

## Air Pollution and Daily Hospital Admissions for Cardiovascular Diseases in Windsor, Ontario

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### Abstract

**Objective**—To examine the role that ambient air pollution plays in exacerbating cardiovascular disease hospitalization in Windsor, Ontario.

**Methods**—The number of daily cardiac hospital admissions was obtained from all Windsor hospitals from April 1, 1995 to December 31, 2000 and linked to concentrations of ambient air pollutants and weather variables. The logarithm of daily counts of hospitalization was regressed on the levels of pollutants, after adjusting for seasonal, weekly cycles, and weather variables using time series analysis with natural splines as smoothing functions.

**Results**—Of all the pollutants considered, sulphur dioxide (SO<sub>2</sub>) had the strongest effect on cardiac hospitalization among the 65 age group. The percentage increase in daily admission was 2.6% for current day sulphur dioxide level (95% CI: 0.5–6.4), 4.0% for 2-day mean level (95% CI: 0.1–6.9), and 5.6% (95% CI: 1.5–9.9) for 3-day mean level for an increase in interquartile range of 19.3 ppb. When particulate PM<sub>10</sub> was included in the model, the contributing effect of sulphur dioxide remained significant for the 65 age group for all three levels.

**Conclusions**—Short-term effects of sulphur dioxide are associated significantly to daily cardiac hospital admissions for people 65 years of age living in Windsor. Since Windsor is a border city, additional monitoring and assessment is recommended to determine if air quality and resultant health effects have deteriorated since traffic congestion at the border has increased following the events of September 11, 2001.

### MeSH terms

air pollution; cardiovascular diseases; hospitalization

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Windsor is situated in Southwest Ontario across the river from Detroit, Michigan, with a population of 208,402 (2001 Census). It was one of the 17 geographical areas in Ontario identified by Health Canada as Areas of Concern<sup>1</sup> (AOC) and in need of extensive health

research. Windsor is one of the most industrialized cities in Canada and is located immediately downwind from steel mills, sludge incineration facilities, and a power plant (coal fired until recently) in Detroit. Based on Health Canada data from 1986 to 1992, Gilbertson and Brophy<sup>2</sup> reported elevated rates of mortality, morbidity and congenital abnormalities for Windsorites. Furthermore, Windsor-Detroit is a major transportation corridor, the most trafficked border crossing between Canada and the United States. After the September 11, 2001 (9/11) terrorist attack, delays and congestions at the border resulted in long lines of trucks on the city streets, spewing toxic pollutants into the air. This study is part of a larger research program to examine the role that ambient air pollution plays in exacerbating cardiovascular disease hospitalization in Windsor, Ontario before 9/11.

Many epidemiological studies in the last two decades reported acute associations between elevated air pollution levels and increased death and hospitalization rates due to cardiovascular diseases.<sup>3–14</sup> In Canada, several reports<sup>15–18</sup> have been published linking air pollution to adverse population health in cities based on data that were collected in the 1980s and early 1990s. For Windsor, Burnett et al.<sup>17</sup> found that the logarithm of the daily high-hour ambient carbon monoxide (CO) concentration on the day of admission showed the strongest and most consistent association among all pollutants, using 1981–1991 data. Schwartz and Morris<sup>4</sup> found that particles with an aerodiameter of 10 µm or less (PM<sub>10</sub>) were associated with daily admissions for ischemic heart disease for people 65 years or older in Detroit, 1986–1989. In a two-pollutant model,<sup>4</sup> both PM<sub>10</sub> and CO showed significant associations with heart failure admissions. In the past ten years, more stringent air quality guidelines were set by the government of Canada and significant decreases in levels have been achieved for SO<sub>2</sub>, CO, and nitrogen dioxide (NO<sub>2</sub>) in Windsor.<sup>20</sup> It is time now to re-examine the cardiac health risk of Windsorites (1995–2000) associated with ambient air pollutants. With increasing concerns that the achievements made in relation to ambient air quality in Windsor are eroding following the impact of post-9/11 vehicular pollution, this study also forms a reference point for future investigations into the health effects of post-9/11 traffic pollution changes on Windsorites.

## METHODS

The study population consisted of all Windsor hospital admissions with cardiovascular diseases: congestive heart failure (ICD-9 code 428), ischemic heart disease (codes 410–414), and dysrhythmias (code 427) during the period of April 1, 1995 to December 31, 2000. Daily hospital admission records for Ontario Health Insurance Plan (OHIP) patients were obtained from the Canadian Institute for Health Information's (CIHI) Discharge Abstract Database (DAD) for the four hospitals in Windsor, which serve not only the city but also its adjoining communities.

The hourly air quality index and air pollution data from four monitoring stations in Windsor were obtained from the Ontario Ministry of the Environment. Every variable considered here was measured on a daily basis, and for each day, the highest hourly reading was taken from all stations. These include gas data: sulphur dioxide (SO<sub>2</sub>), carbon monoxide (CO), nitrogen oxide (NO<sub>2</sub>), ozone (O<sub>3</sub>); COH (coefficient of haze, a measure of organic and inorganic carbon), and inhalable particles (PM<sub>10</sub>). Respirable particle PM<sub>2.5</sub> data were only available

from March 1999 to February 2000, so they were not used in the analyses. Daily weather data, including maximum and minimum temperature, humidity and barometric pressure, were obtained from the Ontario Climate Centre in Toronto. For our analysis, we created a new variable called “change in maximum (minimum) barometric pressure from the previous day.” This variable was found to be an important predictor of daily mortality in earlier Canadian studies.<sup>15</sup>

First, we linked together over 2,000 days of records from several databases, consisting of measurements of pollutants, temperature, humidity, change in barometric pressure from the previous day, and number of cardiac admissions. Data were carefully screened for missing values and outliers, and were edited before any analyses were performed.

To relate short-term effects of air pollution to the number of cardiovascular hospital admissions for each subgroup of data, we used the time series modeling technique which has long been used to obtain relative risks of health events with ambient air pollution levels. We first removed the smoothed seasonal cycles, secular trends, and day-of-the-week to produce a time series of logarithm of hospital admissions that is as close to white noise as possible, as determined by Bartlett’s test.<sup>21</sup> Natural splines were used here to smooth the effects of all continuous covariates because locally weighted regression smoothers within the generalized additive models framework<sup>22</sup> were found to produce bias estimates.<sup>23,24</sup> We then extended the model by incorporating combinations of smoothed weather variables (maximum or minimum of temperature, humidity and change in barometric pressure) that yielded the lowest AIC (Akaike Information Criterion). Last, we added the air pollution variable(s) into the model with no pre-filtering applied. The analysis was conducted using the computer software S-Plus<sup>25</sup> (GLM).

Relative risk of cardiovascular hospitalization attributable to each single pollutant using current day exposure level, average of today and yesterday’s level (2-day mean), and average of current day and previous two days’ level (3-day mean) was estimated for an increase in value of current-day interquartile range. Results are expressed as percentage changes in daily admissions associated with the pollutant. Multi-pollutant models were also fit. In this case, the contributing effect of one pollutant could be assessed in the presence of other pollutants.

## RESULTS

A total of 11,632 cardiac hospitalizations occurred in the study period, with 3,273 in the <65 age group and 8,359 in the ≥65 group. Table I gives the summary statistics of daily average concentrations of all the pollutants and weather variables, as well as daily admissions for patients aged <65 and ≥65. Based on the air quality index (AQI), there were 165 days of poor air quality, 583 days of moderate air quality and 1,352 days of good air quality during the entire study period.

Table II gives the correlation coefficients for the air pollutants and weather variables. Most of the pollutants are positively correlated with each other except for SO<sub>2</sub> and O<sub>3</sub> ( $r = -0.02$ ). Maximum temperature and minimum humidity were highly correlated with O<sub>3</sub>. These

correlations may influence the ability of the analysis to determine the individual effects of each pollutant.

Table III presents the percentage changes in daily hospital admissions and 95% confidence intervals across the interquartile range of exposures to selected pollutants for the two age subgroups (<65, ≥65) when we adjusted for temperature, humidity and change in barometric pressure from the previous day. We found significant associations between cardiac hospitalization and SO<sub>2</sub> (increased hospital admissions of 2.6%, 4.0%, and 5.6% for current day, 2-day and 3-day mean) in the ≥65 age group, with a stronger delayed effect. No other pollutants showed significant associations with cardiac admissions, although most of them showed positive associations. Similar results for SO<sub>2</sub> were obtained when we did not control for change in barometric pressure in the model (2.7%, 4.1%, 5.7%, corresponding to 1, 2, 3-day means).

Next, we assessed the association of SO<sub>2</sub> with cardiovascular admissions in the presence of PM<sub>10</sub>, with temperature, humidity and change in barometric pressure in the model. We found that SO<sub>2</sub> remained significant for current day, 2-day and 3-day mean for the elderly group (increase in admissions of 2.8%, 4.3% and 5.7% respectively). We fitted different multi-pollutant models, but found only SO<sub>2</sub> to be consistently associated with cardiac admissions for the elderly. None of the pollutants showed significant association with admissions for the <65 age group.

## DISCUSSION

In this study, short-term effect of SO<sub>2</sub> on daily cardiac hospital admissions in Windsor was found to be significant for people ≥65 years old, with or without adjusting for PM<sub>10</sub>. Using a single pollutant model, PM<sub>10</sub> was found to be positively associated with admissions, although the effect was not significant in both age groups. These results are in general agreement with the existing literature. Sunyer et al.<sup>12</sup> did a systematic review of the literature examining the relationship between cardiac hospital admissions and SO<sub>2</sub> and particles. Out of 34 reports including particulates, they found 33 with a positive significant association between particles and cardiovascular admissions. For studies including SO<sub>2</sub>, 17 out of 24 found positive and significant associations. Among the 13 reports that analyzed both SO<sub>2</sub> and particles, 9 of them had significant SO<sub>2</sub> effects. In a recent study in Hong Kong,<sup>26</sup> a decrease of SO<sub>2</sub> levels had a notable impact in reducing adverse health effects, although particle levels remained stable. While the epidemiological findings are very consistent, the underlying mechanisms of an acute heart effect of air pollution are still unknown. Several physiopathological pathways have been proposed for the relationship between particles and cardiovascular health.<sup>27–29</sup> SO<sub>2</sub> may act via a different mechanism. A change in heart rate variability on exposure to SO<sub>2</sub> (200 ppb for 1h) was attributed to stimulation of receptors in the upper respiratory tract.<sup>30</sup> Perhaps this is the way SO<sub>2</sub> acts on heart patients. More investigations are needed in this area.

Our results differ from the Detroit study in which Schwartz and Morris<sup>4</sup> found that CO and airborne particles were significant predictors of cardiovascular admissions. Since Detroit and Windsor are neighbours geographically, they share the same environmental concerns.

Indeed, in Windsor, PM<sub>10</sub> had positive (but not significant) association with cardiac admissions for both age groups. However, CO was negatively associated with admissions for people <65, although it was positively associated for people ≥65. In the 10 Canadian cities study, which used data that spanned from 1981 to 1991, Burnett et al.<sup>17</sup> found that the Windsor city-specific relative risk of CO for congestive heart failure hospital admissions in the elderly was 1.01 for both single- and multi-pollutant models. This is certainly within our confidence limits. It is also important to note that significant reduction in CO had been achieved in the subsequent 10 years in Windsor (mean=1.0 ppm in 1991 to 0.3 in 2000) due to more stringent regulatory effort in air quality (The Air Quality in Ontario, 2000 Report<sup>20</sup>). That may explain why CO is no longer a significant contributor to cardiac admissions in recent years. If this is true, these results surely demonstrate that improvement of public health can be achieved when levels of air pollution are reduced.

Limitations of this study are typical of this type of research. They include the adequacy of covariate control and the impact of measurement error in the exposure and outcome variables. Most of the risk factors, such as the presence of chronic conditions and cigarette smoking, do not vary on a day-to-day basis; hence, they are not likely confounders. Fixed-site monitors provide daily pollution levels and are used to represent personal exposures of ambient air pollution. There will be some degree of measurement errors here. However, Windsor is not a large city geographically and we believe that with the four monitors, the measurement errors will not be greater than would be found in other similar-sized cities.

Although the results here suggest a weak association between cardiac hospitalization and most ambient air pollutants except SO<sub>2</sub>, the September 11, 2001 event has brought renewed concerns about the effects of air pollution in Windsor. There have been increasing delays and congestion at the border-crossing points, resulting in long lines of trucks spewing toxic pollutants from their archaic exhaust systems into the air. The combined effect of vehicular and industrial pollutants could make the improvements of the past few years rapidly disappear. As such, the plausible next step is to conduct a post 9/11 follow-up study that examines the impact of ambient air pollution in Windsor. More frequent air quality monitoring is needed to detect any changes that may be taking place.

## Acknowledgments

This research was supported in part by an NSERC operating grant to K. Fung, by a Canadian Institutes of Health Research investigator award to K. Gorey and an associated partnership appointment to I. Luginaah.

## References

1. Health Canada. Health Data and Statistics Compilations for Great Lakes Areas of Concern. Health Canada; 2000.
2. Gilbertson M, Brophy J. Community health profile of Windsor, Ontario, Canada. *Environ Health Perspect.* 2001; 109(Suppl 6):827–43.
3. Schwartz J. Air pollution and hospital admissions for cardiovascular disease in Tucson. *Epidemiology.* 1997; 8:371–77. [PubMed: 9209849]
4. Schwartz J, Morris R. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. *Am J Epidemiol.* 1995; 142:23–35. [PubMed: 7785670]

5. Schwartz J. Air pollution and hospital admissions for heart disease in eight U.S. counties. *Epidemiology*. 1999; 10:17–22. [PubMed: 9888275]
6. Kwon HJ, Cho SH, Nyberg P, Pershagen G. Effects of ambient air pollution on daily mortality in a cohort of patients with congestive heart failure. *Epidemiology*. 2001; 12:413–19. [PubMed: 11416779]
7. Morris RD, Naumova EN. Carbon monoxide and hospital admissions for congestive heart failure: Evidence of an increased effect at low temperatures. *Environ Health Perspect*. 1998; 106:649–53. [PubMed: 9755140]
8. Morris RD, Naumova EN, Munasinghe RL. Ambient air pollution and hospitalization for congestive heart failure among elderly people in seven large US cities. *Am J Public Health*. 1995; 85:1361–85. [PubMed: 7573618]
9. Yang W, Jennison BL, Omaye ST. Cardiovascular disease hospitalization and ambient levels of carbon monoxide. *J Toxicol Environ Health A*. 1998; 55:185–96. [PubMed: 9772102]
10. Poloniecki JD, Atkinson RW, Ponce de Leon A, Anderson HR. Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occup Environ Med*. 1997; 54:535–40. [PubMed: 9326156]
11. Seaton A, MacNee W, Donaldson K, Godden D. Particulate air pollution and acute health effects. *Lancet*. 1995; 345:176–78. [PubMed: 7741860]
12. Sunyer J, Ballester F, Le Tertre A, Arkinson R, Ayres JG, Forastiere F, et al. The association of daily sulfur dioxide air pollution levels with hospital admissions for cardiovascular diseases in Europe (The Aphea-II study). *Eur Heart J*. 2003; 24:752–60. [PubMed: 12713769]
13. Moolgavkar SH. Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. *J Air Waste Manag Assoc*. 2000; 50:1199–206. [PubMed: 10939212]
14. Ponka A, Virtanen M. Low-level air pollution and hospital admissions for cardiac and cerebrovascular diseases in Helsinki. *Am J Public Health*. 1996; 86:1273–80. [PubMed: 8806380]
15. Goldberg M, Burnett R, Valois M, Flegel K, Bailar JC, Brook J, et al. Associations between ambient air pollution and daily mortality among persons with congestive heart failure. *Environmental Research*. 2003; 91:8–20. [PubMed: 12550083]
16. Burnett RT, Dales RE, Krewski D, Vincent R, Dann T, Brook J. Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory diseases. *Am J Epidemiol*. 1995; 142:15–22. [PubMed: 7785669]
17. Burnett RT, Dales RE, Brook JR, Raizenne ME, Krewski D. Association between ambient carbon monoxide levels and hospitalization for congestive heart failure in the elderly in 10 Canadian cities. *Epidemiology*. 1997; 8:162–67. [PubMed: 9229208]
18. Burnett RT, Smith-Doiron M, Steib D, Cakmak S, Brook JR. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. *Arch Environ Health*. 1999; 54:130–39. [PubMed: 10094292]
19. Schwartz J, Morris R. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. *Am J Epidemiol*. 1995; 142:23–35. [PubMed: 7785670]
20. The Air Quality in Ontario 2000 Report. Ontario Ministry of the Environment;
21. Priestly, MB. *Spectral Analysis of Time Series*. San Diego, CA: Academic Press; 1981.
22. Hastie, T., Tibshirani, R. *Generalized Additive Models*. London, UK: Chapman & Hall; 1990.
23. Dominici F, McDermott A, Zeger SL, Samet JM. Airborne particulate matter and mortality: Time-scale effects in four US cities (with invited commentary). *Am J Epidemiol*. 2003; 157:1055–65. [PubMed: 12796040]
24. Ramsay T, Burnett RT, Krewski D. The effect of concurvity in generalized additive models linking mortality to ambient particulate matter. *Epidemiology*. 2003; 14:18–23. [PubMed: 12500041]
25. S-Plus 6. Insightful Corporation; Seattle, Washington;
26. Anderson, HR., Wong, CM., Hedley, AJ. Health effects of air pollution in Hong Kong. Proceedings of the thirteen conference ISEE 2001; Garmish. 2001. p. 580
27. Donaldson K, Stone V, Seaton A, MacNee W. Ambient particle inhalation and the cardiovascular system: Potential mechanisms. *Environ Health Perspect*. 2001; 109(Suppl 4):523–29.

28. Peters A, Dockery DW, Muller JE, Mittleman M. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*. 2001; 103:2810–24. [PubMed: 11401937]
29. Pope CA, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. Cardiovascular mortality and long-term exposure to particulate air pollution—epidemiological evidence of general pathophysiological pathways of disease. *Circulation*. 2004; 109:71–77. [PubMed: 14676145]
30. Tunncliffe WS, Hilton MF, Harrison RM, Ayres JG. The effect of sulphur dioxide exposure on indices of heart rate variability in normal and asthmatic adults. *Eur Respir J*. 2001; 17:604–8. [PubMed: 11401052]



**TABLE I**

Mean, Standard Deviation, and Percentiles of the Daily High Concentrations of Air Pollutants and Daily Numbers of Cardiac Admissions, Windsor, Ontario (April 1995 to December 31, 2000)

Variable (unit)	Mean	Standard Deviation	Minimum	Maximum
Air pollutant				
NO <sub>2</sub> (ppb)	38.9	12.3	0	117
O <sub>3</sub> (ppb)	39.3	21.4	1	129
CO (ppm)	1.3	1.0	0	11.82
SO <sub>2</sub> (ppb)	27.5	16.5	0	129
PM <sub>10</sub> (µg/m <sup>3</sup> )	50.6	35.5	9	349
AQI	28.3	12.8	7	85
COH	0.6	0.4	0	3.6
Weather				
Max Temperature (°C)	14.2	11.2	-15.8	35.7
Min Temperature (°C)	5.3	9.8	-21.4	25.6
Max Humidity	86.1	9.2	50.0	100.0
Min Humidity	53.4	15.0	17.0	98.0
Maxp *	-0.00	0.54	-2.36	2.06
Minp *	-0.00	0.07	-3.42	3.12
Daily cardiac admissions				
<65	1.7	1.3	0	8
65+	4.4	2.2	0	14

\*Maxp - change in maximum barometric pressure from the previous day

Minp - change in minimum barometric pressure from the previous day



TABLE II

Correlation Coefficients Between Air Pollutants and Weather Variables

	PM <sub>10</sub>	NO <sub>2</sub>	SO <sub>2</sub>	CO	O <sub>3</sub>	COH	Mint	Minh	Maxt	Maxh	Maxp	Minp
PM <sub>10</sub>	1.00											
NO <sub>2</sub>	0.33	1.00										
SO <sub>2</sub>	0.22	0.22	1.00									
CO	0.21	0.38	0.16	1.00								
O <sub>3</sub>	0.33	0.26	-0.02	0.10	1.00							
COH	0.39	0.49	0.14	0.31	0.23	1.00						
Mint*	-0.26	-0.22	-0.12	-0.06	-0.45	-0.16	1.00					
Minh*	0.25	0.06	-0.06	0.02	0.67	0.21	-0.19	1.00				
Maxt*	0.34	0.15	-0.01	0.08	0.74	0.28	0.95	-0.34	1.00			
Maxh*	-0.09	-0.09	-0.08	0.03	-0.20	0.03	-0.02	0.63	-0.07	1.00		
Maxp*	-0.14	-0.06	-0.03	-0.08	-0.04	-0.05	-0.13	-0.18	-0.14	-0.23	1.00	
Minp*	-0.13	-0.03	-0.01	-0.04	-0.04	-0.05	-0.13	-0.18	-0.15	-0.27	0.67	1.00

\* Mint - minimum temperature

Maxt - maximum temperature

Minh - minimum humidity

Maxh - maximum humidity

Minp - change in minimum barometric pressure from the previous day

Maxp - change in maximum barometric pressure from the previous day

Percentage Change in Relative Risk Estimates (RR) and 95% Confidence Interval (CI) by Time Series Analysis for Single Pollutants in Relation to Cardiac Hospital Admissions in Windsor

TABLE III

Pollutant	IQR	<65 Age Group (n=3273)		65 Age Group (n=8359)	
		Mean % Change	95% CI	Mean % Change	95% CI
SO <sub>2</sub> current day	19.3	2.3	(-1.8, 6.6)	2.6*	(0.0, 5.3)
2-day mean		3.9	(-1.5, 9.6)	4.0*	(0.6, 7.6)
3-day mean		3.4	(-3.0, 10.1)	5.6*	(1.5, 9.9)
NO <sub>2</sub> current day	16	-0.7	(-5.5, 6.6)	0.8	(-2.2, 3.9)
2-day mean		2.1	(-3.7, 8.2)	0.9	(-2.7, 4.6)
3-day mean		3.7	(-2.9, 10.7)	0.8	(-3.3, 5.0)
CO current day	1.2	-3.1	(-7.4, 1.4)	0.5	(-2.2, 3.3)
2-day mean		-2.7	(-8.1, 3.0)	2.3	(-1.1, 5.9)
3-day mean		-0.5	(-6.7, 6.0)	2.8	(-1.1, 7.0)
O <sub>3</sub> current day	29	-0.1	(-8.7, 9.3)	-2.6	(-7.6, 2.7)
2-day mean		6.7	(-4.3, 18.9)	-0.1	(-6.4, 6.6)
3-day mean		4.2	(-7.7, 17.7)	1.4	(-5.9, 9.2)
TRS current day	8	-1.4	(-4.0, 1.4)	0.2	(-1.4, 1.9)
2-day mean		-2.0	(-5.5, 1.7)	1.0	(-1.2, 3.3)
3-day mean		-1.2	(-5.3, 3.1)	1.0	(-1.6, 3.7)
PM <sub>10</sub> current day	31	0.1	(-1.8, 6.6)	0.1	(-3.3, 3.5)
2-day mean		1.4	(-2.7, 5.7)	0.1	(-2.5, 2.7)
3-day mean		1.5	(-3.2, 6.4)	1.0	(-1.9, 4.1)
COH current day	0.5	-1.2	(-5.5, 3.2)	0.4	(-2.3, 3.2)
2-day mean		1.6	(-3.8, 7.3)	0.4	(-3.0, 4.0)
3-day mean		1.3	(-5.1, 8.1)	2.1	(-2.0, 6.3)

\* p<0.05 level of significance