The Role of Particulate Size and Chemistry in the Association between Summertime Ambient Air Pollution and Hospitalization for Cardiorespiratory Diseases

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In order to address the role that the ambient air pollution mix, comprised of gaseous pollutants and various physical and chemical measures of particulate matter, plays in exacerbating cardiorespiratory disease, daily measures of fine and coarse particulate mass, aerosol chemistry (sulfates and acidity), and gaseous pollution (ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide) were collected in Toronto, Ontario, Canada, in the summers of 1992, 1993, and 1994. These time series were then compared with concurrent data on the number of daily admissions to hospitals for either cardiac diseases (ischemic heart disease, heart failure, and dysrthymias) or respiratory diseases (tracheobronchitis, chronic obstructive lung disease, asthma, and pneumonia). After adjusting the admission time series for long-term temporal trends, seasonal variations, the effects of short-term epidemics, day of the week effects, and ambient temperature and dew point temperature, positive associations were observed for all ambient air pollutants for both respiratory and cardiac diseases. Ozone was least sensitive to adjustment for the gaseous and particulate pollution measures. However, the association between the health outcomes and carbon monoxide, fine and coarse mass, sulfate levels and aerosol acidity could be explained by adjustment for exposure to gaseous pollutants. Increases in ozone, nitrogen dioxide, and sulfur dioxide equivalent to their interquartile ranges corresponded to an 11% and 13% increase in daily hospitalizations for respiratory and cardiac diseases, respectively. The inclusion of any one of the particulate air pollutants in multiple regression models did not increase these percentages. Particle mass and chemistry could not be identified as an independent risk factor for the exacerbation of cardiorespiratory diseases in this study beyond that attributable to climate and gaseous air pollution. We recommend that effects of particulate matter on health be assessed in conjunction with temporally covarying gaseous air pollutants. *Key words*: air pollution, heart disease, ozone, particulate matter, respiratory disease. *Environ Health Perspect* 105:614–620 (1997)

Daily variations in particulate mass have been associated with concurrent fluctuations in both mortality and admission to hospitals for cardiorespiratory diseases in a number of recent studies (*1*). For most of these investigations, only particulate mass of undefined aerodynamic diameter (total suspended particles or TSP) or of less than 10 μm (thoracic particles, denoted by TP or PM_{10}) have been available for study. Some recent studies, however, have demonstrated a greater association with premature mortality (*2*) and hospitalization (*3*) with particulate matter of aerodynamic diameter less than 2.5 μm (fine particles, denoted by FP or $PM_{2.5}$). Other studies have suggested a role for chemistry, including the sulfate and acid concentration of the particle (*4*). However, limited attention has been given in these studies to the role the gaseous pollutants ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO) play in exacerbating cardiorespiratory disease, and their potential confounding influence in estimating the magnitude of the risk attributed to particulate mass alone.

Daily measures of particulate mass (TP, FP), aerosol chemistry (sulfates and acidity), an optical measure of particulate pollution, the coefficient of haze (COH), and gaseous pollution (O_3, NO_2, SO_2, and CO) were collected in Toronto, Ontario, Canada, in the summers of 1992, 1993, and 1994. These time series were then compared with concurrent data on the number of daily admissions to hospitals for either cardiac (ischemic heart disease, heart failure, and dysrthymias) or respiratory (tracheobronchitis, chronic obstructive lung disease including asthma, and pneumonia) diseases in order to examine the effects of the ambient air pollution mix.

Methods

Study population. Metropolitan Toronto consists of six cities (Toronto, North York, East York, Etobicoke, Scarborough, and York) located on the north shore of Lake Ontario with a population of 2.36 million people as indicated by the 1991 census. The major ambient air pollution sources in the area are transportation related, industrial processes, power generation, and regional or long-range transport.

Environmental data. Twenty-four hour measurements of fine and coarse particle mass concentrations were made via a dichotomous sampler starting at 8 A.M. each day at a centrally located downtown Toronto air quality monitoring site. The site was selected to be representative of the greater metropolitan Toronto area with respect to these atmospheric measurements (*5*). Thoracic particulate mass concentrations (PM_{10}) were obtained by summing concentrations of fine and coarse mass concentrations. Daily measurements were made for the 388-day period: 16 June 1992 to 24 September 1992; 1 May 1993 to 22 September 1993; and 1 May 1994 to 22 September 1994. Simultaneous daily measurements of fine particulate strong acidity (H^+) and sulfate (SO_4) were made independent of the mass measurements using an annular denuder system (*5*) colocated with the dichotomous sampler. Daily measurements were taken in the summer since this is the period of the highest concentrations of these pollutants. Missing acid and sulfate data (9%) and particle mass data (3%) were imputed using linear interpolation to obtain a complete time series for analyses.

Hourly measurements of the gaseous pollutants O_3, NO_2, SO_2, and CO and an optical measure of particulate air pollution (COH) were obtained from multiple population-based monitoring stations in metropolitan Toronto for the period concurrent with the particle measurements. The number of monitoring stations varied over the 3-year period of observation: 7–9 stations for O_3, 4–6 stations for SO_2, 5–6 stations for CO, 3–6 stations for COH, and 6–11 stations for NO_2. The hourly measurements were averaged among stations. Three daily summary measures were then calculated for each of the 388 days of observation: daily 24-hour average (avg), daytime average readings from 8 A.M. to 8 P.M. (day), and the daily 1-hr maximum reading (max). Complete summary data were available for analyses using this method. Complete data time series were also obtained for the daily maximum temperature (T) and

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dew point temperature (DP) from Pearson International Airport in the Toronto area.

Hospital admission data. A computerized record is maintained by the Ontario Ministry of Health for each patient discharged from a hospital in Ontario. The number of daily hospital admissions concurrent with the period of environmental data were abstracted for which the discharge diagnosis (*International Classification of Diseases*, 9th Revision; ICD-9) was for a cardiac disease (ICD-9 410–414, ischemic heart disease; 427, cardiac dysrhythmias; 428, heart failure) or respiratory disease (ICD-9 464–466,490, tracheobronchitis; 480–486, pneumonia; 491–494,496, chronic obstructive lung disease). Only unscheduled admissions of those patients not transferred from another institution were selected. Each patient must have resided in the census division covering the six cities constituting metropolitan Toronto, and only acute care hospitals located in the census division were considered. Hospital admissions for newborns and stillborns were not examined.

Analysis. Daily readings of the environmental predictors were related to the daily number of hospital admissions for either respiratory or cardiac diseases in metropolitan Toronto using the relative risk regression model

$$E(y_t) = S_t D_t \exp(\beta' x_t).$$

Here, $E(y_t)$ is the expected number of admissions on the tth day of sampling, S_t is a 19-day linear filter of the number of daily admissions on the tth day of sampling (*6*), D_t is a time series consisting of a repetition of seven unique values represent-

Table 1. Summary statistics of environmental variables for the summers of 1992, 1993, and 1994, in Toronto

			Percentiles						
Variable (units)[a]	Mean	CV	0	5	25	50	75	95	100
O_3 (ppb)	41.2	37	9	22	29	39	51	69	91
NO_2 (ppb)	38.5	29	12	22	31	38	45	58	81
SO_2 (ppb)	7.9	64	0	1	4	7	11	18	26
CO (ppm)	1.8	41	0.6	0.9	1.3	1.7	2.0	3.2	5.5
COH (10^3 ln ft)	0.8	49	0.1	0.3	0.5	0.8	1.1	1.5	2.5
H^+(nmol/m^3)	5.0	188	0	0	0	1	6	23	75
SO_4 (nmol/m^3)	57.1	124	0	5	14	33	71	229	489
TP (μg/m^3)	28.4	57	4	10	16	25	38	58	102
FP (μg/m^3)	16.8	69	1	4	8	14	23	40	66
CP (μg/m^3)	11.6	59	1	4	7	10	14	23	56
T (°C)	23	21	8	12	20	24	27	31	36
DP (°C)	15	33	-1	6	11	15	19	22	24

Abbreviations: CV, coefficient of variation (standard deviation/mean); COH, coefficient of haze; TP, thoracic particles; FP, fine particles; CP, coarse particles; T, temperature; DP, dewpoint.
[a]Statistics for O_3, NO_2, SO_2, CO, and COH are based on the daily 1-hr maximum concentration, while the remaining particulate pollutants are based on daily averages; T and DP are based on the daily maximum readings.

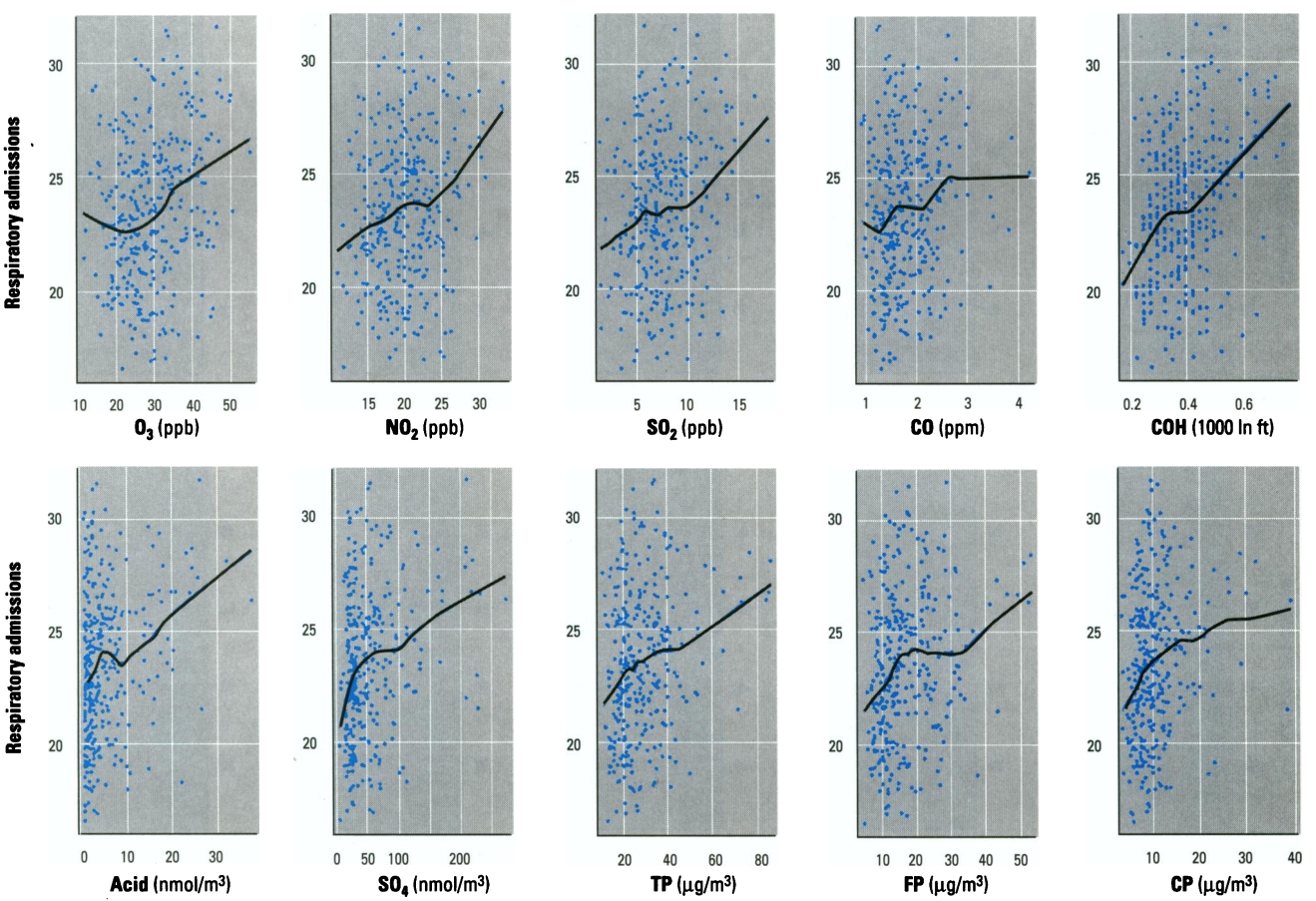

Figure 1. Adjusted daily hospital admission rates for respiratory diseases (y_t/S_tD_t, normalized to the average admission rate) plotted against air pollution concentrations based on metrics given in Table 2. The nonparametric smoothed curve using LOESS is also represented. Abbreviations: TP, thoracic particles; FP, fine particles; CP, coarse particles; COH, coefficient of haze.

ing the average admission rate for each day of the week, and β is a vector of unknown log-relative risk regression parameters relating the vector of environmental predictors x_t to the frequency of daily hospitalizations.

Because the responses of interest are counts, the residual variation was assumed to be proportional to the expected response, accommodating overdispersion or underdispersion relative to Poisson variation. The length of the linear filter S_t and the filter weights were specifically designed to remove long-term trends, as well as seasonal and subseasonal variation in the admission series due to influenza epidemics. D_t removes differences in admission rates among days of the week before an examination of the environmental predictors.

Estimates of the log-relative risks and their standard errors were obtained using generalized estimating equation methods (*7*). Statistical chi-square tests for the adequacy of a log-linear association between the environmental predictors and hospitalization rates were examined by comparing the residual variance of models fit with log-linear regressors and those with nonparametric smoothed functions (LOESS) of the environmental predictors using generalized additive models (*8*).

Several potential exposure measures or pollution metrics were computed for each pollutant as well as for temperature. Air pollution concentrations based on each summary measure were obtained for the day of admission, as well as 1, 2, 3, and 4 days prior to the date of admission. Multiday average concentrations for each of the three summary measures were also calculated for 2-, 3-, 4-, and 5-day periods. The end date of these multiple day averages were also lagged 1, 2, 3, and 4 days prior to the date of admission to hospital. Similar lags and multiday averages were also constructed for daily maximum temperature and daily maximum dew point temperature.

The association with hospitalization for respiratory and cardiac diseases was determined independently for each pollution metric. For each pollutant and climate variable, the daily metric maximizing the ratio of the log-linear regression coefficient to its standard error (t-ratio) was selected for further analysis. These daily metrics are identified according to their daily summary measure (avg, day, max), number of days averaged (1–5), and number of days lagged (0–4).

Results

On average, 23.7 patients were admitted to hospitals daily in metropolitan Toronto for respiratory diseases, ranging from a minimum of 9 patients to a maximum of 56 admissions, while 42.6 patients were admitted daily for cardiac diseases, with a range of 23–66 people. Summary statistics for the air pollution variables are given in Table 1.

The metric selected for both temperature and dew point temperature was based on a 4-day average lagged 1 day. For respiratory diseases, the relative risk (RR) for temperature evaluated at the interquartile range (IQR = 5.33°C) was 1.028 with a t-ratio of 1.66. The corresponding relative risk for dew point temperature (IQR = 5.55°C) was 1.032 with a t-ratio of 1.74. The effects of temperature and dew point temperature were somewhat weaker for cardiac diseases (RR = 1.015 and t = 1.05 for temperature, and RR = 1.018 and t = 1.16

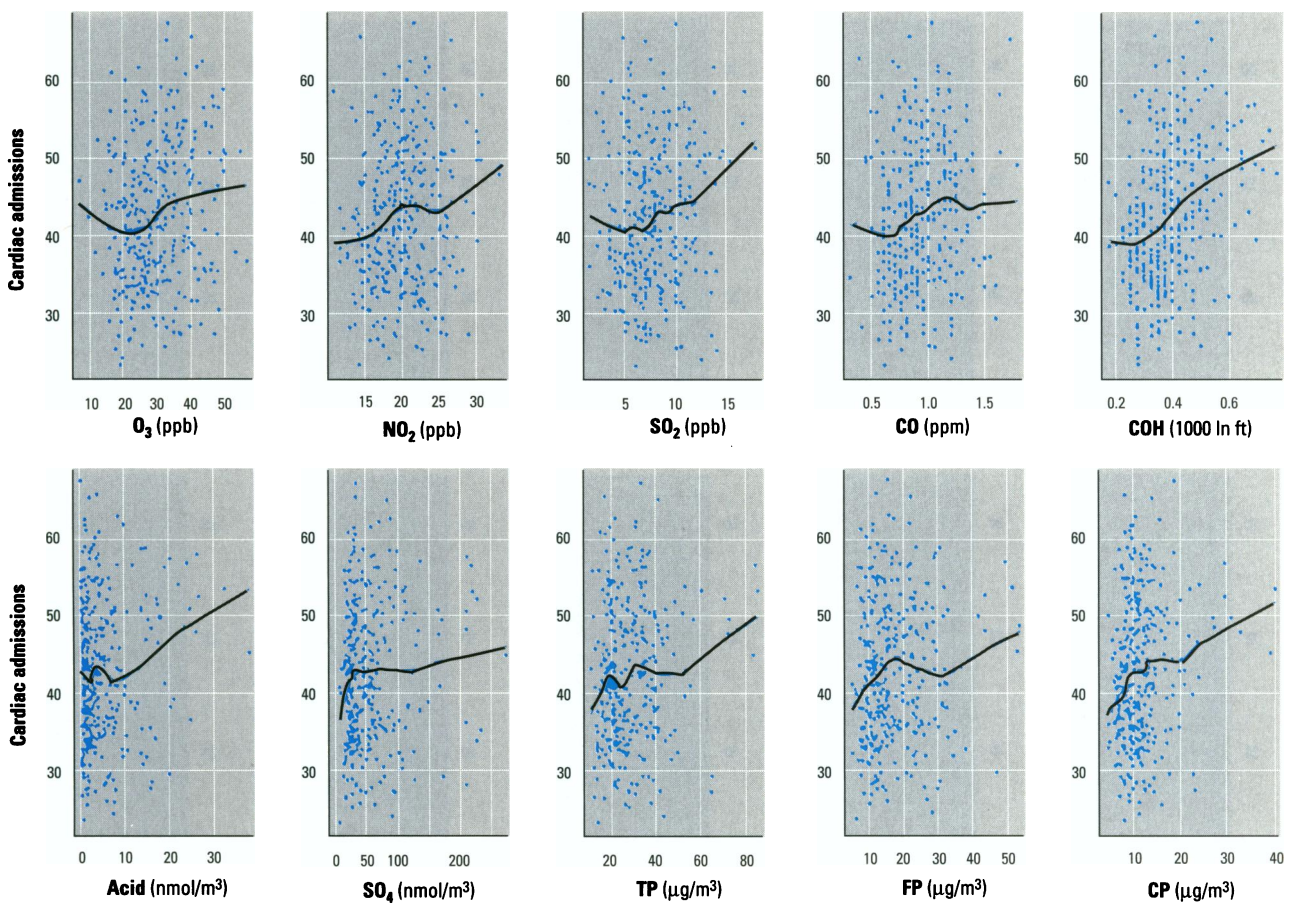

Figure 2. Adjusted daily hospital admission rates for cardiac diseases (y_t/S_tD_t, normalized to the average admission rate) plotted aganist air pollution concentrations based on metrics given in Table 2. The nonparametric smoothed curve using LOESS is also represented. Abbreviations: TP, thoracic particles; FP, fine particles; CP, coarse particles; COH, coefficient of haze.

for dew point temperature).

The relations between the ambient air pollutants and hospitalizations for respiratory and cardiac diseases are displayed in Figures 1 and 2, respectively. The ratio y_t/S_tD_t, normalized to the average admission rate, is plotted against the air pollution metrics given in Table 2, for each of the 388 days of observation. A nonparametric smoothed representation of the concentration–response relation using LOESS (*8*) is also presented. A nonlinear association was apparent for some of the air pollutants. Based on the chi-square tests, there was no evidence to suggest that a linear association with hospitalization rates was not adequate to explain variation in daily admissions for each air pollutant and climate variable with both health outcomes considered ($p>0.9$). Therefore, the linear form of the environmental predictors was used in all further analyses.

The relative risk for each of the air pollutants examined singly or adjusted for both temperature and dew point temperature are given in Table 2. Simultaneous control for climate tended to decrease the air pollution relative risks for respiratory diseases and increase the risk for cardiac diseases. After adjustment for temperature and dew point temperature, the strongest associations with hospitalizations as measured by the t-ratio for both respiratory and cardiac diseases were observed for COH and ozone.

The influence of the method of adjusting the hospital admission time series for temporal trends was examined by repeating the analysis reported in Table 2 using a LOESS smooth of time (span equivalent to 19 days) in place of the 19-day linear filter S_t. Using the LOESS smooth of time, the relative risks were greater than those reported in Table 2, with average increases of 11% for respiratory diseases and 8% for cardiac diseases.

Before examining the results from multiple pollutant regression models, it is instructive to examine the correlation (r) among the environmental variables (Table 3). The correlations were determined using the metrics given in Table 2 for respiratory diseases. Similar correlations were found employing those metrics used in the analysis of cardiac outcomes. The acid and sulfate content of the particulate aerosol are highly correlated with each other (r = 0.86) and are also highly correlated with FP (r = 0.76 and 0.79), while their association is weaker with CP (r = 0.48 and 0.55). FP and CP are also highly correlated with each other (r = 0.72). The gases NO_2 (r = 0.61), SO_2 (r = 0.55), and CO (r = 0.42) are all correlated with TP. However, O_3 is weakly correlated with TP (r = 0.23). In fact, O_3 is weakly correlated with all the other air pollutants (r ranges from 32 with FP to 2 with CO) except sulfates (r = 0.53). There is a strong regional source contribution to both sulfates and O_3 in this area. Dew point temperature tends to be more correlated with the other pollutants than temperature except for O_3. This is due to the strong correlation between temperature and the factors (i.e., ultraviolet radiation, light winds with transport from a southerly direction) influencing O_3 formation around and upwind of Toronto.

The relative risks for two selected pollutant models are given in Table 4 for respiratory diseases and in Table 5 for cardiac outcomes. Each of the particle-related measures was regressed with one of the gaseous pollutants. All models include temperature and dew point temperature. For respiratory diseases, the sulfate effect (RR = 1.029) was reduced in half after adjustment for O_3 (RR = 1.015). A similar attenuation was noted by Burnett et al. (*9*) in their analysis of hospitalizations for respiratory diseases in southern Ontario and by Thurston et al. (*3*) for Toronto. Simultaneous adjustment for CO had little effect on the relative risks for TP, FP, or CP; adjustment for either O_3 or SO_2 decreased the relative risks slightly; however, NO_2 clearly attenuated the relative risks for these particulate mass measurements. The relative risks for TP, FP, and CP were insensitive to adjustment for CO for cardiac diseases. Either O_3, SO_2, or NO_2 could attenuate the relative risks for FP. Attenuation was greatest for TP and CP with NO_2.

Little evidence of an association with CO was observed for either health outcome when regressed with any other ambient air pollutant. The relative risks for O_3 are relatively stable after adjustment for the particle-related pollutants, ranging from 1.052 to 1.063 for respiratory outcomes and 1.051 to 1.069 for hospitalizations for cardiac illnesses. The stability of these associations were examined by year of study. The O_3 relative risks, adjusted for temperature and dew point temperature, were 1.061 for 1992, 1.062 for 1993, and 1.088 for 1994.

Table 2. Relative risks for ambient air pollutants regressed singly or adjusted for temperature and dew point temperature, evaluated at the interquartile range, for admission to hospital for respiratory or cardiac diseases

		Respiratory admissions			Cardiac admissions		
Pollutant (units)	IQR	Metric[a]	RR[+]	RR[++]	Metric	RR[+]	RR[++]
O_3 (ppb)	11.50	Day,3,1	1.068 (6.19)	1.064 (5.13)	Day,3,2	1.057 (3.52)	1.074 (3.85)
NO_2 (ppb)	5.75	Day,5,0	1.048 (5.09)	1.044 (4.47)	Day,4,0	1.049 (3.09)	1.049 (3.13)
SO_2 (ppb)	4.00	Max,4,0	1.040 (4.14)	1.033 (3.16)	Max,4,0	1.041 (2.66)	1.043 (2.61)
CO (ppm)	0.75	Max,3,2	1.022 (3.84)	1.017 (1.70)	Day,2,0	1.019 (2.13)	1.017 (1.99)
COH (10^3 ln ft)	0.25	Day,5,0	1.072 (7.15)	1.037 (5.36)	Day,4,0	1.057 (6.24)	1.062 (5.63)
H^+ (nmol/m^3)	5.25	Avg,2,0	1.029 (4.89)	1.024 (3.48)	Avg,4,2	1.021 (2.25)	1.024 (2.30)
SO_4 (nmol/m^3)	40.00	Avg,4,1	1.030 (5.14)	1.029 (4.07)	Avg,4,2	1.015 (1.60)	1.017 (1.56)
TP (μg/m^3)	14.25	Avg,4,0	1.036 (4.51)	1.030 (3.42)	Avg,4,1	1.028 (2.17)	1.033 (2.24)
FP (μg/m^3)	11.00	Avg,4,1	1.040 (4.46)	1.037 (3.29)	Avg,4,2	1.025 (1.72)	1.031 (1.80)
CP (μg/m^3)	4.75	Avg,5,0	1.023 (4.16)	1.023 (3.41)	Avg,4,0	1.035 (3.47)	1.036 (3.41)

Abbreviations: IQR, interquartile range for pollution metric selected; RR[+], relative risk for specified pollutant evaluated at the IQR; RR[++], relative risk for specified pollutant adjusted for temperature and dew point temperature and evaluated at the IQR; COH, coefficient of haze; TP, thoracic particles; FP, fine particles; CP, coarse particles; avg, average; max, maximum. The ratio of log-relative risk to standard error is given in parentheses.

[a]Metric defined by the triple daily summary measure (avg, day, max), days averaged, and days lagged.

Table 3. Pearson correlations (×100) of environmental variables[a]

	H^+	SO_4	TP	FP	CP	COH	O_3	NO_2	SO_2	CO	T
SO_4	86	100									
TP	62	67	100								
FP	76	79	89	100							
CP	48	55	88	72	100						
COH	33	47	49	45	47	100					
O_3	34	53	23	32	20	31	100				
NO_2	25	34	61	45	57	61	7	100			
SO_2	45	42	55	49	44	50	18	46	100		
CO	33	28	42	42	34	23	2	25	37	100	
T	33	41	34	37	23	31	60	-6	32	16	100
DP	48	52	42	54	23	34	47	-2	30	21	87

Abbreviations: TP, thoracic particles; FP, fine particles; CP, coarse particles; COH, coefficient of haze.

[a]Correlations are determined based on the daily summary measures and number of days averaged for each pollutant that are given in Table 2 for respiratory admissions. For temperature (T) and dew point temperature (DP) 4-day averages lagged 1 day were used.

Table 4. Relative risk[a] for respiratory hospitalizations for particle related pollutants adjusted for a single gaseous pollutant, temperature, and dew point temperature

	GP			
Particle pollutant	O_3	NO_2	SO_2	CO
COH	1.032 (4.58)	1.030 (3.08)	1.034 (4.32)	1.037 (5.10)
GP	1.054 (4.38)	1.018 (1.36)	1.012 (1.10)	1.009 (0.90)
H^+	1.016 (2.24)	1.016 (2.26)	1.018 (2.37)	1.023 (3.12)
GP	1.058 (4.39)	1.037 (3.61)	1.022 (1.96)	1.008 (0.83)
SO_4	1.015 (1.91)	1.019 (2.39)	1.024 (3.15)	1.028 (3.19)
GP	1.052 (3.91)	1.033 (3.05)	1.021 (1.93)	1.009 (0.94)
TP	1.027 (3.16)	1.006 (0.53)	1.022 (2.11)	1.029 (2.97)
GP	1.063 (4.96)	1.039 (2.85)	1.021 (1.72)	1.005 (0.49)
FP	1.027 (2.33)	1.013 (1.00)	1.027 (2.08)	1.035 (2.84)
GP	1.059 (4.56)	1.037 (3.13)	1.022 (1.92)	1.005 (0.51)
CP	1.020 (3.04)	1.009 (1.01)	1.018 (2.36)	1.022 (3.01)
GP	1.062 (4.89)	1.037 (2.96)	1.023 (2.03)	1.007 (0.72)

Abbreviations: GP, gaseous pollutant; COH, coefficient of haze; TP, thoracic particles; FP, fine particles; CP, coarse particles. The ratio of log-relative risk to standard error is given in parentheses.
[a]Relative risk evaluated at the temperature evaluated at the interquartile range (see Table 2).

Table 5. Relative risk[a] for cardiac hospitalizations for particle related pollutants adjusted for a single gaseous pollutant, temperature, and dew point temperature

	GP			
Particle pollutant	O_3	NO_2	SO_2	CO
COH	1.057 (5.03)	1.069 (4.50)	1.062 (4.91)	1.063 (5.50)
GP	1.051 (3.03)	0.990 (0.51)	1.004 (0.26)	1.007 (0.72)
H^+	1.013 (1.16)	1.021 (2.26)	1.019 (1.67)	1.023 (2.14)
GP	1.059 (3.31)	1.038 (2.87)	1.036 (2.09)	1.010 (0.92)
SO_4	0.996 (0.37)	1.012 (1.05)	1.012 (1.17)	1.016 (1.43)
GP	1.069 (3.53)	1.039 (2.89)	1.040 (2.38)	1.011 (1.06)
TP	1.025 (1.68)	1.010 (0.59)	1.020 (1.28)	1.030 (1.98)
GP	1.062 (3.55)	1.036 (2.28)	1.034 (1.86)	1.007 (0.69)
FP	1.014 (0.78)	1.017 (0.95)	1.020 (1.11)	1.028 (1.53)
GP	1.062 (3.48)	1.038 (2.72)	1.038 (2.18)	1.009 (0.80)
CP	1.034 (3.28)	1.025 (1.84)	1.030 (2.55)	1.035 (3.18)
GP	1.063 (3.74)	1.023 (1.42)	1.026 (1.41)	1.006 (0.54)

Abbreviations: GP, gaseous pollutant; COH, coefficient of haze; TP, thoracic particles; FP, fine particles; CP, coarse particles. The ratio of log-relative risk to standard error is given in parentheses.
[a]Relative risk evaluated at the temperature evaluated at the interquartile range (see Table 2).

Table 6. Relative risk[a] for respiratory and cardiac hospitalizations for the particle-related measures simultaneously adjusted for O_3, NO_2, SO_2, temperature, and dew point temperature

	PRP					
	COH	H^+	SO_4	TP	FP	CP
Respiratory admissions						
PRP	1.023 (2.44)	1.006 (0.76)	1.000 (0.02)	1.004 (0.36)	0.999 (0.10)	1.007 (0.82)
O_3	1.056 (4.42)	1.057 (4.34)	1.059 (4.15)	1.059 (4.73)	1.059 (4.66)	1.059 (4.71)
NO_2	1.010 (0.71)	1.027 (2.39)	1.027 (2.36)	1.028 (1.77)	1.028 (2.26)	1.022 (1.71)
SO_2	1.014 (1.20)	1.016 (1.28)	1.019 (1.59)	1.018 (1.51)	1.019 (1.61)	1.018 (1.52)
Total[b]	1.106	1.109	1.109	1.110	1.109	1.109
Cardiac admissions						
PRP	1.059 (3.75)	1.005 (0.46)	0.984 (1.08)	0.996 (0.23)	0.993 (0.33)	1.022 (1.68)
O_3	1.052 (3.09)	1.062 (3.43)	1.076 (3.84)	1.064 (3.78)	1.067 (3.73)	1.064 (3.80)
NO_2	0.986 (0.64)	1.031 (1.70)	1.032 (1.75)	1.032 (1.61)	1.032 (1.71)	1.016 (0.63)
SO_2	1.015 (0.83)	1.028 (1.41)	1.034 (1.76)	1.031 (1.62)	1.032 (1.63)	1.026 (1.37)
Total[b]	1.116	1.131	1.130	1.127	1.128	1.134

Abbreviations: PRP, particle related pollutant; COH, coefficient of haze; TP, thoracic particles; FP, fine particles; CP, coarse particles. The ratio of absolute value of regression coefficient to standard error is given in parentheses.
[a]Relative risks evaluated at IQR (see Table 2).
[b]Relative risk of all pollutants combined, each evaluated at their IQRs.

The corresponding relative risks for cardiac diseases were 1.085, 1.089, and 1.064, respectively. Thus the association between O_3 and hospitalization was stable over the 3 years of observation.

The most stable particle measure after adjustment for the gaseous pollutants was COH, with relative risks ranging from 1.032 to 1.037 for respiratory diseases and 1.057 to 1.069 for cardiac outcomes.

O_3, NO_2, and SO_2 were simultaneously co-regressed with either admissions to hospital for respiratory or cardiac diseases and adjusted for temperature and dew point temperature. The relative risks (t-ratios) for respiratory diseases, evaluated at their respective interquartile ranges, were 1.059 (4.73) for O_3, 1.028 (2.45) for NO_2, and 1.019 (1.64) for SO_2. The corresponding relative risks for cardiac diseases were 1.064 (3.80), 1.30 (1.68), and 1.30 (1.60). The combined relative risk for all three pollutants was 1.109 for respiratory diseases and 1.130 for cardiac diseases.

Each of the particle-related measures were co-regressed with O_3, NO_2, and SO_2 (Table 6). The COH relative risk was attenuated slightly with further adjustment for O_3, NO_2, and SO_2 in addition to temperature and dew point temperature (RR = 1.023, t = 2.44 for respiratory diseases; RR = 1.059, t = 3.75 for cardiac diseases). However, the association with acid was clearly diminished (RR = 1.006, t = 0.76 for respiratory diseases; RR = 1.005, t = 0.46 for cardiac diseases). The association between hospitalizations for respiratory diseases and sulfate concentrations was eliminated after adjustment for these factors (RR = 1.000) as was that for TP (RR = 1.004), FP (RR = 0.999), and coarse particles between 10 and 2.5 μm in aerometric diameter (CP; RR = 1.007). Similar reductions in risk were observed for cardiac diseases with acid aerosols (RR = 1.005), sulfates (RR = 0.984), TP (RR = 0.996), and FP (RR = 0.993). Only CP displayed at least a marginal level of evidence of an association after adjustment for the gaseous pollutants (RR = 1.022, t = 1.68). The O_3 relative risk was not sensitive to adjustment for the other air pollutants (RR = 1.056, t = 4.42 for respiratory diseases; RR = 1.052, t = 3.09 for cardiac diseases).

Discussion

Statistically significant positive associations between daily fluctuations in hospital admissions for both respiratory and cardiac diseases were observed with several ambient air pollutants in the summers of 1992, 1993, and 1994 in Toronto, Canada. The admission time series was adjusted for long-term temporal trends, seasonal varia-

tion, and subseasonal fluctuations (possibly due to influenza epidemics), and day of the week effects. The time series were also adjusted for temperature and dew point temperature.

Several of the ambient air pollutants were positively correlated. As such, estimates of the relative risks for these variables were often sensitive to adjustment for co-pollutants. Ozone was insensitive to adjustment for the environmental factors, possibly due to its weak correlation with the other air pollutants. The coefficient of haze was the strongest predictor of hospitalizations for both respiratory and cardiac diseases among the particle-related pollutants examined in single and multiple pollutant regression models. The statistically significant positive association evident with thoracic, fine, and coarse particulate mass could be largely explained by the gaseous air pollutants.

There are several reasons why a particle mass effect on respiratory and cardiac hospitalizations may not have been observed in this study after simultaneous adjustment for the gaseous pollutants. First, the strongest association between the gaseous pollutants and health were for either daytime averages or daily 1-hr maximum readings. The coefficient of haze is monitored continuously and reported on an hourly basis, but the strongest association with the health outcomes was observed for the daytime average. The other particle-related measures were only monitored on a 24-hr basis, thus potentially more appropriate daily summary measures were not available. To examine the influence of this limitation on our conclusions, the entire analysis was repeated with the gaseous pollutant metrics and COH being limited to the 24-hr averages using the same days averaged and days lagged as that given in Table 2. The relative risks between the particulate-related measures and either health outcome after adjustment for daily averages of the gaseous pollutants were almost identical to the relative risks reported in Tables 4, 5, and 6 based on daytime measures of pollution exposure. Thus, the availability of only daily particle measures does not appear to be a problem.

The time series used for analyses were the average of hourly readings among several population-based monitoring sites for the gaseous air pollutants and COH. However, the other particle-related measures were obtained from a single monitoring site in a downtown Toronto location, co-located with a gaseous monitoring site. Thus, the average of the multiple sites may have been a more accurate measure of the average personal exposure to air pollution for the entire population than that measure recorded at a single site. However, fine mass and sulfate measurements collected at this site have been shown to be highly correlated (r>0.8) with concentrations over a wide area covering and extending beyond the region of interest in this study (*5*). Similar evidence, indicating that a single centrally located site measuring fine particulate matter and sulfates is a reasonable predictor of the average of personal exposures among populations living in urban environments, has been reported for other cities (*10*). Similar conclusions were also drawn for acid aerosols (*11,12*) although recent Toronto area measurements (*5*) show that spatial correlation for acid aerosols is somewhat lower (r ranges from 0.69 to 0.85) than for fine mass and sulfate measurements. As expected, the degree of spatial correlation is smallest for coarse particles (r ranges from 0.44 to 0.53).

The relative risks for the particle mass measures (TP, FP, CP) were most sensitive to simultaneous adjustment for NO_2 (Table 4 and 5). These bivariate regressions were repeated using NO_2 readings obtained from the site co-located with the mass measurements. The relative risks (t-ratios) for TP, FP, and CP were 1.008 (0.80), 1.014 (1.19), and 1.009 (1.21), respectively, for respiratory hospitalizations, values close to those obtained using multiple sites to estimate population exposure to NO_2. The corresponding results for hospital admissions for cardiac diseases were also similar using a single site for NO_2 (RR = 1.013, t = 0.78 for TP; RR = 1.020, t = 1.16 for FP; and RR = 1.023, t = 1.92 for CP) and those presented in Table 5 employing multiple sites. The use of a single monitoring site for the particle mass measurements does not appear to be a major limitation in this study.

Concentrations of acid, sulfate, and mass were very low during the study period, especially compared to the summers of 1986, 1987, 1988 in Toronto, previously examined by Thurston et al. (*3*). However, concentrations of the gases were also much lower in our study compared to those observed in the earlier investigation.

Ozone has been linked to excess visits to the emergency department for respiratory illnesses in Montreal, Quebec (*13*), Saint John, New Brunswick (*14*), and Mexico City (*15*). Hospital admissions for respiratory diseases have also been associated with O_3 in Detroit, Michigan (*16*), New York State (*4*), London, England (*17*), southern Ontario (*18*), Toronto (*3*), and in Canada's 16 largest cities (*19*). The association between O_3 and hospital admissions for cardiac diseases has not been extensively examined. However, O_3 concentrations have been related to nonaccidental mortality in Philadelphia, Pennsylvania (*20*), cardiovascular mortality in London, England (*21*), and in Los Angeles, California (*22*).

Another optical measure of particulate matter, black smoke, has been linked to emergency room visits for chronic obstructive pulmonary disease (COPD) (*23*) and asthma (*24*) in Barcelona, Spain, and for both respiratory and cardiac hospital admissions in Athens, Greece (*25*). Associations were also detected for nonaccidental mortality in Amsterdam (*26*) and with other optical particle measures in Los Angeles (*22*).

Nitrogen dioxide concentrations were associated with emergency department visits for asthma in Barcelona (*24*), cardiorespiratory hospitalizations in Athens (*25*), and admissions for COPD in Helsinki, Finland (*27*). Daily mortality rates were also linked to NO_2 levels in Los Angeles (*22*) and Philadelphia (*20*). In this latter study, NO_2 was found to be a stronger predictor of nonaccidental mortality than particulate mass (as measured by total suspended particulate matter), and simultaneous adjustment for O_3, SO_2, and NO_2 severely attenuated the mass effect, a result similar to that observed in the present investigation.

Although SO_2 has been found to be a predictor of hospitalization for respiratory (*19*) and cardiac (*28*) diseases in a number of Canadian cities using single regression models, the effect could be largely explained by adjustment for the other ambient air pollutants examined, as was the case in the present investigation. However, these studies also demonstrated a strong association with CO concentrations recorded year round, even after adjustment for O_3, NO_2, SO_2, and the COH. The relatively weak association for CO observed in the present investigation may be due to low summertime concentrations.

The purpose of our analyses was to examine the role particulate size and chemistry play in exacerbating cardiorespiratory disease. To that end we collected daily measurements of fine and coarse particles and determined the acid and sulfate content of the mass. These time series were then related to daily variations in the number of hospital admissions for respiratory and cardiac diseases. Even though we could observe statistically significant positive associations for these particle measures with health outcomes after controlling for climatic factors, the apparent association disappeared after adjustment for O_3, NO_2, and SO_2. The exception was the optical measure of particles COH.

It is recommended that all available air pollution measures be considered in assessing

the effects of any single pollutant on health. In our case, for example, TP remained a positive and statistically significant predictor for respiratory hospitalizations after adjusting for either O_3, SO_2, or CO separately, an analysis strategy used by many investigators. However, the TP association could be completely explained by NO_2, a risk factor not as widely considered in North American locales as the other criteria pollutants.

Based on the results from this study, we cannot attribute any adverse health effects to particle mass or chemistry alone. Our results suggest, however, that O_3 is associated with daily hospital admissions for both respiratory and cardiac diseases, after controlling for several measures of particulate mass and chemistry, climatic factors, and other gaseous pollutants. A positive and statistically significant association with the coefficient of haze persisted after simultaneous adjustment for the other gaseous pollutants and climatic factors. The coefficient of haze is strongly influenced by carbon particles, which are primarily from the combustion of diesel fuel; however, it may also be acting as a surrogate for transportation-related pollution or even for locally generated ambient air pollution. Ozone, on the other hand, may be acting, in part, as a surrogate for regional and transported pollution. The combination of the two pollutants may be a reasonable index to measure the pollution burden in an urban environment.

One of the main sources of fine particles in most urban environments is the condensation of gases, so it is not unexpected that the gases themselves should be able to explain variation in health responses attributable to particle mass alone. It is difficult to separate the individual effects of a single pollutant from the combined toxicity of the atmospheric mix using information from a single urban location. Mass may be acting, in part, as a surrogate for the mix, which is more stable over seasons and locations than any single gaseous pollutant. However, in our study, 3–4% increases in admission rates were attributed to mass in single predictor regression models, while 11–13% were attributed to the mix. Underestimates of the public health benefits of air pollution mitigation strategies, in which primary emissions of gaseous pollutants are reduced to limit secondary formation of particulate aerosols, could occur if all the benefits are attributed only to reductions in particulate mass.

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