

The prevalence of chronic obstructive pulmonary disease was evaluated in 12 980 lifelong nonsmoking adults who participated in one of three National US Health and Nutrition Examination Surveys. Also evaluated were the relationships between chronic obstructive pulmonary disease and age, sex, ethnicity, education, income, and certain environmental and occupational factors. Overall, 4% of men and 5% of women reported physician-diagnosed chronic obstructive pulmonary disease. Prevalence increased with age and with decreasing household income, was higher in Whites than in non-Whites, and was particularly high in Hispanic women. Further research is needed to explain the excess risk for chronic obstructive pulmonary disease in economically disadvantaged nonsmokers, and to assess the role of environmental tobacco smoke in nonsmokers' risk for the disease. (Am J Public Health. 1995:85:702-706)

Public Health Briefs

Chronic Obstructive Pulmonary Disease in Lifelong Nonsmokers: Results from NHANES

Alice S. Whittemore, PhD, Susan A. Perlin, ScD, and Yasamin DiCiccio

Introduction

Chronic obstructive pulmonary disease is a process characterized by the presence of chronic bronchitis or emphysema that may lead to the development of airways obstruction.1 Chronic obstructive pulmonary disease is a major cause of morbidity and mortality throughout the world; 82 384 such deaths occurred in the United States in 1988, making the disease the fifth leading cause of death.² Apart from its role as a direct cause of death, chronic obstructive pulmonary disease also contributes to deaths from cardiovascular disease.3 Approximately 85% of chronic obstructive pulmonary disease mortality in men and 69% of its mortality in women can be attributed to cigarette smoking.⁴⁻⁶ In the United States, this higher share of mortality among men⁷ may reflect a higher proportion of male cigarette smokers than of female cigarette smokers in the past.

Little is known about the epidemiology of chronic obstructive pulmonary disease in lifelong nonsmokers. Here we present age-, sex-, and race-specific chronic obstructive pulmonary disease prevalence rates among 12 980 never-smoking adults (3904 men and 9076 women) who participated in one of three US National Health and Nutrition Examination Surveys (NHANES). We use these data to examine associations between chronic obstructive pulmonary disease prevalence and certain demographic characteristics and occupational factors.

Methods

Study subjects were participants in either NHANES I (April 1971 to September 1975), NHANES II (February 1976 to February 1980), or the Hispanic Health and Nutrition Examination Survey (HHANES) (July 1982 to December 1984). These surveys provide a probability sample of the civilian, noninstitutionalized population of the continental United States, aged 6 months to 74 years. This analysis includes all participants aged 18 to 74 years (25 to 74 years for NHANES I) who provided information on their race and chronic obstructive pulmonary disease status and who replied negatively to the question "Have you smoked at least 100 cigarettes during your entire life?"

A history of chronic obstructive pulmonary disease was defined as an affirmative response to either "Has a doctor ever told you that you had chronic bronchitis?" or "Has a doctor ever told you that you had emphysema?" Characteristics examined in relation to chronic obstructive pulmonary disease risk include sex, age, race (White, non-White), income, educational level, body size, industry of principal job or business and job title, home heating equipment and air conditioning information, and residential characteristics (e.g., urban vs rural area, population size of area). Industries and job titles were coded using national indexes of industries and occupations (1970 edition⁸ for NHANES I and II; 1980 edition⁹ for

Alice S. Whittemore and Yasamin DiCiccio are with the Department of Health Research and Policy, Stanford University School of Medicine, Stanford, Calif. Susan A. Perlin is with the Office of Health Research, US Environmental Protection Agency, Washington, DC.

Requests for reprints should be sent to Alice S. Whittemore, PhD, Department of Health Research and Policy, Stanford University School of Medicine, Redwood Bldg, Room T204, Stanford, CA 94305-5092.

This paper was accepted October 4, 1994.

Note. This research, while supported by the US Environmental Protection Agency, has not been subjected to agency review; thus, it does not necessarily reflect the views of the agency, and no official endorsement should be inferred.

TABLE 1—The Prevalence of Self-Reported Physician-Diagnosed Chronic Obstructive Pulmonary Disease among Lifelong Nonsmokers in NHANES, by Age, Sex, Race, and Survey

	NHAN	NES I, 1971–1	975	NHAN	NES II, 1976–1	980	HHA	NES, 1982–1	984		Total	
	No.	Observed No. with COPD	%	No.	Observed No. with COPD	%	No.	Observed No. with COPD	%	No.	Observed No. with COPD	%
					Me	n						
Whites	688	31	4.5	1842	68	3.7	9 41	36	3.8	3471	135	3.9
Non-Whites	121	0	0	295	7	2.4	17	3	17.6	433	10	2.3
Age, y												
<50	423	13	3.1	1291	31	2.4	719	26	3.6	2433	70	2.
5069	317	13	4.1	656	35	5.3	212	12	5.7	1185	60	5.
70+	69	5	7.2	190	9	4.7	27	1	3.7	286	15	5.
Subtotal	809	31	3.8	2137	75	3.5	958	39	4.1	3904	145	3.
					Wor	nen						
Whites	1736	92	5.3	4028	233	5.8	2257	141	6.2	8021	466	5.8
Non-Whites	275	10	3.6	721	34	4.7	59	2	3.4	1055	46	4.
Age, y												
<50	921	43	4.7	2107	70	3.3	1535	90	5.9	4563	203	4.
50-69	885	48	5.4	1953	142	7.3	692	48	6.9	3530	238	6.
70+	205	11	5.4	689	55	8.0	89	5	5.6	983	71	7.
Subtotal	2011	102	5.1	4749	267	5.6	2316	143	6.2	9076	512	5.
Total	2820	133	4.7	6886	342	5.0	3274	182	5.6	12 980	657	5.

Note. NHANES = National Health and Nutrition Examination Survey; HHANES = Hispanic Health and Nutrition Examination Survey; COPD = chronic obstructive pulmonary disease.

HHANES). None of the surveys provided information on exposures to dusts, fumes, or chemicals potentially associated with chronic obstructive pulmonary disease risk. Therefore, reported job titles were used to classify individuals into high, medium, or low categories based on potential for exposure to airborne contaminants that may cause or exacerbate the disease.

Multiple logistic regression implemented on EGRET software¹⁰ was used to estimate regression coefficients relating chronic obstructive pulmonary disease prevalence to personal characteristics and to adjust for potential confounding factors. All P values are two tailed. The surveys were based on a weighted sampling design, with oversampling of non-Whites, individuals of lower income, and the elderly. Chronic obstructive pulmonary disease prevalence in demographic subgroups was computed both with and without the sampling weights, and similar prevalence estimates were found. Since incorporation of the weights in logistic regression is not straightforward, only the unweighted prevalence estimates are presented.

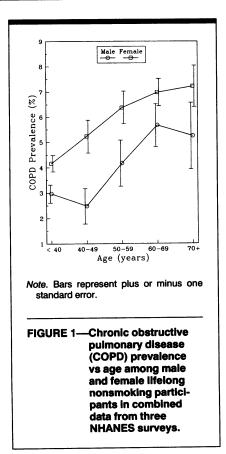
Results

In NHANES I, 2820 participants (809 men, 2011 women) stated that they

were lifelong nonsmokers. This represents 41% of all 6905 participants (3166 men, 3739 women) who provided definitive information about their lifelong smoking habits. In NHANES II, 6886 participants (2137 men, 4749 women) declared themselves lifelong nonsmokers, which was 38% of all 18 337 participants (8589 men, 9748 women) who provided smoking information. Comparable numbers for HHANES were 3274 lifelong nonsmokers (958 men, 2316 women), or 36% of all 9148 subjects (4111 men, 5037 women) who provided smoking data.

Table 1 shows chronic obstructive pulmonary disease prevalence rates by sex, race, and age group for each of the surveys. Prevalence increased with age (see also Figure 1), was higher in women (5.6%) than in men (3.7%), and, within each sex, was higher in Whites than in non-Whites. After adjustment for age and race, prevalence in men did not differ among the three surveys (P = .45). However, age- and race-adjusted prevalence in female HHANES participants was higher than that of women from the other two surveys (P = .02).

Table 2 presents odds ratios (ORs) relating chronic obstructive pulmonary disease prevalence to sex and race, by survey. After adjustment for age, race, and survey, prevalence was significantly higher in women than in men. The overall



odds ratio was 1.4, with 95% confidence interval (CI) of 1.1, 1.7. Age-adjusted prevalence of the disease was higher for

		Z	NHANES	SI			~	NHANES II	ES II			-	HHANES	ES				Total	-	
	No.	COPDª	%	оR	ō	No.	COPD ^a	%	OR	ō	No.	COPDª	%	OR	ō	No.	COPD ^a	%	оR	ō
Sex Men Women	688 ^d 2011	31 102	5.1 5.1	0	0.75, 1.7	2137 4749	75 267	3.5 5.6	1.0 1.4**	1.1, 1.9	958 2316	39 143	4.1 6.2	1.5*	1.0, 2.2	3783 9076	145 512	3.8 5.6	1.0 1.4**	1.1, 1.7
Race Men White Non-White	688 121	31	4.5 0	: • o	::	1842 295	68 7	3.7 2.4	1.0 0.62	0.28, 1.4	941 17	30 30	3.8 17.6	1.0 5.4*	 1.5, 19.7	3471 312	135 10	3.9 3.2	1.0 0.87	0.45, 1.7
women White Non-White	1736 275	92 10	5.3 3.6	1.0 0.67	0.34, 1.3	4028 721	233 34	5.8 4.7	1.0 0.84	0.58, 1.2	2257 59	141 2	6.2 3.4	1.0 0.51	0.12, 2.1	8021 1055	446 46	5.8 4.4	1.0 0.77	0.56, 1.1
Income Men \$25 000+ \$15 000-24 999 \$7 000-14 999 < \$7 000	53 153 294 165	4 4 1 5 8	7.5 5.1 4.8	1.0 0.41 0.66		362 563 393 393	4 17 29 20	1.1 3.0 5.1	1.0 2.9 3.7*	0.95, 8.6 1.3, 10.9 1.4, 13.5	286 250 98	1 ~ 1 0	3.8 9.2 9.2	1.0 2.0 2.0	0.25, 1.9 0.40, 2.5 0.71, 5.5	701 966 1245 656	19 28 37	2.7 2.9 5.6	20, 11.1	0.62, 2.1 0.98, 3.0 1.1, 3.6
Overall trend Women \$25 000-4 \$15 000-24 999 \$7 000-14 999 < \$7 000 Overall trend	86 313 745 775	4 22 4 9 31 22	4.7 7.0 5.2	1.0 1.7 0.93 0.99 0.99	0.64, 1.6 0.55, 5.0 0.31, 2.7 0.33, 3.0 0.67, 1.1	453 939 1659 1385	12 38 102 102	2.7 4.0 7.4		0.77, 2.9 1.0, 3.4 1.0, 3.7 1.0, 1.4	477 516 620 492	23 27 39	4.8 6.3 8.3		0.88, 1.8 0.65, 2.1 0.83, 2.5 1.1, 3.4 1.0, 1.5	1016 1768 3024 2652	39 87 183	3.8 5.6 6.9		0.90, 2.0 0.90, 2.0 1.0, 2.1 1.1, 2.4
Education Men College	269	4 0	5.2			871 705	24 25	5 5 8 6	1.0		260	10	3.8 0.8	1.0		1400	48 46	3.4	0.1	
rign scnooi ≤ elementary Women	135	<u>0</u> r	5.2 2.5		0.20, 1.8	361	82	5.8 5.8	1.0	0.51, 2.1	252	11	6.7	1.4	0.53, 3.5	748	4 5	3.2 6.0	0.97	0.59, 1.6
College High school ≤ elementary	440 989 490	23 35 35 35	5 6 5 7 7 9 7 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9	1.0 0.84 1.4	0.49, 1.4 0.75, 2.7	1191 2166 1079	46 99 105	3.9 4.6 9.7	1.0 1.8 8	0.70, 1.5 1.2, 2.7	376 849 880	25 55	6.9 6.3	1.0 0.77 0.66	0.46, 1.3 0.38, 1.1	2007 4004 2449	94 191 192	4.7 4.8 7.8	1.0 0.91 1.3	0.70, 1.2 0.96, 1.7
Note. NHANES = National Health and Nutrition Examination Survey; HHANES = Hispanic Health and Nutrition Examination Survey; COPD = chronic obstructive pulmonary disease; OR = odds ratio; CI = confidence interval. •Observed number with COPD. •Odds ratios for race are adjusted for age; odds ratios for sex are adjusted for age and race; odds ratios for income (education) are adjusted for age, race, education (income). For NHANES I men, odds ratios are adjusted for age only since nor when the north MANES I men, odds ratios are adjusted for age. race, education (income). For NHANES I men, odds ratios are adjusted for age only since nor when an under a section or add stration or add stration are addivended for age. race, education (income). For NHANES I men, odds ratios are adjusted for age only since nor when an under add stration or add stration are addivended for age.	al Health SOPD. adjusted	and Nutriti for age; c	ion Exa vdds rai	mination tios for VHANE	n Survey; HH. sex are adjus S I reported C	ANES = I sted for a	Hispanic F ge and rat	Health : ce; odc	and Nutri ds ratios	= Hispanic Health and Nutrition Examination Survey; COPD = chronic obstructive pulmonary disease; OR = r age and race; odds ratios for income (education) are adjusted for age, race, education (income). For NHA Subtotals for income and education are smaller than totals because of missing data.	ion Surve ducation	ey; COPD = are adjust in totals be	= chron ted for cause c	lic obst age, ra of missi	ructive pulmor ce, education	hary dises (income)	ise; OR = .	odds r NES I	odds ratio; Cl ₌ NES I men, od	= confidence dds ratios are

Whites than for non-Whites in each survey and sex, but these differences were not statistically significant. The overall odds ratios for non-Whites relative to Whites were 0.87 (95% CI = 0.45, 1.7) for men and 0.77 (95% CI = 0.56, 1.1) for women.

Table 2 also shows chronic obstructive pulmonary disease prevalence in relation to household income, by sex and survey. Odds ratios are adjusted for age, ethnicity, and education. In NHANES II and HHANES but not in NHANES I, strong and statistically significant trends are seen with decreasing household income for both men and women. These trends also are evident when all three surveys are combined. By contrast, only weak and inconsistent trends are seen with educational level after adjusting for age, ethnicity, and income. As an exception, prevalence was elevated among women in NHANES II who did not complete high school compared with prevalence among those with a college degree (OR = 1.8; 95% CI = 1.2, 2.7).

After adjustment for age, race, and income, chronic obstructive pulmonary disease prevalence was unrelated in either sex to body size (height, weight, weight-forheight), type of home heating equipment, and presence of home air conditioning. Information on type of fuel used for cooking (available only for HHANES) revealed no association with chronic obstructive pulmonary disease risk. Similarly, we found no consistent or statistically significant associations between chronic obstructive pulmonary disease and area (urban vs rural) or population size of city or town of residence. Other characteristics examined and found unrelated to prevalence were employment in jobs with high potential for exposures to dusts and fumes, and nativity (US vs foreign born).

There were few individuals who never smoked cigarettes but who used pipes or cigars, and the chronic obstructive pulmonary disease prevalence rates were unchanged when they were excluded.

Discussion

We have examined the prevalence of chronic obstructive pulmonary disease in never-smoking participants of three US national surveys. Chronic obstructive pulmonary disease prevalence increased with age and was higher in women than in men, higher in Whites than in non-Whites, and higher in low-income than in affluent individuals. No clear or consistent associations were seen with body size, area of residence (urban vs rural), type of home heating equipment, home air conditioning, or type of occupation.

The study has several limitations. First, the findings are based on selfreported history of physician-diagnosed chronic obstructive pulmonary disease rather than on lung function measurements. Use of the latter to define the disease would have reduced the sample size since spirometry data were not available for all participants. Apart from loss of power, the use of spirometry data could introduce bias by excluding those individuals unable to provide reproducible lung function measurements.¹¹ Nevertheless some individuals with asthma, idiopathic interstitial fibrosis, or acute bronchitis may misclassify themselves as having chronic obstructive pulmonary disease, leading to an overestimation of prevalence. Moreover, differential errors by race or socioeconomic status could introduce bias.

Second, the surveys lack information on exposures to environmental tobacco smoke. Such exposures have been associated with decreased pulmonary function in nonsmoking women¹² and chronic obstructive pulmonary disease in nonsmoking adults.^{13,14} They could account for the higher prevalence observed in women compared with men (since nonsmoking women are more likely than nonsmoking men to have a smoking spouse¹⁵) and for the trend of increasing risk with decreasing income (since household income and household smoking prevalence are inversely related). Finally, sparse numbers of chronic obstructive pulmonary disease outcomes among nonsmokers and possible misclassification of occupational potential for exposure to airborne contaminants limit the study's sensitivity to detect occupational risk factors.

Despite these limitations, some conclusions seem warranted. In particular, the disease is not vanishingly rare in lifelong nonsmokers. The overall prevalence in adults aged 18 to 74 is about 4% to 6%. The observed chronic obstructive pulmonary disease prevalence of 3.7% in men is similar to the prevalences of 3.7% and 3.4%, respectively, noted in Finnish nonsmoking farmers (although these data included ex-smokers, and sex-specific rates were not provided) and a nonsmoking comparison group.¹⁶ The present prevalence rates also are similar to those noted for self-reported prevalence of chronic cough, phlegm, or wheezing (symptoms consistent with chronic obstructive pulmonary disease) in lifelong nonsmoking men and women in Finland.¹⁷ Unlike the present study, however, the Finnish study did not find an excess of symptoms in women.

The elevated chronic obstructive pulmonary disease prevalence in Hispanic women and in economically disadvantaged adults of both sexes suggests that environmental factors associated with poverty may increase risk for the disease in nonsmokers. Because most chronic obstructive pulmonary disease is due to cigarette smoking, relatively little is known about such factors. Acute respiratory infections in childhood may increase risk for disabling lung disease in adulthood.18 Increased exposure to environmental tobacco smoke, particularly in childhood, may be involved. Occupational exposures to inorganic dusts and fumes,19,20 residence in urban areas,²¹ and use of gas for cooking²² have also been implicated in elevated rates of chronic obstructive pulmonary disease. Additionally, chronic obstructive pulmonary disease has been linked to decreased levels of serum retinol.23 A small fraction of the disease in nonsmokers is due to inherited deficiency of an α_1 -proteinase inhibitor (formerly called α_1 -antitrypsin), although not all individuals who inherit this deficiency develop chronic obstructive pulmonary disease.²⁴ Further research is needed to determine any environmental factors that increase risk of both the inherited and noninherited forms of the disease. \Box

Acknowledgment

This research was supported by the US Environmental Protection Agency through contract no. CR-813945-02-1 to the Societal Institute for the Mathematical Sciences.

References

- Snider GL. Chronic obstructive pulmonary disease: a definition and implications of structural determinants of airflow obstruction for epidemiology. *Am Rev Respir Dis.* 1989;140:S3-S8.
- National Center for Health Statistics. Vital Statistics of the United States, 1988. Vol II. Mortality, Part A. Hyattsville, Md: Centers for Disease Control, National Center for Health Statistics; 1991.
- Kuller LH, Ockene JK, Townsend M, Browner W, Meilahn E, Wentworth DN. The epidemiology of pulmonary function and COPD mortality in the multiple risk factor intervention trial. *Am Rev Respir Dis.* 1989;140:S76–S81.
- Davis RM, Novotny TE. The epidemiology of cigarette smoking and its impact on chronic obstructive pulmonary disease. Am Rev Respir Dis. 1989;140:S82-S83.

Public Health Briefs

- Higgins IT. The epidemiology of chronic respiratory disease. *Prev Med.* 1973;2:14– 33.
- 6. Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service. Washington, DC: US Dept of Health, Education and Welfare, Public Health Service; 1964.
- Feinleib M, Rosenberg HM, Collins JG, Delozier JE, Pokras R, Chevarley FM. Trends in COPD morbidity and mortality in the United States. *Am Rev Respir Dis.* 1989;140:S9–S18.
- Alphabetical Index of Industries and Occupations; 1970 Census of Population. Washington, DC: US Bureau of the Census; 1971. Library of Congress Number 74-612012.
- Alphabetical Index of Industries and Occupations; 1980 Census of Population. 1st ed. Washington, DC: US, Bureau of the Census; 1982.
- Mauritsen R. EGRET. Seattle, Wash: Statistics and Epidemiology Research Corp; 1986.
- 11. Eisen EA, Robins JM, Greaves IA, Wegman DH. Selection effects of repeatability criteria applied to lung spirometry. *Am J Epidemiol.* 1984;120:734–742.

ABSTRACT

The relative contribution of walking to overall leisure-time physical activity participation rates was studied among respondents from the 45 states that participated in the 1990 Behavioral Risk Factor Surveillance System (n = 81557). The percentages of low income, unemployed, and obese persons who engaged in leisuretime physical activity (range = 51.1%to 57.7%) were substantially lower than the percentage among the total adult population (70.3%). In contrast, the prevalence of walking for exercise among these sedentary groups (range = 32.5% to 35.9%) was similar to that among the total population (35.6%). Walking appears to be an acceptable, accessible exercise activity, especially among population subgroups with a low prevalence of leisure-time physical activity. (Am J Public Health. 1995;85: 706-710)

- Brunekreef B, Fischer P, Remijn B, van der Lende R, Schouten J, Quanjer P. Indoor air pollution and its effect on pulmonary function of adult non-smoking women: III. passive smoking and pulmonary function. *Int J Epidemiol.* 1985;14:227– 230.
- 13. Robbins AS, Abbey DE, Lebowitz MD. Passive smoking and chronic respiratory disease symptoms in nonsmoking adults. *Int J Epidemiol.* 1993;22:809–817.
- Kalandidi A, Trichopoulos D, Tzannes S, Saracci R. The effect of involuntary smoking on the occurrence of chronic obstructive pulmonary disease. *Soz Praventivmed*. 1990;35:12–16.
- Helsing KJ, Sandler DP, Comstock GW, Chee E. Heart disease mortality in nonsmokers living with smokers. Am J Epidemiol. 1988;127:915-922.
- Husman K, Koskenvuo M, Kaprio J, Terho EO, Vohlonen I. Role of environment in the development of chronic bronchitis. *Eur J Respir Dis Suppl.* 1987;152:57–63.
- 17. Huhti E. Chronic bronchitis in nonsmokers—does it exist? Eur J Respir Dis Suppl. 1982;118:35–41.

- Speizer FE. The rise in chronic obstructive pulmonary disease mortality: overview and summary. *Am Rev Respir Dis.* 1989;140: S106–S107.
- 19. Becklake MR. Occupational exposures: evidence for a causal association with chronic obstructive pulmonary disease. *Am Rev Respir Dis.* 1989;140:S85–S91.
- Korn RJ, Dockery DW, Speizer FE, Ware JH, Ferris BG Jr. Occupational exposures and chronic respiratory symptoms: a population-based study. *Am Rev Respir Dis.* 1987;136:298–304.
- Holland WW, Reid DD. The urban factor in chronic bronchitis. *Lancet.* 1965;1:445– 448.
- Comstock GW, Meyer MB, Helsing KJ, Tockman MS. Respiratory effects of household exposures to tobacco smoke and gas cooking. *Am Rev Respir Dis.* 1981;124:143– 148.
- Morabia A, Menkes MS, Comstock GW, Tockman MS. Serum retinol and airway obstruction. Am J Epidemiol. 1990;132:77– 82.
- McGowan SE, Hunninghake GW. Neutrophils and emphysema. N Engl J Med. 1989;321:968–970. Editorial.

The Epidemiology of Walking for Exercise: Implications for Promoting Activity among Sedentary Groups

Paul Z. Siegel, MD, MPH, Robert M. Brackbill, PhD, MPH, and Gregory W. Heath, DHSc, MPH

Introduction

Evidence for the health benefits of regular physical activity has mounted in recent years,¹⁻⁶ and an increasing number of health-related organizations have advocated increased physical activity as a critical step toward a healthier population.⁷⁻¹⁰ For several reasons, walking is of special interest in this regard. It is becoming increasingly apparent that light-to moderate-intensity activities such as walking may provide some of the same health benefits as do more vigorous types of physical activity,¹⁰⁻¹³ along with a lower risk of injury and sudden death.¹⁴

Walking also has unique epidemiological features. First, walking is widely reported as the most popular form of physical activity.¹⁵⁻¹⁸ Second, unlike most other leisure-time activities, particularly the more vigorous ones, walking for exercise has been shown to be as prevalent among people with low family incomes as it is among people with higher incomes.^{16,19} The apparent preference for walking among persons of low socioeconomic status (SES) is of particular interest because low SES has been associated with decreased physical activity participation.¹⁹⁻²² Based on this association, national health objectives for the year 2000

Paul Z. Siegel is with the Office of Surveillance and Analysis, National Center for Chronic Disease Prevention and Health Promotion; Robert M. Brackbill is with the National Center for Prevention Services; and Gregory W. Heath is with the Epidemiology Program Office, all at the Centers for Disease Control and Prevention, Atlanta, Ga.

Requests for reprints should be sent to Paul Z. Siegel, MD, MPH, National Center for Chronic Disease Prevention and Health Promotion, Office of Surveillance and Analysis, Mailstop K30, Centers for Disease Control and Prevention, Atlanta, GA 30341-3724.

This paper was accepted on September 27, 1994.