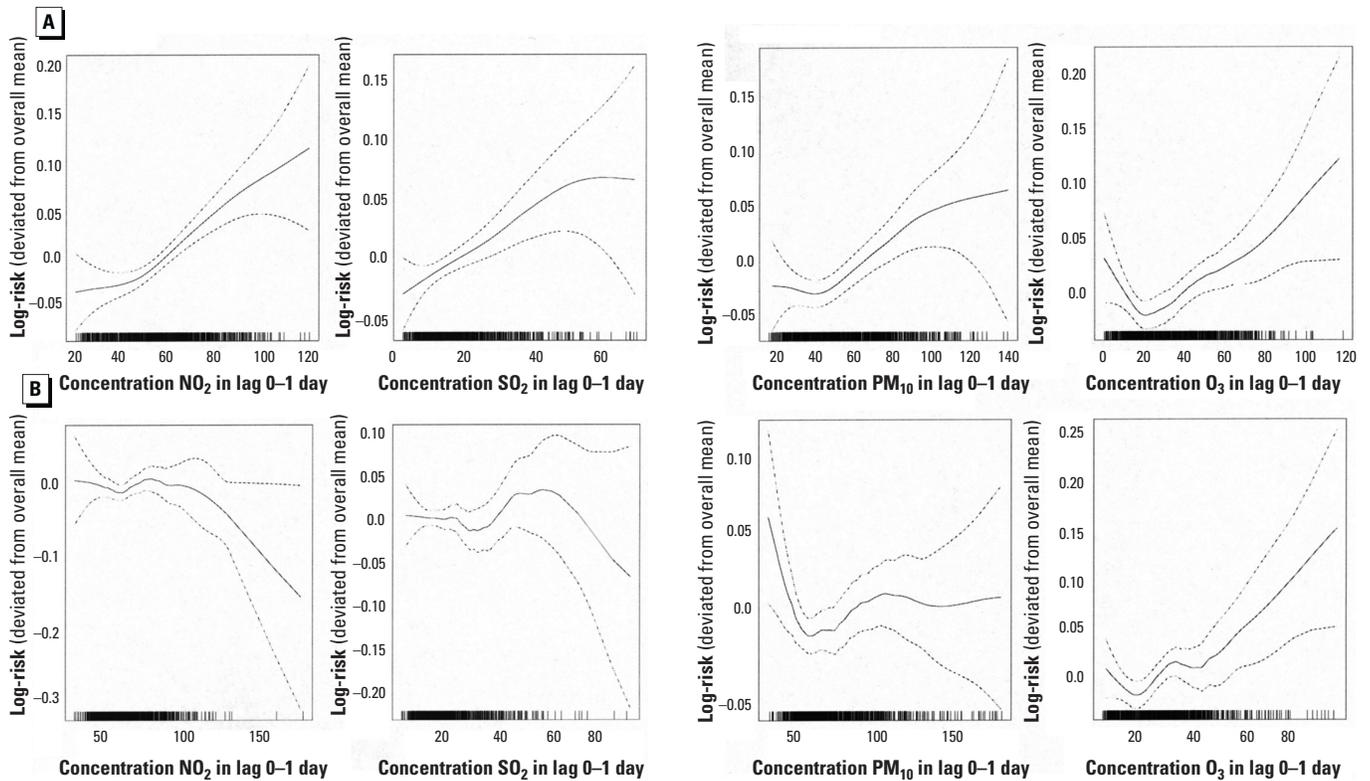


Figure 4. Exposure response curves for respiratory admissions (≥ 65 years of age) in (A) Hong Kong and (B) London for all pollutants under study.

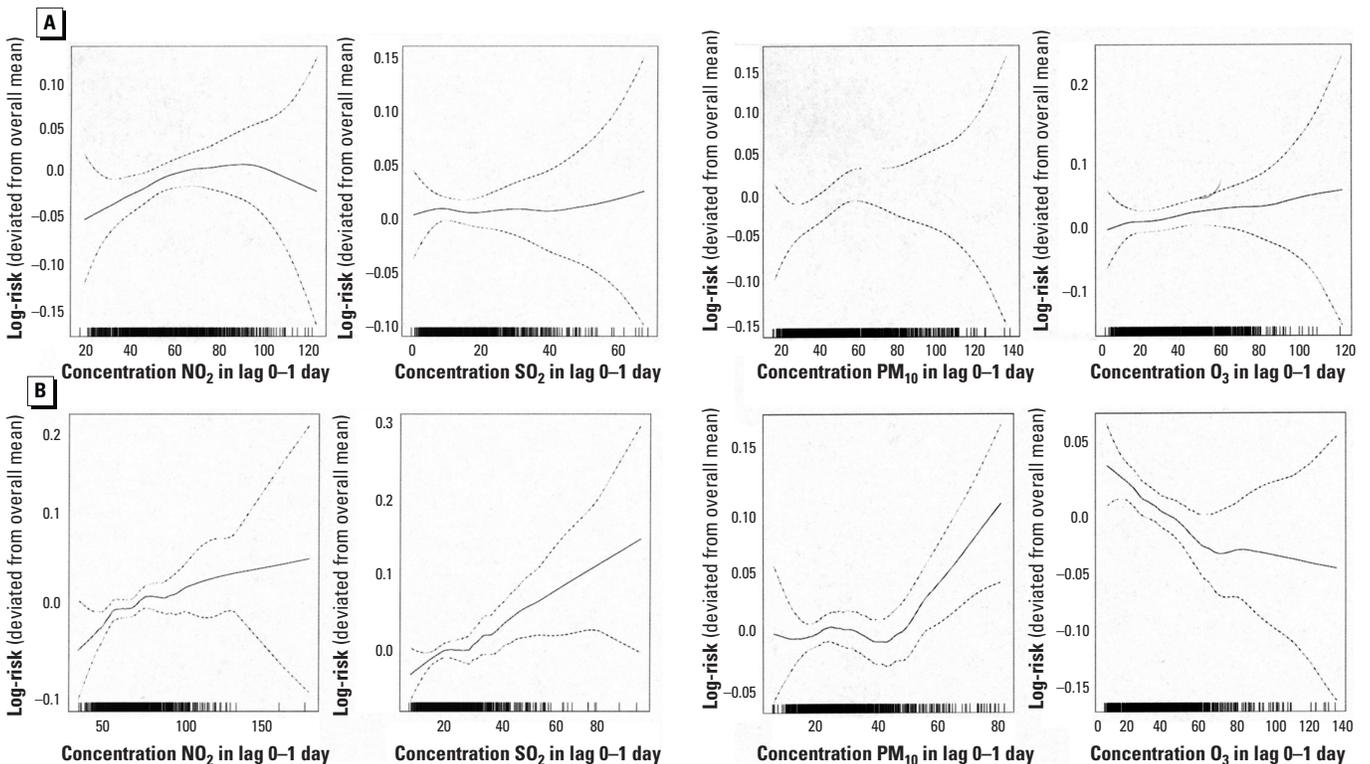


Figure 5. Exposure response curves for IHD (all ages) in (A) Hong Kong and (B) London for all pollutants under study.

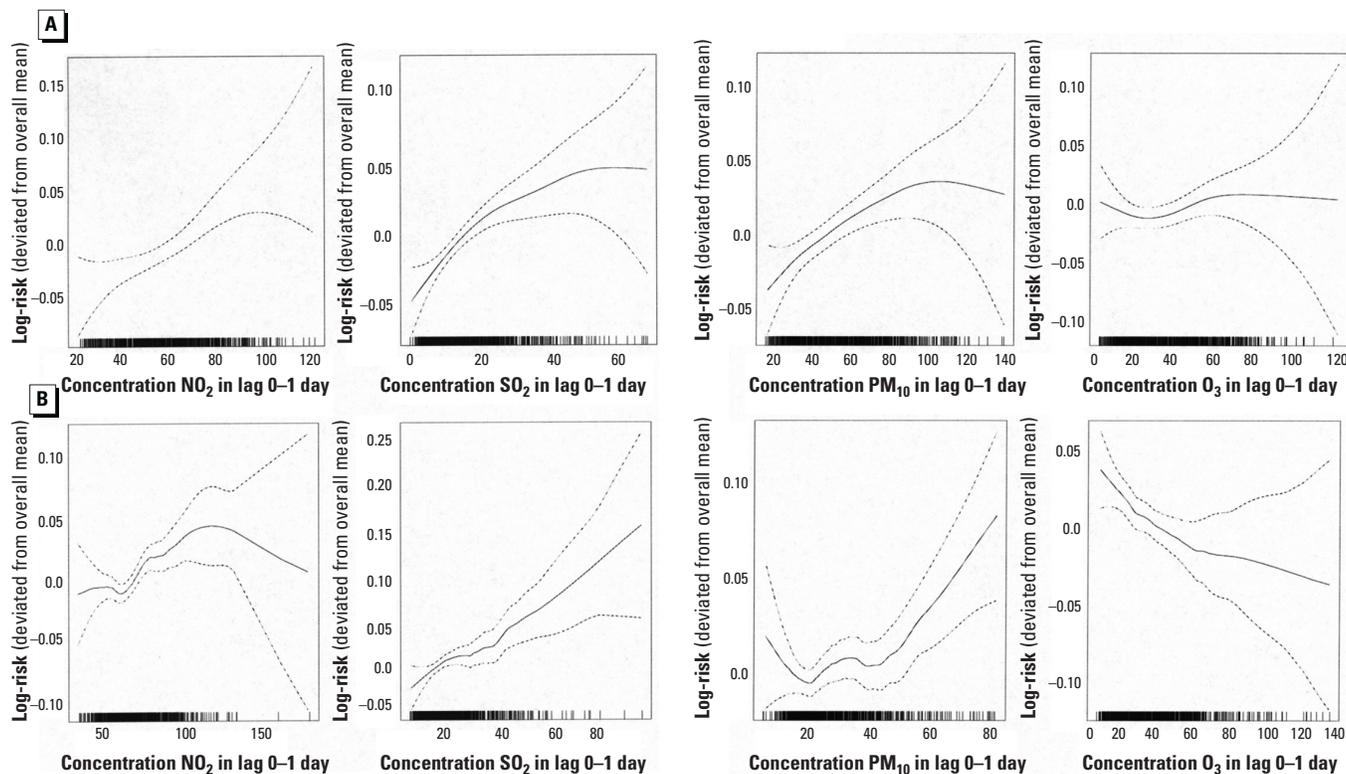


Figure 6. Exposure response curves for cardiac admissions (all ages) in (A) Hong Kong and (B) London for all pollutants under study.

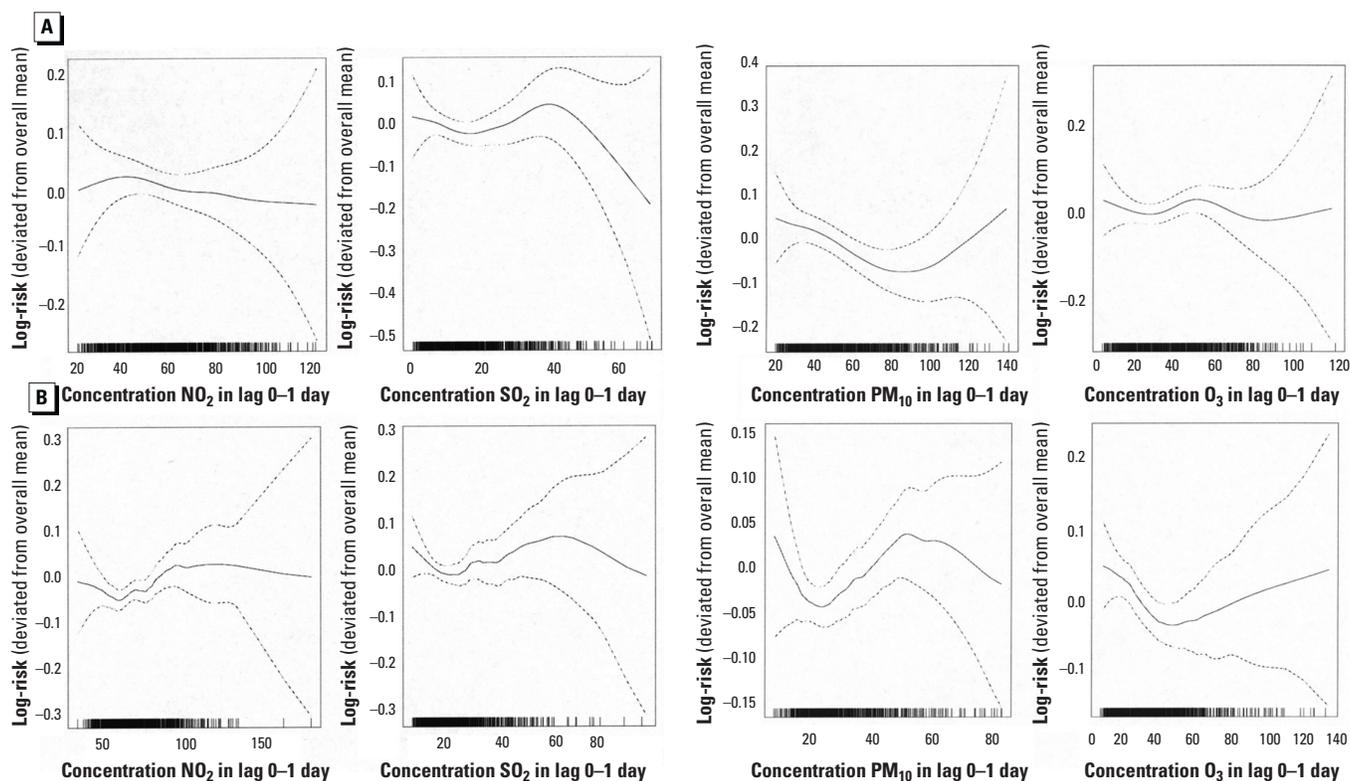


Figure 7. Exposure response curves for asthma (15–64 years of age) in (A) Hong Kong and (B) London for all pollutants under study.

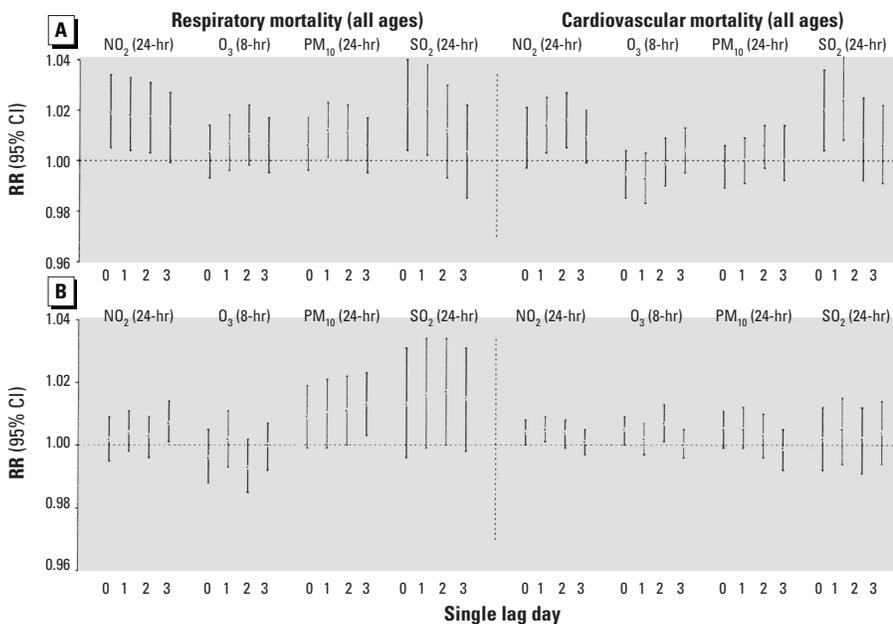


Figure 8. Effect of pollutants at a single day lag on mortality due to cardiovascular and respiratory diseases in (A) Hong Kong and (B) London. Values shown are relative risk (RR) and 95% confidence interval (CI) for a $10 \mu\text{g}/\text{m}^3$ increase in concentration.

Relevance of results. The principal aim of this study was to determine if the effects of air pollution on daily hospital admissions are consistent between Hong Kong and London. An important component of causal thinking in observational studies is whether the associations are consistent in widely varying environments. This is one way in which concerns about unknown or inadequately controlled confounding can be addressed. In air pollution time-series studies, this is especially important because other components of causal reasoning such as size of effect, biological plausibility, and coherence are less convincing than many would wish. Hong Kong and London differ markedly in many respects that could affect confounding or effect modification, especially in climate and lifestyle. We have based our comparison on the size, significance, and direction of estimates of effect, lag pattern, exposure–response relationship, and seasonal effects. We conclude overall that there are considerable similarities in the effects of each pollutant, although the explanations for some differences, including a lack of association with asthma admissions in adults in Hong Kong, differences in the lag pattern for all respiratory admissions, and opposite directions of effect for O_3 and cardiac admissions, remain uncertain. Overall, we consider the similarities to outweigh the differences; thus, we conclude that our study strengthens the argument for the causality of air pollution associations with hospital admissions.

The comparison has done less to clarify which component of the pollution mixture is

important. The O_3 associations with respiratory disease are at least independent of other pollutants and in line with studies elsewhere. There is also evidence that O_3 is potentially toxic at near ambient levels (2). For cardiac admissions, the PM_{10} associations were less independent of NO_2 (and in some cases SO_2) than the reverse. Toxicologic evidence suggests that NO_2 and SO_2 are unlikely to have effects at this level, which points toward them being surrogates for some other toxic component. PM_{10} is widely regarded as important in spite of meagre human toxicologic corroboration. The cities were similar in the proportion of fine particles comprising PM_{10} and in other components such as sulfate (as an indicator of secondary particles) and carbon (as an indicator of primary particles) (36,39–41). Our study, like many others, suggests that traffic sources are important but cannot be more specific. Analysis using many, rather than only two, cities may be one way of learning more about the effects of different pollution mixtures (42).

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