

## Asthma and Low Level Air Pollution in Helsinki

ANTTI PÖNKÄ, M.D.

Department of Environmental Health  
Helsinki City Health Department  
Helsinki, Finland

**ABSTRACT.** The effects of relatively low levels of air pollution and weather conditions on the number of patients who had asthma attacks and who were admitted to a hospital were studied in Helsinki during a 3-y period. The number of admissions increased during cold weather ( $n = 4\,209$ ), especially among persons who were of working age but not among children. Even after standardization for temperature, all admissions, including emergency ward admissions, were significantly correlated with ambient air concentrations of nitrogen dioxide ( $\text{NO}_2$ ), nitric oxide ( $\text{NO}$ ), sulfur dioxide ( $\text{SO}_2$ ), carbon monoxide ( $\text{CO}$ ), ozone ( $\text{O}_3$ ), and total suspended particulates (TSP). Regression analysis revealed that  $\text{NO}$  and  $\text{O}_3$  were most strongly associated with asthma problems. Effects of air pollutants and cold were maximal if they occurred on the same day, except for  $\text{O}_3$ , which had a more pronounced effect after a 1-d lag. The associations between pollutants, low temperature, and admissions were most significant among adults of working age, followed by the elderly. Among children, only  $\text{O}_3$  and  $\text{NO}$  were significantly correlated with admissions. Levels of pollutants were fairly low, the long-term mean being  $19.2\ \mu\text{g}/\text{m}^3$  for  $\text{SO}_2$ ,  $38.6\ \mu\text{g}/\text{m}^3$  for  $\text{NO}_2$ ,  $22.0\ \mu\text{g}/\text{m}^3$  for  $\text{O}_3$ , and  $1.3\ \text{mg}/\text{m}^3$  for  $\text{CO}$ . In contrast, the mean concentration of TSP was high ( $76.3\ \mu\text{g}/\text{m}^3$ ), and the mean temperature was low ( $+4.7\ ^\circ\text{C}$ ). These results suggest that concentrations of pollutants lower than those given as guidelines in many countries may increase the incidence of asthma attacks.

EXPERIMENTAL AND EPIDEMIOLOGICAL STUDIES have revealed that ambient air pollution and cold weather increase the frequency of acute asthmatic episodes. The results of experimental studies have been more consistent than have those of epidemiological studies.

Asthmatics are adversely affected by many air pollutants and by cold weather. Bronchoconstriction occurs in certain asthmatic subjects upon exposure to  $\text{SO}_2$  levels as low as  $286\ \mu\text{g}/\text{m}^3$  (0.1 ppm) for 10 min during exercise.<sup>1</sup> In most studies, it has been shown that changes in pulmonary function occur when concentrations of  $\text{SO}_2$  are approximately  $1\,430\ \mu\text{g}/\text{m}^3$  (0.5 ppm).<sup>2</sup> Cold weather, either alone or in combination with  $\text{SO}_2$  or  $\text{NO}_2$ , causes and potentiates bronchospasm and airway hyperreactivity among asthmatics, especially dur-

ing exercise.<sup>3-5</sup> Less ambiguous results have been reported in studies of  $\text{NO}_2$  exposure. Orehek et al.<sup>6</sup> reported potentiation of the carbachol bronchoconstrictor response after exposure to  $188\ \mu\text{g}/\text{m}^3$  (0.1 ppm)  $\text{NO}_2$  in 13 of 20 asthmatics. Bronchoconstriction and airway hyperreactivity to cold air have been observed after exposure to  $560\ \mu\text{g}/\text{m}^3$  (0.3 ppm)  $\text{NO}_2$ .<sup>2,5</sup> Exposure to ozone at concentrations as low as  $234\ \mu\text{g}/\text{m}^3$  (0.12 ppm) increased airway reactivity in healthy subjects.<sup>7</sup>

The frequency and intensity of asthmatic episodes are related to season, e.g., autumn and frequency increases during the winter in the northern hemisphere. The reasons for a seasonal relationship are not clear.<sup>8-11</sup> Perhaps the reasons are multifactorial and vary with climate, vegetation, ambient air pollution, and other factors. Seasonal occurrence of asthma may also result

from exposure to infections, indoor air irritants, acute changes in weather, and antigens (e.g., pollen, house mites, organic compounds).<sup>12-17</sup>

Ambient air pollution generally contributes to an increased frequency of asthma attacks,<sup>18-20</sup> and separate correlations have been observed with increased concentrations of  $\text{SO}_2$ , nitrogen oxides ( $\text{NO}_x$ ), suspended particulates,  $\text{O}_3$ , and other photochemical oxidants.<sup>10,21-25</sup>

The concentrations at which ambient air pollutants increase the frequency of asthma attacks at levels observed with epidemiological methods are not unequivocally known. Nor is there adequate information on the synergistic effects with various pollutants and other factors, e.g., cold, relative importance of the different causative factors, and differences in sensitivity of different age groups. The result of many studies have been impaired by small sample sizes, confounding socioeconomic factors, insufficiently documented pollutant levels, and biases in collecting information about illnesses.

In Helsinki, where there is a population of 0.5 million, we investigated the incidence of asthma attacks that required treatment in hospital wards. This incidence was correlated with ambient air  $\text{SO}_2$ ,  $\text{NO}$ ,  $\text{NO}_2$ ,  $\text{CO}$ ,  $\text{O}_3$ , TSP concentrations, temperature, relative humidity, and wind speed.

### Materials and methods

**Air pollutants and meteorologic variables.** Air pollution measurements are conducted in Helsinki by district municipal authorities. Sulfur dioxide is measured by coulometric instruments at four automatic monitoring stations,  $\text{NO}_x$  by chemiluminescence at two stations,  $\text{CO}$  by nondispersive infrared spectrometry at two stations, and  $\text{O}_3$  by ultraviolet absorption at one station. Total suspended particulates are collected by high-volume samplers at six stations. At one weather station, measurements of temperature, wind speed, and relative humidity are recorded hourly.

Helsinki City includes a relatively small area, i.e.,  $185\ \text{km}^2$ . The main sources of air pollutants are energy production by coal-fired and oil-fired power plants, road traffic, and, to a small extent, industrialization. In 1987, the total emission of  $\text{NO}_2$  was 17 600 tons, of which 32% was derived from traffic, 67% from energy production, and 1% from industry. Ninety-seven percent of  $\text{CO}$  is emitted from cars. The monitoring stations that measure  $\text{NO}_x$  and  $\text{CO}$  are located on the main streets, where the bulk of  $\text{NO}_x$  is produced by traffic. Emissions of  $\text{SO}_2$  total 22 500 tons, of which 93% is from energy production, 2% from traffic, and 5% from industries. The stacks at the power plants are 100- to 150-m high, whereas the exhaust gases from cars spread at street level. At street level, 60-80% of  $\text{NO}_x$  is derived from traffic. Therefore,  $\text{NO}_x$  and  $\text{CO}$  are indicators of pollution resulting from traffic, and  $\text{SO}_2$  is an indicator of pollution generated by energy production.

**Incidence of admissions for asthma.** Data concerning hospital admissions for asthma attacks were obtained from the register that documented all periods of illness that required hospitalization. The register con-

tained information on the dates and main causes of hospitalization. The data covered all the municipal hospitals and Helsinki University Central Hospital, where more than 95% of patients with asthma who required hospitalization were treated. This study included only patients that were hospitalized primarily for acute asthma. Patients who were admitted to emergency wards and who needed more effective treatment were also treated subsequently in bed wards, but they were studied separately.

Diagnosis of asthma was based on the World Health Organization's Ninth International Classification of Diseases (ICD-9). Only patients with bronchial asthma, diagnosis number 493 of the classification, were included; therefore, those with chronic bronchitis were excluded. In Finland, diagnosis of asthma is always based on the consultation and statement of a specialist, which are needed for the provision of medication. The statement must include a thorough history of the disease, findings of a clinical examination, and results of blood tests and pulmonary function tests. An increase in the peak expiratory flow of 15% or more by bronchodilating drugs, a decrease of at least 15% after exercise, or spontaneous changes in the peak expiratory flow of at least 20% within 6 h is needed as a criterion of asthma diagnosis among adults and children who are old enough to cooperate in peak expiratory flow measurements. For each patient who visits a hospital ward or an outpatient department, documents are completed according to a certain format. These are always checked, as is the diagnosis, by a senior specialist.

**Statistical methods.** The number of patients admitted each day was calculated and correlated with the corresponding mean daily concentrations of  $\text{SO}_2$ ,  $\text{NO}_2$ ,  $\text{NO}$ ,  $\text{CO}$ , TSP,  $\text{O}_3$ , temperature, wind speed, humidity, and minimum hourly temperature during the day.

Cold affects the incidence of asthma; therefore, partial correlations were also calculated after standardization for minimum temperature. The minimum temperature was standardized because it was more strongly correlated with the frequency of asthma attacks than was daily mean temperature. Correlations were also calculated for 1- and 2-d lags. The log-transformed values of the variables were also used in the analysis.

Correlations were calculated separately for persons who were 0-14, 15-64, and 65+ y of age. The relative importance of the various factors<sup>26,27</sup> was estimated by stepwise regression.

The population in Helsinki numbered 488 604, 491 148, and 491 777 in 1987, 1988, and 1989, respectively.

### Results

**Number of admissions.** During the 3-y period, 4 209 hospitalizations for asthma occurred, i.e., average of 3.84 admissions/d. More than half (2 414) were admitted by the emergency ward. In 1987, the number of admissions was 1 534; in 1988, 1 484; and in 1989, 1 221. There were 1 359 cases who were in the 0-14 y age group, 1 685 cases were 15-64 y of age, and 1 165 were at least 65 y of age.

Variable	Mean	Range	SD
SO <sub>2</sub> (μg/m <sup>3</sup> )	19.2	0.2–94.6	12.6
NO <sub>2</sub> (μg/m <sup>3</sup> )	38.6	4.0–169.6	16.3
CO (mg/m <sup>3</sup> )	1.3	0–7.0	0.8
O <sub>3</sub> (μg/m <sup>3</sup> )	22.0	0–89.9	13.1
TSP (μg/m <sup>3</sup> )	76.3	6.0–414.0	51.6
Mean temperature (°C)	4.7	–37.0–+26.4	9.3
Minimum daily temperature (°C)	2.4	–39–+24.0	9.3
Relative humidity (%)	82.9	37.4–100.0	12.0
Wind speed (m/s)	4.7	0.6–10.9	1.8

	Minimum temperature	SO <sub>2</sub>	NO <sub>2</sub>	NO	O <sub>3</sub>	TSP
Mean temperature	.9867 ( $<.0001$ )	–.5507 ( $<.0001$ )	–.1407 ( $<.0001$ )	–.3043 ( $<.0001$ )	.1559 ( $<.0001$ )	–.0256 (.5089)
Minimum temperature	1	–.5712 ( $<.0001$ )	–.1701 ( $<.0001$ )	–.3162 ( $<.0001$ )	.1504 ( $<.0001$ )	–.0363 (.3490)
SO <sub>2</sub>		1	.4516 ( $<.0001$ )	.4773 ( $<.0001$ )	–.1778 ( $<.0001$ )	.1919 ( $<.0001$ )
NO <sub>2</sub>			1	.6664 ( $<.0001$ )	–.2582 ( $<.0001$ )	.1962 ( $<.0001$ )
NO				1	–.5479 ( $<.0001$ )	.1097 (.0034)
O <sub>3</sub>					1	.1836 ( $<.0001$ )

Note: *p* values appear in parentheses.

**Pollutants and meteorological variables.** Concentrations of most pollutants in the ambient air were relatively low. The mean concentrations (presented as the mean of the means at various stations) of SO<sub>2</sub>, NO<sub>2</sub>, CO, and O<sub>3</sub> during the 3-y period were 19.2 μg/m<sup>3</sup>, 38.6 μg/m<sup>3</sup>, 1.3 mg/m<sup>3</sup>, and 22.0 μg/m<sup>3</sup>, respectively. The mean concentration of TSP was 76.3 μg/m<sup>3</sup>, which was a high value that resulted from the (a) meteorological conditions; (b) the use of studded tires during winter, which caused erosion of street surfaces; and (c) the use of sand on streets to treat icy surfaces. Mean values and ranges for the various variables are presented in Table 1. When we compared the different stations, the mean long-term concentrations of SO<sub>2</sub> had a 1.4 to 2.3-fold variation; those of TSP, a 2.6 to 2.8-fold variation; and those of NO<sub>2</sub>, a 1.3 to 1.6-fold variation.

Daily values of the different variables and the hourly minimum temperature were correlated (Table 2). Highly significant negative correlations were obtained when SO<sub>2</sub>, NO<sub>2</sub>, and NO were compared with temperature, whereas O<sub>3</sub> correlated positively with temperature. There was a highly significant correlation between SO<sub>2</sub> and NO, NO<sub>2</sub>, and TSP, but SO<sub>2</sub> was correlated inversely with O<sub>3</sub>.

**Relation of air pollutants and weather to admissions.** The frequency of all admissions for asthma was highly significantly correlated with daily concentrations of SO<sub>2</sub>, NO, NO<sub>2</sub>, and CO and with the minimum hourly temperature. Frequency of admissions was also significantly correlated with TSP, but it was not correlated with concentration of O<sub>3</sub> or the mean daily temperature. The number of asthma cases seen in the emergency wards correlated at least on the *p* = .05 level with all pollution variables and with cold weather (Table 3).

The weekly mean concentrations of SO<sub>2</sub>, NO<sub>2</sub>, and TSP, and the number of asthma cases seen each week for 3 y are presented in Figures 1–3.

A better estimate of the significance of the air pollutants was desired; therefore, partial correlations of these pollutants and admissions for asthma were also calculated after standardization for temperature. Even after standardization, the number of cases seen at the emergency wards was positively correlated with all pollutants, except CO, as was the number of all cases with all pollutants, except O<sub>3</sub> (Table 4).

Except for O<sub>3</sub>, the significance of the correlations disappeared after a 1- or 2-d lag. The effect of O<sub>3</sub> on numbers of cases was greatest if there was a 1-d lag and

	All admissions	Admissions by emergency wards
SO <sub>2</sub>	.0926 (.0022)	.1319 ( $<.0001$ )
NO	.2128 ( $<.0001$ )	.1213 ( $<.0001$ )
NO <sub>2</sub>	.1774 ( $<.0001$ )	.1213 ( $<.0001$ )
CO	.1578 ( $<.0001$ )	.0650 (.0335)
O <sub>3</sub>	.0074 (.8119)	.0739 (.0170)
TSP	.0919 (.0136)	.1075 (.0039)
Mean temperature	–.0541 (.0841)	–.0915 (.0034)
Minimum temperature	–.0592 (.0585)	–.1006 (.0013)

Note: *p* values appear in parentheses.

was only slightly less if there was a 2-d lag (Table 5). The effect of cold weather was also observed after these lags.

Relative humidity had no effect on numbers of cases. However, wind speed was correlated with admissions for asthma on the same day (*p* = .036) and after a 1-d lag.

Concentrations of all other pollutants, except O<sub>3</sub>, were higher on Monday through Friday than on Saturday and Sunday. Therefore, correlations were calculated separately for weekdays and for weekends. During weekends, the correlations were not significant; during weekdays, however, numbers of cases were associated with NO, NO<sub>2</sub>, and O<sub>3</sub>. The mean daily number of cases during weekends was 2.44 and was 4.40 for Monday through Friday.

**Correlations with age.** The relationship between admissions for asthma and pollutants and cold weather was as follows: 15–64-y age group > 65+y age group > 0–14-y age group.

Among children, only O<sub>3</sub> and NO correlated with admissions by emergency wards. Ozone, NO, and CO were also correlated with all admissions at the *p* < .05 level. Among those who were 15–64 y of age, the situation was reversed: all pollutants, except O<sub>3</sub>, and low temperature contributed to an increase in number of cases. The effect of gaseous pollutants was greater than was the effect of TSP. Among the elderly, SO<sub>2</sub>, NO, NO<sub>2</sub>, and cold weather correlated significantly with ad-

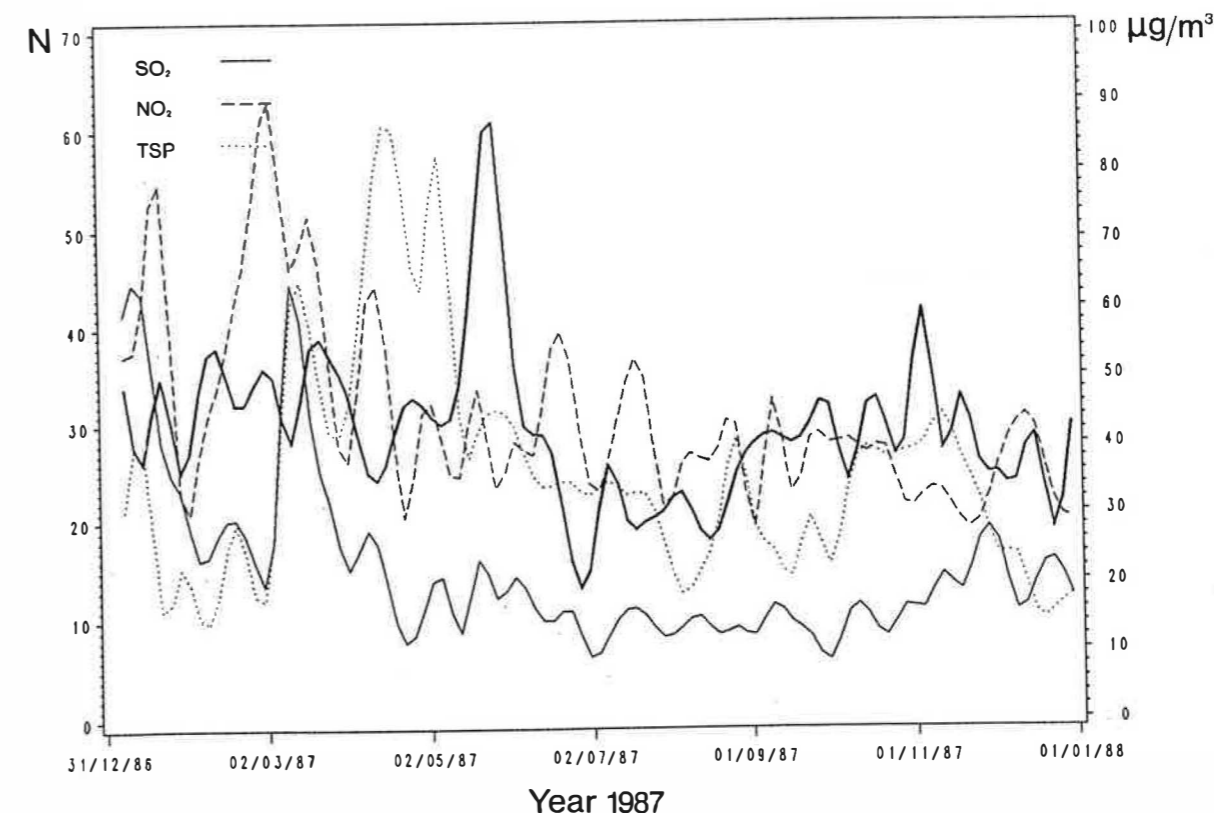


Fig. 1. Weekly mean concentrations of SO<sub>2</sub>, NO<sub>2</sub>, and TSP, and numbers of admissions that resulted from asthma (thick line) in 1987.

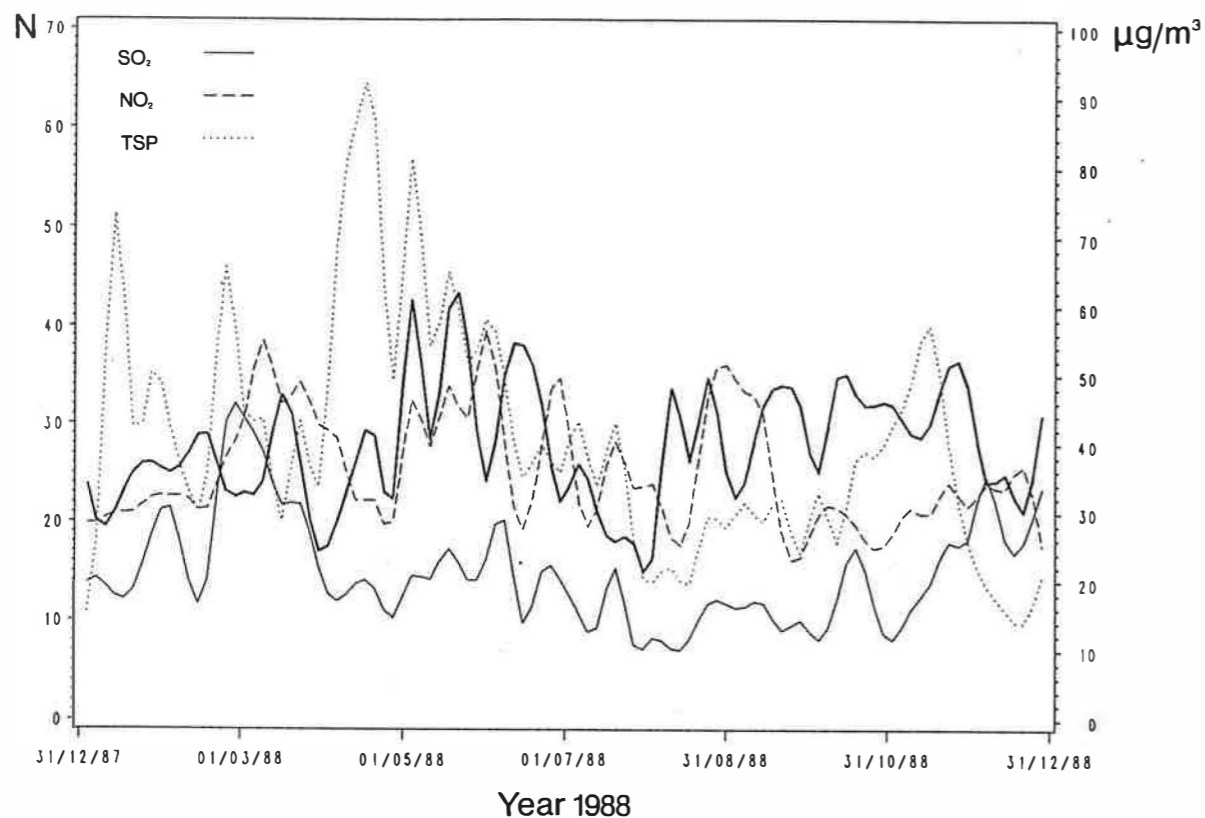


Fig. 2. Weekly mean concentrations of  $\text{SO}_2$ ,  $\text{NO}_2$ , and TSP, and numbers of admissions that resulted from asthma (thick line) in 1988.

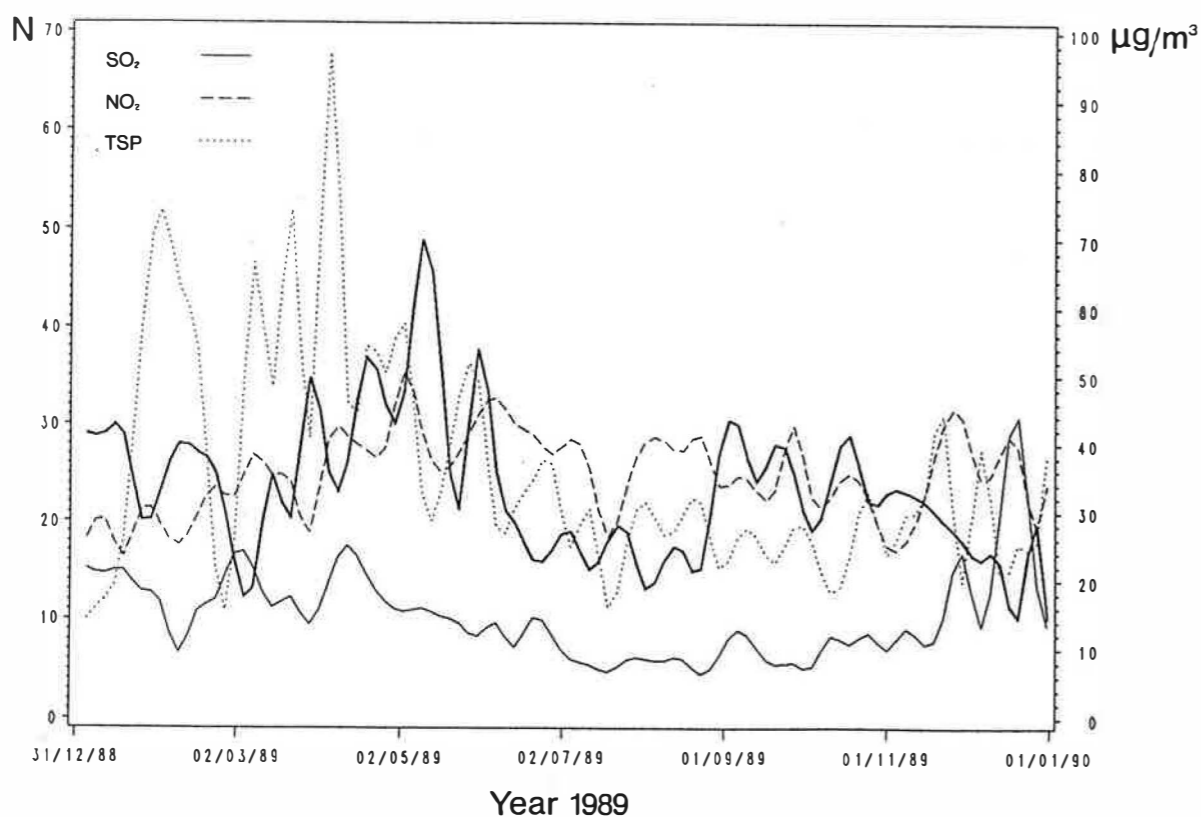


Fig. 3. Weekly mean concentrations of  $\text{SO}_2$ ,  $\text{NO}_2$ , and TSP, and numbers of admissions that resulted from asthma (thick line) in 1989.

Table 4.—Partial Correlations between Admissions for Asthma and Mean Daily Pollutant Concentrations After Standardization for Minimum Temperature

	All admissions	Admissions by emergency wards
$\text{SO}_2$	.0770 (.0172)	.1050 (.0011)
NO	.2054 ( $<.0001$ )	.1664 ( $<.0001$ )
$\text{NO}_2$	.1830 ( $<.0001$ )	.1137 (.0004)
CO	.1426 ( $<.0001$ )	.0391 (.2273)
$\text{O}_3$	.0289 (.3725)	.1083 (.0008)
TSP	.0875 (.0301)	.0995 (.0137)

Note:  $p$  values appear in parentheses.

missions to emergency wards, and CO correlated significantly with all asthma cases. In contrast,  $\text{O}_3$  had no effect on asthma among the elderly.

**Separation of the effects of weather and pollution variables.** Temperature was significantly associated with admissions; therefore, we examined the effects of pollutants by using multiple-regression analysis after standardization of minimum temperature. In the step-wise analysis, NO was the most strongly associated with all admissions ( $p < .0001$ ), followed by  $\text{O}_3$  ( $p < .0001$ ). The association with CO was almost significant ( $p = .038$ ). The effects of  $\text{NO}_2$  and  $\text{SO}_2$  were at least partially masked by NO. If the model included only  $\text{NO}_2$ ,  $\text{SO}_2$ ,  $\text{O}_3$ , and CO, the order of significance was as follows:  $\text{NO}_2$  ( $p < .0001$ ), CO ( $p < .0001$ ),  $\text{O}_3$  ( $p = .006$ ), and  $\text{SO}_2$  (not significant). The explanatory power of the model was low ( $r^2 = 0.093$ ), but it increased slightly when log-transformed values of the variables were used ( $r^2 = .139$ ).

**Comparison of admissions during periods of high and low pollution.** The number of admissions per day

were also compared during the highest quartile of air pollutants and during the three lowest quartiles (Table 7). During the period of high  $\text{SO}_2$  pollution, the mean number of all admissions was 7% greater than during the period of lower pollution. During periods of high NO,  $\text{NO}_2$ , CO, and TSP pollution, the mean number of all admissions was 28%, 29%, 15%, and 18%, respectively, greater than during the period of lower pollution.

## Discussion

This study suggests that ambient air pollutants increase asthma attacks at lower concentrations than previously believed. However, the results of earlier epidemiological studies are inconsistent. The cold climate also increased the effects of pollutants on the airways.

No clear-cut limits can be provided on the basis of health effects, but these increase gradually as concentrations of pollutants increase. Perhaps the comprehensive collection of data for a large population over a long period, as was done in the present study, can reveal the health effects more precisely than did some earlier studies that were completed with more limited material.

Several studies have reported that frequency of asthma attacks increases at low levels of pollution. Cohen et al.,<sup>21</sup> in a follow-up of 20 asthmatics for 7 mo, observed significant correlations between attack rates and temperature and between attack rates and pollution levels (after temperature was standardized). The most important single factor was cold weather. When (a) the long-term  $\text{SO}_2$  concentration exceeded  $200 \mu\text{g}/\text{m}^3$  (0.07 ppm), (b) TSP exceeded  $150 \mu\text{g}/\text{m}^3$ , or (c) temperature was less than  $32^\circ\text{F}$ , the increase in asthma attacks was significant. If the wind speed exceeded 4 mph or the relative humidity was less than 80%, the increase was almost significant.

Goren and Hellmann<sup>25</sup> investigated the prevalence of asthma and respiratory symptoms among schoolchildren who lived in a more polluted area and a less polluted area in Israel. The relative risk of asthma was 2.66-fold in the more polluted area. The mean monthly  $\text{SO}_2$  and  $\text{NO}_x$  concentrations in the more polluted area did not exceed  $11\text{--}45 \mu\text{g}/\text{m}^3$  and  $8\text{--}33 \mu\text{g}/\text{m}^3$ , respec-

Table 5.—Correlations between Admissions for Asthma and Mean Daily Concentrations of Ozone and Temperatures Simultaneously and with 1- and 2-d Lags

	Same day		1-d lag		2-d lag	
	All admissions	Admissions by emergency wards	All admissions	Admissions by emergency wards	All admissions	Admissions by emergency wards
Ozone	.0074 (NS)	.0739 (.0170)	.1499 ( $<.0001$ )	.1581 ( $<.0001$ )	.1407 ( $<.0001$ )	.1509 ( $<.0001$ )
Mean daily temperature	-.00541 (0.0841)	-.0915 (.0034)	-.0503 (NS)	-.0821 (.0087)	-.0400 (NS)	-.0778 (.0130)
Minimum temperature	-.0592 (.0585)	-.1006 (.0013)	-.0586 (.0612)	-.0911 (.0036)	-.0407 (NS)	-.0822 (.0087)

Note:  $p$  values that exceeded .10 are provided in parentheses.

Table 6.—Correlations between Admissions for Asthma and Pollutants and Temperatures, by Age Group

	0-14 y		15-64 y		> 64 y	
	All admissions	Admissions by emergency wards	All admissions	Admissions by emergency wards	All admissions	Admissions by emergency wards
SO <sub>2</sub>	-.01391 (NS)	.0332 (NS)	.1039 (.0006)	.1199 ( $<.0001$ )	.0796 (.0085)	.1169 ( $<.0001$ )
NO	.0737 (.0155)	.0772 (.0112)	.0170 ( $<.0001$ )	.1543 ( $<.0001$ )	.1557 ( $<.0001$ )	.1419 ( $<.0001$ )
NO <sub>2</sub>	.0166 (NS)	.0061 (NS)	.1648 ( $<.0001$ )	.1189 ( $<.0001$ )	.1501 ( $<.0001$ )	.1392 ( $<.0001$ )
CO	.0747 (.0146)	.0107 (NS)	.1257 ( $<.0001$ )	.0906 (.0030)	.0913 (.0028)	.0303 (.3223)
O <sub>3</sub>	.0658 (.0336)	.1051 (.0007)	-.0480 (NS)	-.0091 (NS)	.0061 (NS)	.0203 (NS)
TSP	.0489 (NS)	.0592 (NS)	.0685 (.0662)	.0775 (.0376)	.0530 (NS)	.0686 (.0656)
Mean daily temperature	.0102 (NS)	.0011 (NS)	-.0604 (.0538)	-.1082 (.0005)	-.0506 (NS)	-.0913 (.0035)
Minimum temperature	.0107 (NS)	-.0040 (NS)	-.0721 (.0213)	-.1165 (.0002)	-.0467 (NS)	-.0948 (.0024)

Note: p values that exceeded .10 are provided in parentheses.

Table 7.—Mean Daily Number of Admissions During the Low and High Pollution Periods

		All admissions	Admissions by emergency wards	Mean concentration of pollutants
SO <sub>2</sub>	Low	3.78	2.08	11.0 µg/m <sup>3</sup>
	High	4.05	2.57	23.1
	p	NS	.0002	
NO	Low	3.60	2.01	49.0 µg/m <sup>3</sup>
	High	4.59	2.72	116.3
	p	$<.0001$	$<.0001$	
NO <sub>2</sub>	Low	3.63	2.06	28.1 µg/m <sup>3</sup>
	High	4.59	2.58	45.8
	p	$<.0001$	$<.0001$	
CO	Low	3.73	2.18	0.8 mg/m <sup>3</sup>
	High	4.27	2.23	1.7
	p	.0053	NS	
O <sub>3</sub>	Low	3.58	2.15	12.3 µg/m <sup>3</sup>
	High	3.84	2.28	29.4
	p	NS	NS	
TSP	Low	3.64	2.06	42.3 µg/m <sup>3</sup>
	High	4.28	2.57	93.1
	p	.0030	.0018	

tively, but the maximum half-hour concentrations were rather high at 133-836 µg/m<sup>3</sup> and 38-528 µg/m<sup>3</sup>, respectively.

In Los Angeles, Whittemore and Korn<sup>22</sup> found a higher attack rate among adult asthmatics if the concentrations of oxidants and particulates were high or if the temperature low. The medians of 8-mo mean concentrations of particulates and oxidants were 51-121 µg/m<sup>3</sup> and 0.03-0.15 ppm, respectively.

Bates et al.<sup>10</sup> observed that the number of attendances that resulted from asthma at the emergency departments of Vancouver hospitals correlated with

SO<sub>2</sub> but not with NO<sub>2</sub> or O<sub>3</sub> levels. This phenomenon was observed in the 15-60-y age group during the summer and in the over 61-y age group during the winter. This association was observed during the same day or if there was a 1-d lag. Exact concentrations of pollutants were not provided, but the highest hourly concentrations of SO<sub>2</sub>, NO<sub>2</sub>, and O<sub>3</sub> reached 286-572 µg/m<sup>3</sup> (0.1-0.2 ppm), 564-1 128 µg/m<sup>3</sup> (0.3-0.6 ppm), and 400-800 µg/m<sup>3</sup> (0.2-0.4 ppm), respectively.

A German questionnaire survey<sup>28</sup> revealed that among children, asthma was more common in areas where there were high NO, NO<sub>2</sub>, and CO levels. The mean an-

nual concentrations in the polluted area were 120-570 µg/m<sup>3</sup> for NO, 80-280 µg/m<sup>3</sup> for NO<sub>2</sub>, and 2.0-9.0 mg/m<sup>3</sup> for CO. In contrast, SO<sub>2</sub> levels of 120-140 µg/m<sup>3</sup> and O<sub>3</sub> levels of 70-130 µg/m<sup>3</sup> did not have any effect.

Unlike the above-mentioned studies, some other studies have not reported associations between the incidence of asthma attacks and air pollutants in similar concentrations.<sup>29-32</sup> This unambiguous finding cannot be explained by the cold weather, although cold is associated with an increased incidence of asthma attacks.<sup>21,22,31,33</sup>

Even though a similar correlation was observed in the present study between the frequency of hospital admissions for asthma attacks and air pollution, it must be remembered that the incidence of asthma as a disease was not considered in this study; rather, frequency of acute attacks among persons with bronchial asthma was noted.

The harmful effects of SO<sub>2</sub>, TSP, and NO<sub>2</sub> on asthmatic persons are generally known. Conversely, NO and CO are not toxic to airways. The correlations observed in our study suggest that these compounds are indicators of air pollution, especially pollution that results from traffic.

The frequency of admissions by emergency wards was more highly correlated with SO<sub>2</sub>, O<sub>3</sub>, TSP, and cold weather than was frequency of all admissions, but all admissions correlated slightly better with pollutants that primarily indicated traffic pollution on the street level, i.e., NO, NO<sub>2</sub>, and CO. Persons with severe symptoms were first treated at the emergency wards and were later transferred to ordinary bed wards; therefore, the difference may have resulted from more severe reactions in the respiratory tract caused by SO<sub>2</sub>, TSP, O<sub>3</sub>, and cold weather among certain groups of sensitive asthmatic persons. However, the better correlation of all admissions with NO, NO<sub>2</sub>, and CO may have reflected a more comprehensive exposure of the population to traffic pollution when compared with that caused by energy production.

Despite that fact that the association of incidence of asthma attacks with relatively low levels of pollutants and cold weather was obvious in our study, these factors account for an explanatory power of approximately 14% in regression analysis. Other factors were probably more important.

The results of the present study, and those of some other studies, show that ambient air pollutants, at concentrations lower than the guidelines provided in many countries and by the World Health Organization,<sup>2</sup> may cause an increase in the incidence of asthma.

\*\*\*\*\*

Submitted for publication August 27, 1990; revised; accepted for publication January 21, 1991.

Requests for reprints should be sent to: Antti Pönkä, M.D., Chief, Department of Environmental Health, Helsinki City Health Department, Viipurinkatu 2, 00510 Helsinki, Finland.

\*\*\*\*\*

References

1. Sheppard D, Saisho A, Nadel JA, Boushey HA. Exercise increases sulfur dioxide-induced bronchoconstriction in asthmatic subjects. *Am Rev Respir Dis* 1981; 123:486-91.

2. Air Quality Guidelines for Europe. WHO Regional Publications, European series no. 23. World Health Organization, Copenhagen; 1987.

3. Bethel RA, Sheppard D, Epstein J, Tam E, Nadel JA, Boushey HA. Interaction of sulfur dioxide and dry cold air in causing bronchoconstriction in asthmatic subjects. *Appl Physiol* 1984; 57: 419-23.

4. Linn WS, Shamoo DA, Vinet TG, et al. Combined effect of sulfur dioxide and cold in exercising asthmatics. *Arch Environ Health* 1984; 39:339-46.

5. Bauer MA, Utell MJ, Morrow PE, Speers DM, Gibb FR. Inhalation of 0.30 ppm nitrogen dioxide potentiates exercise-induced bronchospasm in asthmatics. *Am Rev Respir Dis* 1986; 134:1203-08.

6. Orehek J, Massari JP, Gayraud P, Grimaud C, Charpin J. Effect of short-term, low-level nitrogen dioxide exposure on bronchial sensitivity of asthmatic patients. *J Clin Invest* 1976; 57:301-07.

7. Folinsbee LJ, McDonnell WF, Horstman DH. Pulmonary function and symptom responses after 6.6-hour exposure to 0.12 ppm ozone with moderate exercise. *J Air Pollut Control Assoc* 1988; 38:28-35.

8. Booth S, DeGroot J, Markusa R, Horton RJM. Detection of asthma epidemics in seven cities. *Arch Environ Health* 1965; 10:152-55.

9. Goldstein JF, Currie B. Seasonal patterns of asthma: a clue to etiology. *Environ Res* 1984; 33:201-15.

10. Bates DV, Baker-Anderson M, Sizto R. Asthma attack periodicity: a study of hospital emergency visits in Vancouver. *Environ Res* 1990; 51:51-70.

11. Weiss KB. Seasonal trends in US asthma hospitalizations and mortality. *JAMA* 1990; 263:2323-28.

12. Clarke CW. Relationship of bacterial and viral infections to exacerbations of asthma. *Thorax* 1979; 34:344-47.

13. Murray AB, Ferguson AC, Morrison B. The seasonal variation of allergic respiratory symptoms induced by house dust mites. *Ann Allergy* 1980; 45:347-50.

14. Packe GE, Ayres JG. Asthma outbreak during a thunderstorm. *Lancet* 1985; i:199-204.

15. Lebowitz MD, Collins L, Holberg CJ. Time series analyses of respiratory responses to indoor and outdoor environmental phenomena. *Environ Res* 1987; 43:332-41.

16. Platt-Mills TAE, Hayden ML, Chapman MD, Wilkins SR. Seasonal variation in dust mite and grass-pollen allergens in dust from the houses of patients with asthma. *J Allergy Clin Immunol* 1987; 79:781-91.

17. Anto JM, Sunyer J, Rodriguez-Roisin R, Suarez-Cervera M, Vazquez L, and the Toxicoepidemiological Committee. Community outbreaks of asthma associated with inhalation of soybean dust. *N Engl J Med* 1989; 320:1097-1102.

18. Shrenk HH, Heimann H, Clayton GD, Gafaer WM, Wexler H. Air pollution in Donora, epidemiology of the unusual smog episode of October 1948: preliminary report. *Public Health Bulletin* No. 306, Washington, DC, 1949.

19. Logan WPD. Mortality in the London fog incident. *Lancet* 1953; i:336-38.

20. Ministry of Health. Morbidity and mortality during the London fog. *Rep Pub Health Med Subj*, No. 95. London: Her Majesty's Station office, 1954.

21. Cohen AA, Bromberg S, Buechley RW, Heiderscheit MA, Shy CM. Asthma and air pollution from a coal-fueled power plant. *Am J Public Health* 1972; 62:1181-88.

22. Whittemore AS, Korn EL. Asthma and air pollution in the Los Angeles area. *Am J Public Health* 1980; 70:687-96.

23. Ussetti P, Roca J, Agusti AGN, Montserrat JM, Rodriguez-Roisin R, Agusti-Vidal A. Another asthma outbreak in Barcelona: role of oxides of nitrogen. *Lancet* 1984; i:156.

24. Imai M, Yoshida K, Kitabatake M. Mortality from asthma and chronic bronchitis associated with changes in sulfur oxides air pollution. *Arch Environ Health* 1986; 41:29-35.

25. Goren AJ, Hellmann S. Prevalence of respiratory symptoms and diseases in schoolchildren living in a polluted and in a low polluted area in Israel. *Environ Res* 1988; 45:28-37.

26. Draper NR, Smith H. *Applied regression analysis*, 2nd ed. New York: John Wiley & Sons, 1981.

27. SAS user's guide: statistics, 5th ed. Cary, NC: SAS Institute Inc., 1985.

28. Wichmann H-E, Hubner HR, Malin E, et al. The relevance of health risks by ambient air pollution, demonstrated by a cross-section study on croup syndrome in Baden-Wurttemberg. *Off Gesundh* 1989; 51:414-20 (in German).
29. Perry CB, Chai H, Dickey DW, et al. Effects of particulate air pollution on asthmatics. *Am J Public Health* 1983; 73:50-56.
30. Goldstein JF, Weinstein AL. Air pollution and asthma: effect of exposure to short-term sulfur dioxide peaks. *Environ Res* 1986; 40:332-45.
31. Vedal S, Schenker MB, Munoz A, Samet JM, Batterman S, Speizer TE. Daily air pollution effects on children's respiratory symptoms and peak expiratory flow. *Am J Public Health* 1987; 77:694-98.
32. Berciano FA, Dominguez J, Alvarez FV. Influence of air pollution on extrinsic childhood asthma. *Ann Allergy* 1989; 62:135-41.
33. Fleischer SLM, Asnani GC. The influence of weather on asthma in Nairobi. *Int J Biometeor* 1978; 22:263-70.

SUBSCRIBE

## archives of Environmental Health

### ORDER FORM

- ☐ YES! I would like to order a one-year subscription to **Archives of Environmental Health**, published bimonthly. I understand payment can be made to Heldref Publications or charged to my VISA/MasterCard (circle one).
- ☐ \$85.00 annual rate

ACCOUNT# \_\_\_\_\_ EXPIRATION DATE \_\_\_\_\_

SIGNATURE \_\_\_\_\_

NAME/INSTITUTION \_\_\_\_\_

ADDRESS \_\_\_\_\_

CITY/STATE/ZIP \_\_\_\_\_

COUNTRY \_\_\_\_\_

ADD \$12.00 FOR POSTAGE OUTSIDE THE U.S. ALLOW 6 WEEKS FOR DELIVERY OF FIRST ISSUE.

### SEND ORDER FORM AND PAYMENT TO:

HELDREF PUBLICATIONS, ARCHIVES OF ENVIRONMENTAL HEALTH  
1319 EIGHTEENTH STREET, NW, WASHINGTON, DC 20036-1802  
PHONE (202) 296-6267 FAX (202) 296-5149  
SUBSCRIPTION ORDERS 1(800) 365-9753

- For 47 years, this noted journal has provided objective documentation of the effects of environmental agents on human health. In one single source **Archives of Environmental Health: An International Journal** brings together the latest research from such varying fields as epidemiology, toxicology, biostatistics, and biochemistry.
- In a field where today's certainty often becomes tomorrow's myth, this journal publishes new research with scientific integrity and rigorous methodology.

## Ambient Air Pollution and Cancer in California Seventh-day Adventists

PAUL K. MILLS, Ph.D., M.P.H.  
DAVID ABBEY, Ph.D.  
W. LAWRENCE BEESON, M.S.P.H.  
FLOYD PETERSEN, M.P.H.  
Department of Public Health and  
Preventive Medicine  
School of Medicine  
Loma Linda University  
Loma Linda, California

**ABSTRACT.** Cancer incidence and mortality in a cohort of 6 000 Seventh-day Adventist nonsmokers who were residents of California were monitored for a 6-y period, and relationships with long-term ambient concentrations of total suspended particulates (TSPs) and ozone (O<sub>3</sub>) were studied. Ambient concentrations were expressed as mean concentrations and exceedance frequencies, which are the number of hours during which concentrations exceeded specified cutoffs (e.g., federal and California air quality standards). Risk of malignant neoplasms in females increased concurrently with exceedance frequencies for all TSP cutoffs, except the lowest, and these increased risks were highly statistically significant. An increased risk of respiratory cancers was associated with only one cutoff of O<sub>3</sub>, and this result was of borderline significance. These results are presented in the context of setting standards for these two air pollutants.

ADVERSE HEALTH EFFECTS associated with ambient air pollution have been scrutinized by environmental scientists for most of the twentieth century.<sup>1,2</sup> There is evidence that air pollution contributes to morbidity from airway obstructive disease and other forms of respiratory disease, cancer, and cardiovascular disease.<sup>3,4</sup> It is difficult to evaluate this evidence because the effects of tobacco smoke are difficult to separate from the effects of air pollution, especially in urban areas where the numbers of smokers and concentrations of air pollutants are higher than in more rural areas. Cigarette smoking has been implicated in the etiology of cardiovascular disease, respiratory diseases, lung cancer, and several other forms of cancer in humans (e.g., cancers of the bladder and pancreas).

Most studies of air pollution and cancer in humans have been epidemiologic investigations that used

cross-sectional or correlational design.<sup>5,6</sup> In this approach, indices of air pollution in certain geographic locales were correlated with the age-adjusted cancer mortality rates in the same areas, with or without adjustment for consumption of cigarettes. Most of these studies have not demonstrated that an increased cancer risk occurs with an increase in air pollution levels. In fact, the U.S. Environmental Protection Agency criteria document for particulate matter and sulfur oxides concluded: "... nor does there presently exist credible epidemiological evidence linking increased cancer rates to elevations in particulate matter as a class, i.e., undifferentiated as to chemical content."<sup>7</sup> In many instances, however, an inability to detect relationships can be linked with limitations in study design, poor statistical power, or other methodological weaknesses.

Seventh-day Adventists provide a unique opportunity