

A STUDY OF EFFECTS OF AMBIENT URBAN AIR POLLUTION USING PERSONAL SAMPLERS; A PRELIMINARY REPORT

F. Silverman, P. Corey, S. Mintz, P. Olver, and R. Hosein

The Gage Research Institute, University of Toronto, 223 College Street, Toronto, Ontario, Canada M5T 1R4

Air pollution has been associated with an increased incidence of respiratory disease. However, significant differences may exist between air pollution levels measured at conventional fixed monitoring stations and actual levels inhaled by a subject. Furthermore, studies of effects of air pollution might best be done using asthmatics as study subjects, since they have irritable airways. This is a preliminary report of a study using a control and asthmatic group in which effects of air pollution are assessed by symptom and medication diaries and simple pulmonary function tests. Air pollution exposure is measured using a small portable sampler for particulates, SO₂ and NO₂; these samplers are carried by the subject ("personal") and are situated inside and outside the home. Levels obtained are compared to data obtained from the same type of sampler located at a fixed monitoring station. Preliminary analysis of the data shows that the levels of pollutants are low and there are significant differences between the four air pollution measurements, with weak correlations among the various measurements. In this preliminary report, change in pulmonary function during the day correlates only with personal NO₂ measurements. This suggests the need for estimating air pollution exposure using "personal" samplers, when investigating health effects.

Introduction

Health impact of exposure to air pollution, particularly at low ambient levels, is still somewhat uncertain. There have been studies suggesting an association of air pollution with an increased incidence of respiratory diseases (Cassell *et al.*, 1969; Verma *et al.*, 1969; Carnow *et al.*, 1969; Fry *et al.*, 1962) and asthmatic attacks (Schoettlin and Landau, 1961; Schrenk *et al.*, 1949; Ministry of Health, 1954; Glasser *et al.*, 1967; Zeidberg *et al.*, 1961; Zweiman *et al.*, 1972). However, there have been difficulties involved in clearly defining effects of air pollution; two major areas require clarification. In the first place, the studies have suffered from inadequacies in precise assessment of each person's exposure to air pollution (Lee and Mage, 1979). Until recently, studies of effects of air pollution have relied on pollution exposure measurements obtained from fixed location air pollution stations monitoring outside air. The assumption has been made that indoor pollution bears a fixed relationship to outdoor pollution.

However, it is becoming increasingly evident that there are substantial differences between air pollution levels measured at such sites and measurements carried

out a short distance away; these differ from measurements made inside structures and the levels actually encountered by an individual carrying out daily activities (Lee and Mage, 1979; Dockery and Spengler, 1977; Berk *et al.*, 1979; Moschandreas and Morse, 1979; Colome and Spengler, 1979; Cortese and Spengler, 1976; Spengler *et al.*, 1979). Therefore, assessment of health effects of air pollution should actually assess an individual's exposure to air pollution, i.e., "personal exposure" (Lee and Mage, 1979; Dockery and Spengler, 1977).

Another problem has been the use of severe symptoms or signs as sole indicators of pollution effects. Such studies would ignore minor fluctuations in disease in, for example, a group of patients with preexisting lung disease. The definition of the health status of the study group has also been uncertain in at least some studies.

The present study was undertaken to address these problems. Health effects of ambient air pollution are being examined in two groups of people in Toronto, Ontario. Both a carefully characterized group of persons suffering from asthma and a healthy nonasthmatic control group are included in an ongoing study of health effects of air pollutants. Asthmatics were chosen as

study subjects because previous evidence (Zweiman *et al.*, 1972; Silverman, 1979; EPA, 1979b) suggested that they might be more sensitive to adverse effects of inhaling air pollution. Since one of the characteristics of asthma is irritable airways, it might be expected that asthmatics would have altered sensitivity to respiratory irritant air pollutants.

The objectives of these studies are:

(1) to estimate human exposure to air pollution by measuring air pollution levels from: (a) a conventional fixed location air pollution monitoring network, (b) indoor air pollution monitoring, (c) outdoor air pollution monitoring, and (d) "personal" air pollution monitoring;

(2) to assess the relationships among these estimates of exposure;

(3) to assess the relative strength of each of these four estimates of exposure as demonstrated by its association with health effects.

The following report describes the design of the present study and some preliminary analyses of initial data.

Methods

Health effects of ambient urban air pollution are being examined in a group of comprehensively documented asthmatic patients and a group of healthy nonasthmatic subjects. Effects are assessed by recording in a diary (symptoms, medication use, and activities), as well as by simple spirometry. Outdoor air pollution exposure is assessed by data obtained from the Ontario Ministry of the Environment (OMOE) air pollution monitoring network, as well as meteorologic data obtained from The Atmospheric Environment Service, Environment Canada. Diary information and air pollution network data is obtained daily for at least 1 yr on each individual; each subject carries a personal air pollution sampler [for sulphur dioxide (SO_2), nitrogen dioxide (NO_2) and particulates] and undergoes spirometric testing daily for a period of 4 weeks (2 weeks during the "heating" season and 2 weeks during the "nonheating" season). In addition, samplers of the same design as the personal samplers are placed inside and outside the homes of a subset of the subjects. One sampler is also placed at a central OMOE station daily.

Subject selection

All subjects are nonsmokers, do not have gas stoves, are not using a fireplace (during sampling periods), and live near one of the stations of a conventional fixed location (OMOE) air pollution monitoring network.

The asthmatic volunteers are selected from the patient population of The Gage Research Institute Asthma Clinic. There are approximately 725 patients attending the clinic. Each 6-12 months, the patients have a comprehensive assessment: a complete and uniformly documented (computer-ready) history and physical exami-

nation; biochemistry and serology; chest X-ray; and measurements of pulmonary function. Diagnosis of asthma is based on history (consistent with intermittent diffuse airways obstruction), physical examination (expiratory rhonchi), and lung function tests (improvement of airways obstruction following a standardized inhalation of the bronchodilator salbutamol). Asthmatics are selected for the current study if they wheeze at least a few times a week.

Attempts are made to match healthy nonasthmatics with asthmatics (for age, sex, and similarity of homes) by selecting subjects from an existing volunteer file or by having the asthmatics ask their neighbours to cooperate. Nonasthmatic subjects are assessed by a general medical and respiratory questionnaire and limited pulmonary function tests to establish their nonasthmatic status.

Site selection

Over a 1-yr period, pollutant levels in 26 homes in the Toronto area are being determined using at least 13 asthmatic homes and 13 nonasthmatic ones (paired for similarity of homes and geographic location). Indoor levels are compared to outdoor levels as measured by the same type of sampler, outside each home and at the OMOE network station (as well as measurements made by standard air quality monitors at the OMOE station). A Residential Characteristics description form is filled out for each residential indoor/outdoor sampling site in order to classify residences subjectively as airtight, average, or permeable, type of heating systems, ventilation systems, and so on. The indoor sampler is placed in a major activity room other than the kitchen (usually the living room, family room, or recreation room), preferably on the main floor. The sampler is placed approximately 3-5 ft above the floor, away from hot air vents and windows. Outdoor samplers are placed at least 50 ft from the street, in the back yard, not under a tree and approximately 2-3 ft above the ground.

Air pollution sampling

Indoor, outdoor, and personal sampling is carried out for approximately 8 h daily, for 4 weeks, from Monday to Friday each week. Four samples for each day of the week (i.e., 4 Mondays, 4 Tuesdays, etc.) are obtained on each subject/home spread over 2 seasons (half in the "heating" season and half in the "nonheating"). The same sampler is operated at the OMOE station daily throughout the study.

Details of the samplers are described elsewhere (Mintz *et al.*, 1982). The sampler is a battery-operated pump system, consisting of a filter assembly leading to two impingers, each containing appropriate absorbing reagent. The samplers are completely prepared and checked each day before they are taken into the field for use; then the air-flows through the bubbling systems for SO_2 and NO_2 are recorded and the timer started. The

subject is instructed on use and care of the personal sampler but is asked not to handle the indoor-outdoor samplers. At the end of the sampling period, the flows and elapsed times of all samplers are recorded. The filters are stored flat in small Petri dishes until conditioning and weighing can be completed. The SO₂ concentration is assessed by the method of West and Gaeke (1956) and the U.S. EPA (EPA, 1971); NO₂ by the TGS-ANSA method (Mulik *et al.*, 1974).

Quality control

The samplers undergo quality control procedures on a regular basis in an environmental chamber. Near-ambient NO₂ and SO₂ concentrations are obtained by dilution of pure pollutant gases with filtered air and are continuously fed and exhausted through the chamber. Concentrations are verified by Monitor Lab SO₂ and NO₂ Analyzers (models 8850 and 8840, respectively). These direct-reading instruments are calibrated regularly by the OMOE Air Resources Branch, Instrumentation Unit, using calibrated gas sources traceable to NBS standards. The multipollutant sampling probe is placed within 10 cm of the SO₂ and NO₂ analyzer sampling probes. The chemical analysis concentrations obtained by the sampler are compared with the average of 5-min readings for SO₂ and NO₂, respectively, over an 8-h time period. SO₂ and NO₂ flows are recorded hourly. For particulate collection quality assurance, the samplers are run in parallel and the filter loads compared.

Activities and health effects

Subjects chosen for study are asked to fill in diaries daily; at the beginning of the study, a trained technician explains the questions and the possible answers. The diary is designed for easy computer handling; it covers the subjects' well-being and medications, as well as their daily activity pattern. Completed diaries are reviewed with the subject during visits to the home and unclear answers are clarified. This diary is filled in for at least 1 yr.

During the time of intensive sampling, a more detailed questionnaire is administered by the technician at the end of each sampling day. The subject's location and time spent wearing the sampler is verified. The subject continues filling in the daily diary, which is checked on days when the multipollutant samplers are being used. Lung function tests (spirometry, using a Vitalograph) are performed on each subject at the beginning and end of each monitoring day. Measurements include: forced vital capacity (FVC), forced expiratory volume in one second (FEV_{1.0}) and forced expiratory flow during the middle half of the FVC (FEF_{25%-75%}).

Statistical analyses

All data management and statistical analyses were carried out using the statistical package SAS (SAS User's Guide, 1979). Mean pollutant levels were compared

across the four sampling sites using Duncan's multiple comparisons test (Duncan, 1975). Pairwise associations among the pollutant levels sampled at the four sampling sites were assessed using the Pearson product-moment correlation coefficient and verified using Kendall's non-parametric correlation coefficient. Correlations between the mean daily pollutant level and the corresponding daily change in pulmonary function were obtained separately for asthmatics and nonasthmatics. For all of these analyses, the measurements taken from each individual were pooled, thus making the sampling unit the person-day. Furthermore, comparisons across the four sampling sites involved only those person-days that had complete information from all four sampling sites.

Results

The following results were obtained from preliminary analysis of data obtained for 12 asthmatic and 11 non-asthmatic subjects (and their homes) monitored for at least 2 weeks (summer or winter) and in some cases for up to 4 weeks, both summer and winter. Table 1 shows mean pollutant levels for NO₂, SO₂, and particulates for 17 subjects at the four sites [personal, outside homes, inside homes, and at the network station (OMOE)], using identical samplers at all the sites. In order to make unbiased comparisons between the four sites, means and correlation coefficients were calculated using only the subset of days for which there was complete information across the four sites. The data has not yet been analyzed for season (heating, nonheating). The *N* shown is the number of person-days with complete pollutant information at all four sites on a given day. The means joined by a line are not significantly different from each other at the 5% level using Duncan's multiple comparisons test. SO₂ and NO₂ concentrations are given in µg/m³. In all cases, the levels were low.

All comparisons were significantly different except (1) "personal" and outdoor SO₂ levels (for 102 person-days) and (2) indoor and outdoor particulate levels (for 106 person-days). The data does, however, show that for all three pollutants the mean level measured at the OMOE site was significantly different from that mea-

Table 1. Mean pollutant concentrations.

	NO ₂ µg/m ³	SO ₂ µg/m ³	Particulates µg/m ³
<i>N</i> *	104	102	106
Personal	37 ± 20	9 ± 16	106 ± 65
Outdoor	31 ± 21	9 ± 13	71 ± 35
Indoor	25 ± 15	3 ± 8	73 ± 40
OMOE	48 ± 24	13 ± 16	92 ± 44

**N* = Number of person-days with complete information.

**Means joined not significantly different ($p > 0.05$) Duncan's multiple comparisons test.

sured by the personal sampler carried by the subject. For the pollutants NO₂ (for 104 person-days) and SO₂ the indoor levels were significantly lower than the outdoor levels; the conventional fixed location (OMOE) site also registered higher concentrations than the other sites. The personal sampler registered the highest particulate values.

In order to assess health effects of air pollution accurately, one might use the estimates from any of the four sites (even though they are different), if they were highly intercorrelated. However, Table 2 shows that the correlation coefficients of the NO₂ measurements taken at the four locations, although significant, are very small. In particular, note the low correlation of 0.49 for the personal and OMOE NO₂ levels; the highest correlations are 0.68 (OMOE vs outdoor) and 0.69 (outdoor vs indoor). These correlations only account for approximately 24% and 48% of the variation in the data.

Table 3 gives the correlation coefficients of the SO₂ measurements taken at the four locations. The concentrations are extremely low and the correlations are even smaller than for NO₂. In particular, one should note the low correlation (0.17) for the personal and OMOE SO₂ levels and 0.08 for personal vs indoor; neither is significant.

Pulmonary function measurements were made at the beginning and end of each day. The changes in pulmonary function were calculated by subtracting the afternoon value from the morning value; a positive difference would correspond to a decrease in pulmonary function over the day. The correlation coefficients between the daily change in pulmonary function and NO₂ levels measured by the sampler carried by 6 nonasthmatics and at the OMOE site for a total of 40 days are shown in Table 4. In this case, in order to make unbiased comparisons, the correlation coefficients were calculated using only that subset of days for which there was both complete NO₂ information across the four sites and also complete pulmonary function information. A positive correlation corresponds to a decrease in pulmonary function with an increase in pollutant level. In all cases the coefficients are low, both for the personal and OMOE monitoring data, and none are significant. Table 5 shows the correlation coefficients between change in pulmonary function and NO₂ levels for eight asthmatics for a total of 41 days. The correlations,

Table 2. Correlation matrices for NO₂ concentrations at four locations.*

		Indoor	Outdoor	OMOE
NO ₂ N** = 104	Personal	0.53	0.55	0.49
	Indoor		0.69	0.60
	Outdoor			0.68
	OMOE			

* All correlations are significant ($p < 0.05$).

**N = Number of subject-days with complete pollutant information at all four locations.

Table 3. Correlation matrices for SO₂ concentrations at four locations.

		Indoor	Outdoor	OMOE
SO ₂ N* = 102	Personal	0.08	0.24**	0.17
	Indoor		0.43**	0.29**
	Outdoor			0.18

*N = Number of subject-days with complete pollutant information at all four locations.

**Correlation significant ($p < 0.05$).

although low, are significantly positive for all three pulmonary function tests when the NO₂ was measured by the sampler carried by the asthmatic, but none were significant when the NO₂ levels were measured at the OMOE site.

Discussion

Interest in personal air pollution monitoring has been increasing (EPA, 1979a) due to the increased awareness of differences in air pollution levels at different locations. There are many factors which contribute to such differences. Outdoor air pollution levels are commonly measured at air pollution stations located at fixed sites some distance from an individual. Meteorologic and other factors can cause wide variations in air pollution levels at small distances (both horizontally and vertically) from such stations. Also, personal air pollution exposure depends upon mobility, activity, occupation, and life style patterns of the population (Cortese and Spengler, 1976; Godin *et al.*, 1972; Wright *et al.*, 1975). Furthermore, pollutants are released into indoor environments as a result of the occupants' activities, e.g., cooking, cigarette smoking, etc.; these factors may depend on the season of the year. Measures to improve insulation for energy conservation reduce ventilation and can lead to increased levels of pollutants indoors as compared with outdoors (Berk *et al.*, 1979).

In these preliminary analyses of the data available so far, the levels of pollutants measured are low, but differ among the four methods of estimation: "conventional," "indoor," "outdoor," and "personal." The intercorrela-

Table 4. Correlation matrices. NO₂ concentrations and changes in pulmonary function.*

		Nonasthmatics N** = 40	
		Personal	OMOE
NO ₂	Δ FVC†	0.15	0.14
	Δ FEV†	0.13	0.10
	Δ FEF† _{25%-75%}	0.28	0.10

* All correlations are not significant ($p > 0.05$).

**N = Number of subject-days with complete pollutant and pulmonary function data.

†a.m. value - p.m. value.

Table 5. Correlation matrices. NO₂ concentrations and changes in pulmonary function.

		Asthmatics N* = 41	
		Personal	OMOE
NO ₂	Δ FVC**	0.46†	0.23
	Δ FEV**	0.52†	0.22
	Δ FEF** 25%–75%	0.53†	0.07

*N = Number of subject-days with complete pollutant and pulmonary function data.

** a.m. value – p.m. value.

†p < 0.005.

tions of the pollutant levels among the four methods of estimation, while significant in some instances, are relatively weak. Further, the data showed that changes in pulmonary function over the day in asthmatics were significantly related to NO₂ levels when measured by the personal sampler, but not when measured at the fixed location site. Therefore, in terms of health effects assessment, the estimates of exposure are not interchangeable. The personal sampler carried by the subjects is more likely to be representative of the environments encountered by the subjects.

The preliminary finding that changes in lung function over the day are correlated with NO₂ levels (correlations of approximately 0.50) in asthmatics but not in nonasthmatics suggest that asthmatics are more susceptible to ambient levels of NO₂ than nonasthmatics. This is somewhat surprising, given the relatively low levels of NO₂ recorded (approximately 37 μg/m³) and must be viewed with caution in light of the preliminary nature of the analyses. The NO₂ levels account for only 25% of the variation in the daily change in pulmonary function. Other substances in the environment may be acting additively or synergistically in producing the admittedly small changes in pulmonary function. While the possibility exists, from these studies, for examining SO₂ and particulates individually or in combination (from the personal sampler) and possibly other substances (from the diary data), this has not yet been done.

Health effects depend on exposure, and health effects studies therefore rely upon estimates of exposure; it would appear that the method of choice for assessing personal air pollution exposure would use samplers carried by each subject under study. The logistics and costs of a large scale epidemiologic study with personal monitoring are great and complex. However, our preliminary data would suggest that raw data from fixed monitoring stations are really no substitute for personal sampling. Perhaps these raw data could be adjusted by taking into account, for example, varying activity patterns, outdoor and indoor concentrations, meteorological variables, and household factors in a study population.

Acknowledgements—This work was supported by Health and Welfare, Canada, Ministry of the Environment of Ontario, York-

Toronto Lung Association, and The World Health Organization/United Nations Environment Program. We gratefully acknowledge the technical assistance of B. Batten, S. Chung, L. Dixon, M. Lynch, and B. Urch and the secretarial help of A. Au Yeung.

References

- Berk, J. V., Hollowell, C. D., and Lin, C. I. (1979) Indoor air quality measurements in energy-efficient homes. Presented at 72nd Annual Meeting of the Air Pollution Control Association, June 24–29, Cincinnati, OH. Session 14: Indoor-Outdoor Air Pollutants, 79-14.2, 1–23.
- Carnow, B. W., Leppes, H. W., Shekelle, R. B., and Stammler, J. (1969) Chicago air pollution study: SO₂ levels and acute illness in patients with chronic broncho-pulmonary disease, *Arch. Environ. Health* **18**, 768–776.
- Cassell, E. J., Lebowitz, M. D., Mountain, I. M., Lee, H. T., Thompson, D. J., Wolter, D. W., and McCarroll, J. R. (1969) Air pollution, weather and illness in a New York population, *Arch. Environ. Health* **18**, 523–530.
- Colome, S. D. and Spengler, J. D. (1979) Elemental composition of indoor and outdoor respirable particulates. Presented at 72nd Annual Meeting of the Air Pollution Control Association, June 24–29, Cincinnati, OH. Session 14: Indoor-Outdoor Air Pollutants, 79-14.6, 1–16.
- Cortese, A. D. and Spengler, J. D. (1976) Ability of fixed monitoring stations to represent personal carbon monoxide exposure, *J. Air Pollut. Control Assoc.* **26**, 1144–1150.
- Dockery, D. W. and Spengler, J. D. (1977) Personal exposure to respirable particles and sulfates versus ambient measurements. Presented at 70th Annual Meeting of the Air Pollution Control Association, June 20–24, Toronto, Ontario, Canada. 77-44.6, 1–11.
- Duncan, D. B. (1975) *t*-tests and intervals for comparisons suggested by the data, *Biometrics* **31**, 339–359.
- Environmental Protection Agency (1971) National primary and secondary air quality standards, *Federal Register* **36**, 8186–8201.
- Environmental Protection Agency (1979a) Proceedings of the Symposium on the Development and Usage of Personal Monitors for Exposure and Health Effects Studies. EPA-600/9-79-032. Health Effects Research and Environmental Monitoring and Support Laboratories, Research Triangle Park, NC.
- Environmental Protection Agency (1979b) Revisions to the national ambient air quality standards for photochemical oxidants. EPA, Washington, DC.
- Fry, J., Dillane, J. B., and Fry, L. (1962) Smog: 1962 vs. 1952, *Lancet* **2**, 1326.
- Glasser, M., Greenburg, L., and Field, F. (1967) Mortality and morbidity during a period of high levels of air pollution. New York, Nov. 23–25, 1966, *Arch. Environ. Health* **15**, 684–694.
- Godin, G., Wright, G., and Shephard, R. J. (1972) Urban exposure to carbon monoxide, *Arch. Environ. Health* **25**, 305–313.
- Lee, R. E. and Mage, D. T. (1979) Personal Exposure Monitors—A Survey. Presented at 72nd Annual Meeting of the Air Pollution Control Association June 24–29, Cincinnati, OH. Session 14: Indoor-Outdoor Air Pollutants, 79-14.1, 1–16.
- Ministry of Health (1954) Mortality and morbidity during the London fog of December 1952. Reports on Public Health and Related Subjects, No. 95. Ministry of Health, London.
- Mintz, S., Hosein, H. R., Batten, B., and Silverman, F. (1982) A personal sampler for three respiratory irritants, *J. Air. Pollut. Control Assoc.* (in press).
- Moschandreas, D. J. and Morse, S. S. (1979) Exposure estimation and mobility patterns. Presented at 72nd Annual Meeting of the Air Pollution Control Association, June 24–29, Cincinnati, OH. Session 14: Indoor-Outdoor Air Pollutants, 79-14.4, 1–16.
- Mulik, J. D., Fuerst, R., Guyer, M., Meaker, J., and Sawicki, E. (1974) Development of optimization of 24-hour manual methods for the collection and colorimetric analyses of atmospheric NO₂, *Int. J. Environ. Anal. Chem.* **3**, 333–348.
- SAS User's Guide (1979) SAS Institute Inc., P.O. Box 8000, Cary, NC.
- Schoettlin, C. E. and Landau, E. (1961) Air pollution and asthmatic attacks in the Los Angeles area, *Pub. Health Reports* **76**, 545–548.
- Schrenk, H. H., Heimann, H., Clayton, C. D., Gafafer, W. M., and

- Wexler, H. (1949) Air pollution in Donora, Pa: epidemiology of the unusual smog episode of October, 1948, *Pub. Health Bull.* **306**, 21-22.
- Silverman, F. (1979) Asthma and respiratory irritants (ozone), *Environ. Health Perspect.* **29**, 131-136.
- Spengler, J. D., Ferris, B. G. Jr., Dockery, D. W., and Speizer, F. E. (1979) Sulfur dioxide and nitrogen dioxide levels inside and outside homes and the implications on health effects research, *Environ. Sci. Technol.* **13**, 1276-1280.
- Verma, M. P., Schilling, F. J., and Becker, W. H. (1969) Epidemiological study of illness absences in relation to air pollution, *Arch. Environ. Health* **18**, 536-543.
- West, P. W. and Gaeke, G. C. (1956) Fixation of sulphur dioxide as sulfitomercurate III and subsequent colorimetric determination, *Anal. Chem.* **28**, 1816-1819.
- Wright, G. R., Jewczyk, S., Onrot, J., Tomlinson, P., and Shephard, R. J. (1975) Carbon monoxide in the urban atmosphere, *Arch. Environ. Health* **30**, 123-129.
- Zeidberg, L. D., Prindle, R. A., and Landau, E. (1961) The Nashville air pollution study. I. Sulfur dioxide and bronchial asthma, *Am. Rev. Resp. Dis.* **84**, 489-503.
- Zweiman, B., Slavin, R. G., Feinberg, R. J., Falliers, C. J., and Aaron, T. H. (1972) Effects of air pollution on asthma: A review, *J. Allergy Clin. Immunol.* **50**, 305-314.