Daily Air Pollution Effects on Children's Respiratory Symptoms and Peak Expiratory Flow

SVERRE VEDAL, MD, MSc, MARC B. SCHENKER, MD, MPH, ALVARO MUNOZ, PhD, JONATHAN M. SAMET, MD, MSc, STUART BATTERMAN, PhD, AND FRANK E. SPEIZER, MD

Abstract: To identify acute respiratory health effects associated with air pollution due to coal combustion, a subgroup of elementary school-aged children was selected from a large cross-sectional study and followed daily for eight months. Children were selected to obtain three equal-sized groups: one without respiratory symptoms, one with symptoms of persistent wheeze, and one with cough or phlegm production but without persistent wheeze. Parents completed a daily diary of symptoms from which illness constellations of upper respiratory illness (URI) and lower respiratory illness (LRI) and the symptom of wheeze were derived. Peak expiratory flow rate (PEFR) was measured daily for nine consecutive weeks during the eight-month study period. Maximum hourly concentrations of sulfur dioxide, nitrogen dioxide, ozone, and coefficient of haze for each 24-hour period, as well as minimum hourly temperature, were correlated with daily URI, LRI, wheeze, and PEFR using multiple regression models adjusting for illness occurrence or level of PEFR on the immediately preceding day. Respiratory illness on the preceding day was the most important predictor of current illness. A drop in temperature was associated with increased URI and LRI but not with increased wheeze or with a decrease in level of PEFR. No air pollutant was strongly associated with respiratory illness or with level of PEFR, either in the group of children as a whole, or in either of the symptomatic subgroups; the pollutant concentrations observed, however, were uniformly lower than current ambient air quality standards. Moreover, since exposure estimation based on monitoring of ambient air likely results in misclassification of the true exposure, the negative findings of this study must be interpreted cautiously. (*Am J Public Health* 1987; 77:694–698.)

Introduction

Cross-sectional studies and most longitudinal studies on the health effects of air pollution are not designed to detect daily changes in respiratory symptoms or pulmonary function that might be related to daily changes in air pollution concentrations. Panel studies, in which subjects are followed over time and daily exposures and outcomes for each individual in the panel are monitored, have been undertaken in an attempt to identify these acute health effects.[1] These features result in potential biases that must be taken into account.

Korn and Whittemore[2] have suggested a method of analysis that adjusts for bias due to non-response and to time-dependency. They applied their method to a group of Environmetal Protection Agency Community Health and Environment Surveillance System (CHESS) panel studies in which asthmatic subjects were followed for the occurrence of asthma attacks.[3] Subjects tended to have more attacks on days with cooler temperature, higher oxidant, or higher particulate pollution. Perry, *et al,*[4] used a similar analytic method and followed subjects for symptoms of asthma and nebulizer use as well as measuring daily peak expiratory flow rate (PEFR). Fine particulate nitrates were associated with increased symptoms and with increased nebulizer use, but no associations were found for total particulates or particulate sulfates, for gaseous pollutants, or for temperature.

This report presents data from a panel study of children who were identified from a cross-sectional sample of school children used for investigating the health effects of air pollution in the Chestnut Ridge region of western Pennsylvania. This region was selected because of its high concentration of coal-fired power plants and its extensive air pollution monitoring network. Results from the cross-sectional analyses of adult women and of children from this population have been previously reported.[5,6] This panel study was not limited to asthmatic subjects and included analysis of upper and lower respiratory illness in addition to wheeze occurrence and measurement of PEFR. Analyses were performed both with and without control for possible biases due to non-response or time-dependency.

Methods

Sample Population

The children in this study were a subsample from the 1979 Chestnut Ridge cross-sectional study of over 4,000 elementary school age children.[6] As part of the cross-sectional study, parents completed a modified version of the American Thoracic Society Children's Respiratory Questionnaire (ATS-DLD-78C).[7] The subsample was obtained by first limiting participants to the six schools located in the study area that had consistently higher levels of air pollution over the previous four years. Three approximately equally sized groups of children, classified by responses to the respiratory symptom questionnaire in 1979, were selected to participate. The first group comprised all children with persistent wheeze, defined as wheeze with colds and occasionally apart from colds, or wheeze on most days or nights. A second group comprised all children without persistent wheeze but with cough and/or phlegm production for most days of the week for at least three months. The third group of children had neither persistent wheeze nor chronic cough or phlegm production and was selected to obtain an age, sex, and geographic distribution similar to those of the other two groups.

Parents of the children who agreed to participate in the eight-month follow-up study begining in the fall of 1980 completed the ATS-DLD-78C questionnaire again as they

From Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston. Address reprint requests to Sverre Vedal, MD, Department of Medicine, University of British Columbia, Vancouver General Hospital, 2775 Heather Street, Vancouver, British Columbia, Canada V5Z 3J5. Dr. Schenker is with the Occupational and Environmental Health Unit, Department of Internal Medicine, University of California at Davis; Dr. Speizer is with Channing Laboratory, Department of Medicine; Dr. Munoz is with the Department of Epidemiology, Johns Hopkins University, School of Hygiene and Public Health; Dr. Samet is with the Department of Medicine, University of New Mexico School of Medicine; and Dr. Batterman is with the Massachusetts Institute of Technology, Energy Laboratory. This paper, submitted to the Journal June 7, 1985, was revised and accepted for publication October 9, 1986.

had one and one-half years previously. Responses were compared between the two sets of questionnaires.

Daily Diaries and Peak Flow Measurement

Parents and children were instructed at the beginning of the school year in completing daily diaries of respiratory symptoms. The diaries were eight-month calendars with space for entry of numbers corresponding to symptoms for each day.[8] Possible symptom entries included hoarseness, sore throat, cough, phlegm from the chest, pain in the chest, wheezing, fever, ear pain or discharge, runny or stuffed nose, headache or muscle ache, and burning, aching, or redness of the eyes. Parents were contacted by telephone every two weeks to ensure that the diary was being completed. Responses were read from the diary at those times and recorded independently by the staff.

For nine consecutive weeks during this eight-month period, morning PEFR measurements were obtained on each school day, with approximately one-fourth of the children supplying measurements during any nine-week period. Each child was assigned a specific Mini-Wright Peak Flow Meter (Armstrong Industries, Inc., Northbrook, IL) which was used throughout the study period, and performed the PEFR maneuver three times each school morning. The maximum of the three efforts was used for the analysis.

Three symptom outcomes were defined from the diary accounts using definitions conforming to those used in the Tecumseh study of respiratory illness:[9] 1) wheeze; 2) upper respiratory illness (URI), defined as the presence of coryza; and 3) lower respiratory illness (LRI), defined by wheeze, pain on breathing, or phlegm production. An episode for LRI or URI was defined as the first day of at least two consecutive days of symptoms if preceded by at least two days without LRI or URI, respectively. URI was only diagnosed if LRI was not also present. An episode of wheeze was defined for each day wheeze occurred. Incidence rates for each symptom were calculated as the ratio of the number of episodes of each symptom to the number of person-days for which data were present and for which subjects were at risk for a symptom episode.

Air Pollutants and Temperature

Pollutant data were obtained from two sources. Sulfur dioxide (SO_2) concentrations were measured at 17 monitoring stations and averaged. Daily changes in level at a single SO_2 monitor were generally reflected at all monitors, with most correlation coefficients between monitor pairs ranging from 0.4 to 0.8.[10] Nitrogen dioxide (NO_2) and ozone (O_3) concentrations, and coefficient of haze (CoH), were measured at a single monitoring site in the study region. Maximum hourly levels for each 24-hour period (7 pm through 6 pm) were used to reflect the daily level for each pollutant. Minimum hourly temperature was recorded at a centrally located monitoring station. Details of the terrain, meteorology, pollutant sources, and monitor locations have been reported.[6,10]

Analysis

Three separate analyses were performed. First, a preliminary analysis was done using incidence rates of LRI, URI, and wheeze episodes. Incidence rates were calculated for the entire sample and for the three subgroups. The association between levels of air pollutants or temperature, and incidence rates, was evaluated by calculating incidence rates for strata of air pollutants and temperature levels. Also, incidence rates for each symptom were divided at the mean into high and low incidence groups. Mean pollutant concentrations and temperature for each group were compared.

This method of analysis is limited by failure to account for interdependency of symptoms from one day to the next or one episode to the next, and for missing data in which either more symptomatic or less symptomatic subjects drop into or out of the study. We used two other analytical approaches to account for these limitations. One approach, proposed by Korn and Whittemore,[2] models each subject's probability of having an episode of a given symptom by a multiple logistic regression equation. The independent variables were the symptom outcome on the previous day, the maximum daily pollutant values on the current day, and the minimum current daily temperature. The coefficients of the explanatory variables in the logistic models are estimates of the change in symptom logarithmic odds ratios for each unit change in the explanatory variables. The variable indicating presence or absence of symptoms on the previous day attempts to remove from the analysis the dependency of current symptoms on previous symptom history. The summary coefficient over all subjects was calculated as a weighted average of the individual coefficients, each coefficient weighted by the inverse of its variance. The model used corresponds to the "fixed effects" case reported by Korn and Whittemore.[2] Symptoms odds ratios were calculated as the antilogarithms of the regression coefficients and their 95 per cent confidence intervals were calculated.[11] Subgroup susceptibility was evaluated by averaging summary coefficients only within a given symptom subgroup.

A similar approach was used to evaluate the association between PEFR and air pollution or temperature. The PEFR was modeled as a linear function of the previous day's PEFR and the other explanatory variables used in the preceding logistic analysis. However, in this analysis each subject's mean PEFR was allowed to vary; the effect of PEFR on the previous day and the current day's pollutant concentrations and temperature were assumed to be the same for each subject. Specifically we use the model:

$$Y_{it} = \alpha_0 + \alpha_i + \delta Y_{i,\,t-1} + \beta x_t + e_{it}$$

where Y_{it} represents the level of PEFR for individual i on day t, α_0 the intercept, α_i the addition to the intercept required to obtain the mean PEFR for individual i ($\Sigma\alpha_i = 0$), δ the coefficient of the effect of PEFR on day t-1, β the vector of coefficients representing the temperature and pollutant variables on day t, and e the error term which is assumed to be normally distributed with zero mean and unknown variance σ^2. Coefficients from this model estimate the change in PEFR for each unit change in the explanatory variables.

Results

Of the 351 subjects selected for the eight months of follow-up, 128 participated in the completion of diaries, 144 performed PEFRs, and 122 had the ATS Children's Questionnaire completed again in 1980. The 229 children whose parents did not complete the second questionnaire were compared with those children who participated. Nonparticipating children were comparable in age (9.5 vs 10.1 years) and sex (55 per cent male vs 62 per cent male), but had slightly less chronic cough (34 per cent vs 42 per cent) and chronic phlegm production (22 per cent vs 28 per cent) based on the initial questionnaire responses than participating children. Nonparticipating children had a 10 per cent prevalence of physician-diagnosed asthma compared to 7 per cent in participating children. Among the 122 children with a

TABLE 1—Maximum Air Pollutant Concentrations and Minimum Temperatures in Chestnut Ridge, September 1980–April 1981

	Mean	Range
SO_2 ($\mu g/m^3$)	51.2	18–176
CoH (CoH units)	0.38	0.1–1.3
NO_2 ($\mu g/m^3$)	40.5	12–79
O_3 ($\mu g/m^3$)	32.4	0–129
Temperature (°C)	−1.3	−22–+20

second parent-completed questionnaire, 33 (27 per cent) initially reported persistent wheeze, 59 (48 per cent) reported chronic cough or phelgm, and 30 (25 per cent) reported neither set of symptoms. Consistency of response between the questionnaire completed in 1979 and the questionnaire completed one and one-half years later in 1980 was 74 per cent for persistent wheeze and 71 per cent for chronic cough or phlegm production. Because of this change in symptom designation over one and one-half years, subjects were grouped according to current rather than initial symptoms.

Concentrations of the pollutants were lower during the follow-up period (Table 1) than was anticipated from concentrations measured in previous years. The mean of maximum hourly SO_2 concentrations in 24 hours of the 17 monitors was often considerably lower than the maximum hourly concentration which might be measured at a single monitor. For example, the highest mean maximum SO_2 level was 176 ug/m^3, but the highest level recorded by a single monitor was 604 ug/m^3. Levels of individual SO_2 exposures in some instances were therefore higher than those reported by the mean levels. However, as noted previously, the change in level from day to day was generally reflected by all monitors. Minimum temperature and ozone concentrations were highly correlated (Table 2). Only modest correlations were present between the other environmental variables.

An average of 79 per cent of subjects completed diaries in each month from November through March. Fewer subjects completed diaries in September and October because of staggered recruitment. Fewer subjects also completed diaries in April, the last study month.

Symptom Incidence Rates

The incidence rate of URI did not vary across symptom group (Table 3). Wheeze incidence, as expected, and LRI incidence were most common in those with persistent wheeze. No consistent trend in incidence rates was present for any of the symptom episodes over the observed ranges of air pollutants or temperatures. Minimum temperature, however, was lower on days with a higher incidence of URI or wheeze; no pollutant was substantially higher on days with higher symptom incidence (Table 4).

TABLE 2—Correlation Coefficients among the Pollutants and Temperature in Chestnut Ridge, September 1980–April 1981

	Minimum Temperature	SO_2	CoH	NO_2
SO_2	−.18			
CoH	−.32	.29		
NO_2	.27	.15	.30	
O_3	.57	.05	−.31	.38

TABLE 3—Incidence Rates (per 1,000 person-days) of Symptom Episodes in Chestnut Ridge Children Stratified by Symptom Group

	Symptom Episodes		
Symptom Group†	URI*	LRI*	Wheeze
Persistent wheeze	19	15	62
Cough or phlegm	18	9	9
Asymptomatic	21	4	9
All subjects	20	8	21

*URI = Upper respiratory illness
LRI = Lower respiratory illness
†See text for definitions

TABLE 4—Mean Level of Air Pollutant or Temperature on Days with High versus Low Symptom Incidence Rates, Chestnut Ridge, September 1980–April 1981

Symptom	Incidence†	SO_2 ($\mu g/m^3$)	CoH (CoH units)	NO_2 ($\mu g/m^3$)	O_3 ($\mu g/m^3$)	Minimum Temperature (°C)
LRI	high	52.1	.42	40.2	27.0	−2.2
	low	50.9	.37	40.6	34.1*	−1.0
URI	high	52.1	.38	38.5	29.4	−3.2
	low	50.1	.38	42.6	35.5*	0.6*
Wheeze	high	50.2	.40	41.2	28.8	−3.7
	low	52.1	.36	39.9	35.4*	0.7*

*$p < 0.05$ for difference between high vs low incidence.
†Incidence rates were divided at the mean into high and low rates.

Regression Analysis

Regression models could not be fit for subjects who never had symptoms. Consequently, 111 subjects were included in the regression analysis of URI, 55 were included in the analysis of LRI, and only 26 in the analysis of wheeze. Estimates from the Korn and Whittemore analysis indicated that the presence or absence of symptoms on the previous day was an important predictor ($p < .001$) of the occurrence of symptoms on the current day. Because neither O_3 nor NO_2 were predictive of any symptom outcome, the regression models included only symptom status of the previous day, minimum temperature, and maximum SO_2 and CoH as the independent variables. Low temperature was associated with increased occurrence of LRI and URI but not with wheeze (Table 5). For LRI, for example, given two consecutive days with the same SO_2, CoH, and symptom status as on the first day, but with temperature on the second day being 20°C less than on the first day, the odds of LRI occurring on the day with the lower temperature would be 1.3 times the odds of LRI on the day with the higher temperature. Neither SO_2 nor CoH was associated with any important increase in daily

TABLE 5—Odds Ratios (95% confidence intervals) of Respiratory Symptoms Given Changes* in the Environmental Variables, Chestnut Ridge Region, September 1980–April 1981

Symptoms	20°C Decrease in Temperature	50 $\mu g/m^3$ Increase in SO_2	0.5 unit Increase in CoH
LRI	1.3 (1.04, 1.68)	0.9 (0.61, 1.35)	1.3 (0.93, 1.82)
URI	1.2 (1.05, 1.37)	1.0 (0.92, 1.08)	0.9 (0.76, 1.07)
Wheeze	1.1 (0.79, 1.49)	1.2 (0.78, 1.56)	1.0 (0.97, 1.06)

*Amount of change was chosen to represent a substantial change in either temperature or air pollutant.

symptom occurrence, although increased CoH tended to be related to increased LRI occurrence. Subgroups of children with either chronic cough or phlegm production, or with persistent wheeze, similarly had no increase in daily symptoms associated with increases in the pollutants.

Level of PEFR on the previous day was the strongest predictor of daily PEFR. A drop in daily temperature was weakly associated with a decrease in PEFR, with a drop in temperature of 20°C being associated with a drop in PEFR of only 1 L/min ($p = 0.12$). As with respiratory symptoms, none of the pollutants was associated with a substantially decreased PEFR. The subgroup with persistent wheeze tended to be sensitive to changes in SO_2 concentration.

Models for symptoms and for PEFR were fit examining associations with temperature and pollutant levels lagged by 24 hours and by 48 hours. All associations were weaker with the lagged variables in the models than with the unlagged variables.

Discussion

Because of strong correlation between the occurrence of respiratory symptoms on any given day and symptoms on preceding or subsequent days, we used a multiple regression model similar to that proposed by Korn and Whittemore[2] in their analysis of asthma occurrence and air pollution. A modification of this approach was used for analyzing PEFR, a continuous measure which also has a strong day-to-day correlation. We found no important association in this sample of elementary school aged children between respiratory symptoms of URI, LRI, or wheeze and relatively low levels of air pollution. Potentially more susceptible subgroups, such as children with chronic cough or phlegm production, or with persistent wheeze, were not more susceptible to these levels of air pollution. Children's PEFR was similarly unassociated with the pollution levels measured. Cooler temperature was associated with more URI and LRI, but not with more wheeze, or with lower PEFR. Because characterization of exposure depended on ambient measurements, however, the absence of observed associations may have reflected misclassification of actual exposure.

The results of the analysis of symptoms and air pollution using the Korn and Whittemore method generally corroborated those using incidence rates, but only the former method detected an association between lower temperature and more LRI. Stebbings and Hayes[1] have argued that incidence rates (attack rates) not only reflect day-to-day symptom variation but also variation over longer time periods such as weeks or seasons which might be included in the follow-up period. Also, symptom incidence rates may be affected by nonresponse rates varying over the study period. Nonresponse was more prevalent early and late in this study which could have resulted in a bias of the relationship between respiratory symptoms and pollution or temperature. The Korn and Whittemore type of analysis is designed, first, to remove the time-dependency of symptoms by adjusting for symptom occurrence on the previous day, and second, to make the analysis insensitive to non-response bias by using each individual as his own control. For these reasons this approach seems justified. It is not surprising that the two different methods, one designed to remove the deficiencies in the other, show some varying results.

The observed association of LRI and URI with cooler temperatures is consistent with prior related observations. Whittemore and Korn[3] observed more asthma attacks with cooler temperatures, as have others.[12,13] Rhinitis in adults was associated with temperature in a random sample of adults in Tucson, Arizona.[14] Temperature has also been associated with respiratory mortality.[15]

A limitation of this study that is shared by all other such studies is that the ambient pollution concentrations may not adequately reflect exposures of individual subjects. Since most of a child's time during a school year is spent indoors, and since indoor pollutant concentrations, especially for NO_2 and particulates, can be markedly different from those outdoors,[16,17] the outdoor concentrations measured in this study may not have been valid estimates of each subject's exposure. However, if day-to-day variation in indoor concentrations reflected the variation in the outdoor concentrations, even though the absolute concentrations differed, then the results from the Korn and Whittemore analysis would be satisfactory. Analysis of incidence rates by levels of pollution, on the other hand, would be affected by misclassification of the exposure if measured and actual absolute pollution concentrations differed. Such misclassification, if random, would result in a bias toward the null state of no association between level of pollution and respiratory symptoms.[18] When indoor sources of NO_2 and particulates are present, however, even the day-to-day variations outdoors may not reflect those variations occurring indoors.

Another weakness of this study is that PEFR is primarily a measure of large airways function. Thus, to the degree to which the anticipated effect is due to small airways abnormalities, PEFR may not be a sensitive measure of pulmonary function decrement due to air pollution.

Air pollution concentrations measured during the study period were lower than anticipated given the concentrations observed in preceding years. On no day did the levels at any single monitor exceed the National Primary Air Quality Standards.[19] This study can at best then be interpreted as showing no acute effects of these pollutants on respiratory symptoms or PEFR in children at levels which are lower than the current standards.

Despite selecting this study population from a population-based cross-sectional survey, the low participation rate of subjects selected suggests that the children in this sample should not be taken to represent any general population of children. However, for the study findings to be invalid it would have been necessary for those children susceptible to low levels of air pollution to have selectively not participated.

The conclusions, then, which might be drawn from this study need to be tempered by the relatively low levels of air pollution encountered during the course of follow-up and by the possibility of misclassification of pollutant exposures. Higher levels might be associated with respiratory symptoms. Also, a susceptible subgroup might be affected only by levels higher than those observed. Future studies would be improved by use of more sensitive measures of pulmonary function and by stronger links between subjects and their individual exposures.

ACKNOWLEDGMENTS

This research was presented in part at the national meeting of the American Thoracic Society, Kansas City, May 1983.

Supported in part by Department of Energy Contract No. EP-78504968, Environmental Protection Agency Cooperative Agreement No. CR811650-01, National Research Service Award No. 07427 from the National Institutes of Health. Dr. Samet is recipient of Research Career Development Award No. 5 K04 HL00951 from the Division of Lung Diseases, National Heart, Lung and Blood Institute.

REFERENCES

1. Stebbings JH, Hayes CG: Panel studies of acute health effects of air pollution. 1. Cardiopulmonary symptoms in adults, New York, 1971–1972. Environ Res 1976; 11:89–111.
2. Korn EL, Whittemore AS: Methods for analyzing panel studies of acute health effects of air pollution. Biometrics 1979; 35:795–802.
3. Whittemore AS, Korn EL: Asthma and air pollution in the Los Angeles area. Am J Public Health 1980; 70:687–696.
4. Perry GB, Chai H, Kickey DW, *et al:* Effects of particulate air pollution on asthmatics. Am J Public Health 1983; 73:50–56.
5. Schenker MB, Speizer FE, Samet JM, *et al:* Health effects of air pollution due to coal combustion in the Chestnut Ridge region of Pennsylvania. Arch Environ Health 1983; 38:325–330.
6. Schenker MB, Vedal S, Batterman S, Samet JM, Speizer FE: Health effects of air pollution due to coal combustion in the Chestnut Ridge region of Pennsylvania: cross-sectional survey of children. Arch Environ Health 1986; 41:104–108.
7. Ferris BJ Jr: Epidemiology Standardization Project. Am Rev Respir Dis 1978; 118 (6, part 2): 36–47.
8. Tager IB, Speizer FE: Surveillance techniques for respiratory illness. Arch Environ Health 1976; 31:25–28.
9. Monto AS, Napier JA, Metzner HL: The Tecumseh study of respiratory illness. 1. Plan of study and observations on syndromes of acute respiratory disease. Am J Epidemiol 1971; 94:269–279.
10. Batterman S, Golomb D: Health effects of air pollution due to coal combustion in the Chestnut Ridge region of Pennsylvania. Massachusetts Institute of Technology Energy Laboratory Report MIT-EL 85-008, August 1985
11. Schlesselman JJ: Case-control studies: designs, conduct, analysis. New York: Oxford University Press, 1982; 247.
12. Derrick EH: The seasonal variation in asthma in Brisbane: its relation to temperature and humidity. Int J Biometeorol 1965; 9:239–251.
13. Fleischer SLM: The influence of weather on asthma in Nairobi. Int J Biometeorol 1978; 22:263–270.
14. Robertson G, Lebowitz MD: Analysis of relationships between symptoms and environmental factors over time. Environ Res 1984; 33:130–143.
15. States SJ: Weather and deaths in Pittsburgh, Pennsylvania: a comparison with Birmingham, Alabama. Int J Biometeorol 1977; 21:7–15.
16. Spengler JD, Duffy CP, Letz R, *et al:* Nitrogen dioxide inside and outside 137 homes and implications for ambient air quality standards and health effects research. Environ Sci Technol 1983; 17:164–168.
17. Spengler JD, Dockery DW, Turner WA, *et al:* Long-term measurements of respirable sulfates and particulates inside and outside homes. Atmospher Environ 1981; 15:23–30.
18. Kleinbaum DS, Kupper LL, Morgenstern H: Epidemiologic research: principles and quantitative methods. Belmont, CA: Lifetime Learning Publications, 1982; 223.
19. American Thoracic Society Statement on Health Effects of Air Pollution. New York: American Thoracic Society, 1978.

Newly Revised Fact Book on *Smoking, Tobacco, and Health*

The Office on Smoking and Health of the Centers for Disease Control recently issued its newly revised publication, *Smoking, Tobacco, and Health,* a 43-page fact book which for nearly 20 years has been an important source of information for high school and college students on the medical, social and economic aspects of cigarette smoking—the chief preventable cause of death in the United States.

The new issue of the booklet was mailed earlier this year to all public and private high school libraries throughout the country so that it would be available as a research and background resource for teachers and students. In addition, as long as supplies permit, the Office on Smoking and Health will send individual copies to teachers and students on request.

To obtain a copy of the booklet, *Smoking, Tobacco, and Health,* Pub. No. (CDC) 87-8397, contact the: Office on Smoking and Health, Centers for Disease Control, US Public Health Service, Department of Health and Human Services, Rockville, MD 20857. Telephone: 301/443-5287.