Obstructive Lung Disease and Low Lung Function in Adults in the United States

Data From the *National Health and* Nutrition Examination Survey, 1988-1994

David M. Mannino, MD; Robert C. Gagnon, MS; Thomas L. Petty, MD; Eva Lydick, PhD

Background: Obstructive lung disease (OLD) is an important cause of morbidity and mortality in the US adult population. Potentially treatable mild cases of OLD often go undetected. This analysis determines the national estimates of reported OLD and low lung function in the US adult population.

Methods: We examined data from the Third National Health and Nutrition Examination Survey (NHANES III), a multistage probability representative sample of the US population. A total of 20050 US adults participated in NHANES III from 1988 to 1994. Our main outcome measures were low lung function (a condition determined to be present if the forced expiratory volume in 1 second-forced vital capacity ratio was less than 0.7 and the forced expiratory volume in 1 second was less than 80% of the predicted value), a physician diagnosis of OLD (chronic bronchitis, asthma, or emphysema), and respiratory symptoms.

Results: Overall a mean (SE) of 6.8% (0.3%) of the population had low lung function, and 8.5% (0.3%) of the population reported OLD. Obstructive lung disease (age-adjusted to study population) was currently reported among 12.5% (0.7%) of current smokers, 9.4% (0.6%) of former smokers, 3.1% (1.1%) of pipe or cigar smokers. and 5.8% (0.4%) of never smokers. Surprisingly, 63.3% (0.2%) of the subjects with documented low lung function had no prior or current reported diagnosis of any OLD.

Conclusions: This study demonstrates that OLD is present in a substantive number of US adults. In addition, many US adults have low lung function but no reported OLD diagnosis, which may indicate the presence of undiagnosed lung disease.

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From the Air Pollution and Respiratory Health Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Ga (Dr Mannino); SmithKline Beecham Pharmaceuticals, Collegeville, Pa (Drs Gagnon and Lydick); and the University of Colorado Health Sciences Center, Denver, Colo (Dr Petty).

OBSTRUCTIVE lung diseases (OLDs), which include chronic bronchitis, emphysema, and asthma, are the fourth most common cause of death in the United States and accounted for more than 109000 deaths in 1997.[1] Obstructive lung disease is the only major disease among the top 5 causes of death that is rising in prevalence and mortality.[1] It is now estimated that nearly 16 million people in the United States have chronic bronchitis and emphysema, which is commonly referred to as chronic obstructive pulmonary disease (COPD).[3] Costs of hospitalizations, physician visits, and consumption of health care resources for COPD were almost $15 billion in 1993.[4] In addition, asthma may affect as many as 14.6 million people in the United States[3,5] with associated direct medical costs of $6.0 billion in 1993.[6]

Because of the increase in prevalence and mortality of OLD, and the medical costs associated with them, it is important to identify patients and to treat them before they reach the symptomatic and costly stages of dii. The Lung Health Study showed that when patients with mild to moderate OLD quit smoking, their lung function declined only slightly over the next 5 years.[7] In contrast, similar patients who continued to smoke had rapid rates of decline in lung function. In addition, the Lung Health Study showed a high incidence of mortality related to lung cancer, heart attack, and stroke in subjects with only mild to moderate airflow obstruction.[7]

Because a high proportion of OLD has eluded diagnosis, it is impossible to know the true prevalence of these diseases in the United States today. To estimate the magnitude of OLD in the United States, we analyzed data from the Third National Health and Nutrition Examination Survey (NHANES III)[8] in an effort to determine how much of the US population might have low levels of lung function, OLD, and respiratory symptoms.

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SUBJECTS AND METHODS

STUDY POPULATION

The NHANES III was conducted from 1988 to 1994 by the National Center for Health Statistics of the Centers for Disease Control and Prevention, Atlanta, Ga.[8] In this study a stratified multistage clustered probability design was used to select a representative sample of the US population, yielding results that can be extrapolated to the entire US population. Study participants completed extensive questionnaires in the household and a comprehensive physical examination, including pulmonary function testing, either in the household or at a specially equipped mobile examination center. A total of 81 sites were included in the final sample. The study was approved by the National Center for Health Statistics Institutional Review Board.

SUBJECTS

Our study sample was limited to adults aged 17 years and older who classified themselves as whites or blacks, had pulmonary function testing performed in either the home or the mobile examination center, and had complete data on their race, smoking status, height, and presence of respiratory symptoms. Of the 20050 adult study participants, 543 were not of white or black race, 3355 did not have pulmonary function testing done, 8 were missing data on smoking status, race, or height, and 56 were missing dam on 1 or more respiratory symptom. In addition, we excluded 4 women from the analysis who were current cigar or pipe smokers. After the exclusions, we had data from 16084 subjects available for our main analysis.

VARIABLE DEFINITION

The race of the participants was classified as either white or black and was determined by self-report on the questionnaire. We defined subjects as being current smokers, former smokers, pipe or cigar smokers, or never smokers based on their responses to series of questions. One had to have smoked more than 100 cigarettes to qualii as a former or current smoker and could not be currently smoking cigarettes to qualify as a pipe or cigar smoker. Former smokers were asked how old they were when they last smoked cigarettes. Subjects were asked "Has a doctor ever told you that you asthma?" and similar questions about chronic bronchitis and emphysema. Subjectswith a positive response to the question about asthma or chronic bronchitis were also asked 'Do you still have asthma?" and "Do you still have chronic bronchitis?" We considered subjects who responded that they currently had asthma or bronchitis, or that they had ever had a diagnosis of emphysema, to have a current diagnosis d OLD. We considered subjects with a positive response to a previous diagnosis of either chronic bronchitis or asthma, but a negative response to current disease, to have a past diagnosis of OLD. We classified subjects as having a symptom if they gave a positive response to the following questions involving specific symptoms (cough. phlegm, wheezing, and dyspnea): "Do you usually cough on most days for 3 consecutive months or more during the year?" "Do you bring up phlegm on most days for 3 consecutive months or more during the year?" 'Have you had wheezing orwhistlinginyourchcstatany time in the past 12 months?" and "Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?" If a subject had a positive response to any of these 4 symptoms, we considered that subject to have a respiratory symptom.

For most analyses, we stratified subjects into 6 age strata; 17 to 24 years, 25 to 44 years, 45 to 64 years, 65 to 74 years, 75 to 84 years, and 85 years and older. For use in logistic regressions we classified subjects as having or not having cardiovascular disease (positive response to physician-diagnosed stroke, myocardial infarction, or congestive heart failure); obesity (body mass index [BMI]>28, calculated as the weight in kilograms divided by the square of the height in meters); inactivity (based on self report as being less active than peers); and low socioeconomic status (based on family income below poverty level).

PULMONARY FUNCTION DATA

Using either a dry rolling seal spirometer in the mobile examination center or a portable spirometer in the home examination. spirometry was conducted on the examinees. Procedures for testing were based on the 1987 American Thoracic Society recommendations.' To obtain acceptable protocol curves, examinees performed 5 to 8 forced expirations. We used published prediction equations for forced expiratory volume in 1 second (FEV_1) to calculate the predicted FEV_1 for whites and blacks, stratified by sex.[10] We then determined the value of the FEV, (as a percentage of the predicted value) for each subject. We defined subjects with an FEV_1–forced vital capacity (FVC) ratio of less than 0.70 and an FEV_1 less than 88% of their predicted value as having low lung function, and further divided this group into subjects with an FEV_1 of 58% or more and those with less than 58% of their predicted value, corresponding to people with stage 1 vs stage 2 or 3 OLD."

ANALYSIS

We calculated all estimates using thesampling weight to represent adults aged 17 years and older in the United States. The purpose of these sampling weight calculations was to adjust for unequal probabilities of selection and to account for nonresponse. They were poststratified to the US population as estimated by the Bureau of the Census. For analyses, we used both SAS and SUDAAN. a program that adjusts for the complex sample design when calculating variance estimates.[12] Most of our analyses stratified the data by race and sex, with further stratification by either smoking status or age. We also present data stratified by reported lung dii and level of lung function. In these strata and substrata. we determined the estimated national population, the mean FEV_1 as percent predicted, the mean FEV_1-FVC ratio, and the number and age-adjusted percentage d the population with low lung function. We used the 6 age classes noted above and the overall age distribution of the study participants to age-adjust our results. In addition, we determined the number and age-adjusted percentage of the population with past or current OLD and the age-adjusted percentage d the population with cough, phlegm, wheezing, dyspnea, or any respiratory symptom. We also determined the age-adjusted percentage of the population stratified by race, sex. and smoking status with low lung function but with no current diagnosis of any OLD. Finally, we modeled, using logistic regression, factors predicting subjects who were unable to perform pulmonary function testing, subjects with low lung function, and subjects with low lung function yet no reported OLD.

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Table 1. Characteristics of Participants and Estimated National Population, Stratified by Race, Sex, and Smoking Status*

Race/ Sex	Smoking Status	No. of Participants	Estimated Population	Age, y	FEV_1 Percent Predicted	FEV_1-FVC Ratio	No. of Participants With OLD in Past	Participants With OLD in Past, %†	No. of Participants With OLD Now	Participants With OLD Now, %†
Black/F	Current smoker	686	2 954 000	39.0 (0.4)	96.0 (0.8)	0.80 (0.005)	133 000	4.4	300 000	10.7
	Former smoker	308	1 281 000	48.4 (1.1)	89.2 (1.2)	0.80 (0.007)	70 000	5.0	123 000	9.4
	Never smoker	1650	6 823 000	38.0 (0.5)	99.0 (0.4)	0.84 (0.003)	231 000	3.7	479 000	7.1
Black/M	Current smoker	805	3 337 000	39.5 (0.5)	95.7 (0.5)	0.79 (0.004)	138 000	1.8	215 000	6.6
	Former smoker	417	1 559 000	48.6 (1.0)	96.1 (1.1)	0.77 (0.006)	28 000	3.3	105 000	5.9
	Pipe or cigar smoker	75	260 000	54.5 (2.1)	88.2 (2.4)	0.74 (0.016)	10 000	1.3	21 000	8.1
	Never smoker	865	3 771 000	32.1 (0.5)	97.7 (0.6)	0.83 (0.003)	97 000	3.6	223 000	5.8
White/F	Current smoker	1159	19 779 000	38.7 (0.6)	90.9 (0.7)	0.78 (0.003)	1 275 000	6.3	2 727 000	14.7
	Former smoker	1106	16 260 000	50.0 (1.0)	94.0 (0.8)	0.77 (0.004)	911 000	5.6	2 002 000	12.1
	Never smoker	3652	41 120 000	44.5 (0.7)	97.8 (0.4)	0.81 (0.002)	1 461 000	3.5	2 865 000	6.8
White/M	Current smoker	1429	22 149 000	38.3 (0.5)	91.5 (0.5)	0.76 (0.003)	1 094 000	4.9	2 217 000	11.2
	Former smoker	1834	21 449 000	51.6 (0.6)	93.8 (0.7)	0.74 (0.004)	924 000	4.5	1 961 000	7.9
	Pipe or cigar smoker	165	2 627 000	50.8 (1.4)	97.1 (1.2)	0.75 (0.008)	54 000	1.3	92 000	2.7
	Never smoker	1933	26 016 000	37.4 (0.6)	98.3 (0.5)	0.80 (0.002)	854 000	2.9	1 084 000	4.6
Total		16 084	169 352 000	42.8 (0.4)	95.3 (0.3)	0.79 (0.002)	7 280 000	4.3	14 354 000	8.5

*FEV_1 indicates the mean forced expiratory volume in 1 second; FVC, forced vital capacity; OLD, obstructive lung disease. Data an from the Third National Health and Nutrition Examination Survey, 1988-94 and, unless otherwise indicated, are mean (SE).
†Age adjusted to all study participants.

RESULTS

Our final data set contained 16084 subjects representing an estimated 1693 million adults in the United States. Relative SEs for the data presented are less than 10%. except for data on pipe and cigar smokers, where the RSEs are less than 35%. Among the population, an estimated 14.3 million (8.5%) had current OLD, and another 7.3 million (4.3%) had OLD in the past, but not currently (Table 1). The proportion of the population with past or current OLD varied by sex, race, and smoking status, with women reporting more disease than men, whites reporting more disease than blacks, and current or former smokers reporting more disease than never smokers (Table 1). Obstructive lung disease was present among 12.5% of current smokers, 9.4% of former smokers, 3.1% of pipe or cigar smokers, and 5.8% of never smokers (all age-adjusted to study population). Former smokers were, on average, older than never and current smokers (Table 1). Mean level of lung function, either as the FEV_1-FVC ratio or FEV_1 percent predicted, was always lower among current smokers, former smokers, and pipe or cigar smokers, compared with never smokers (Table 1). Among whites, the reported COPD component of OLD generally increased with age, while among blacks this trend was less apparent (Figure 1).

Subjects frequently reported more than 1 OLD. For example, 25.0% of subjects with current bronchitis reported that they had current asthma, and 34.3% of subjects with current asthma reported that they had current chronic bronchitis. Similarly, 19.4% of the subjects with emphysema reported that they had current asthma, and 25.0% of the subjects with emphysema reported that they had current chronic bronchitis. Among subjects who reported having at some point been diagnosed with asthma, 63.7% reported current asthma, and 58.3% of subjects who reported having been at some point diagnosed with chronic bronchitis also reported a current diagnosis.

Pulmonary function testing was not obtained on 3355 of the original 20050 subjects. The significant predictors of not having testing done were the presence of cardiovascular disease (odds ratio [OR], 1.4; 95% confidence interval [CI], 1.2-1.7) and age (OR, 1.9; 95% CI, 1.4-2.5 for those aged 65-74 years; OR, 2.9; 95% CI, 2.3-3.8 for those aged 75-84 years; and OR, 5.3; 95% CI, 3.8-7.3 for those aged 85 years and older, compared with those aged 17-24 years). Overall, 6.8% of the population, or an estimated 11.5 million people had low lung function (Table 2). An additional 7.2% of the population had an FEV_1-FVC ratio of less than 0.7 but an FEV_1 greater than 80% of the predicted value. Significant predictors of low lung function included current smoking (OR, 4.3; 95% CI, 3.2-5.7), former smoking (OR, 2.0; 95% CI, 1.5-2.5). pipe or cigar smoking (OR, 2.7; 95% CI, 1.4-5.0), current OLD (OR, 5.4; 95% CI, 4.1-7.0), prior OLD (OR, 1.8; 95% CI, 1.0-3.0). and age (ORs increased from 2.1 for those aged 25 to 44 years to 21.0 for those aged 85 years and older, compared with 17- to 24-year-olds, all CIs significant). Moderate to severe lung obstruction (FEV_1 less than 50% of the predicted value and an FEV_1-FVC ratio of less than 0.7) was more common among current and former smokers and among people aged 45 years and older (Figure 2 and Figure 3). Overall, 1.5% of the population, or an estimated 2.6 million people, had an FEV_1 of less than 50% of the predicted value, including an estimated 900000 people with an FEV_1 of less than 35% of the predicted value. Lower levels of lung function were associated with higher levels of reported symptoms (Table 3).

Current and former smokers reported respiratory symptoms more frequently than never smokers (Table

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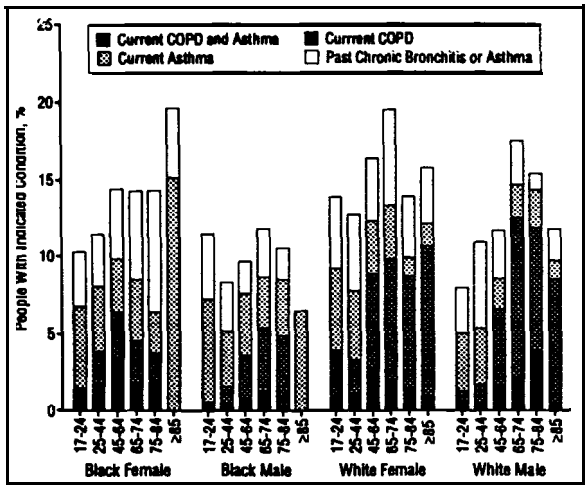

Figure 1. *The age-specific percentage of people (numbers are age ranges in years), stratified by race and sex, with current chronic obstructive pulmonary disease (COPD) (chronic bronchitis and pneumonia) and asthma, current COPD, current asthma, and past chronic bronchitis or asthma From Me Third National Health and Nutrition Examination Survey, 1988-94.*[6]

2). Reporting of symptoms varied across disease strata (Table 3). Among subjects with low lung function, 66.1% reported at least 1 respiratory symptom (Table 3), compared with 34.4% of subjects with normal pulmonary function. There was a great deal of overlap between subjects reporting respiratory symptoms, those with low lung function, and those with current or prior OLD (**Figure** 4, Table 3).

Overall, 63.3% of the population with low lung function did not have a current diagnosis of OLD. Even among subjects with moderate to severe pulmonary impairment, as indicated by an FEV_1 of less than 50% of the predicted value. 44.8% did not have a current diagnosis of OLD. Across sex, race, and smoking categories this proportion ranged from 40% to 88% (**Figure 5**). People with a past diagnosis of obstructive lung disease accounted for a small proportion of this group of subjects not currently diagnosed (Figure 5). The only significant predictors of low lung function without a current diagnosis of OLD were current smoking (OR. 1.6; 95% CI, 1.0-2.5), inactivity (OR, 0.6; 95% CI, 0.4-0.9), and cardiovascular disease (OR, 0.5; 95% CI, O-3-0.7).

COMMENT

These data show that low lung function and OLD occur commonly in a nationally representative sample of the US population. Low lung function is found in more than 10% of the population over age 45 years, but is not associated with reported current OLD 63.3% of the time. This finding is important, because when these patients can be identified in early and less symptomatic stages of disease, interventions can be expected to alter the course and prognosis of disease.[7,13,14] Clearly. smoking cessation is the most important feature in management and is apt to be beneficial when it is accomplished at an early age with only mild evidence of airflow obstruction. While the finding of low lung function does not by itself diagnose OLD, its presence should alert clinicians to the possibility of OLD being present, resulting in subsequent evaluations and interventions.

These results confirm the findings of many previous studies, which show associations between cigarette smoking and pulmonary effects, including low lung function and respiratory symptoms.[13,15,16] A somewhat surprising finding, though. was that cigar or pipe smokers had a higher prevalence of low lung function than never smokers, along with more reported respiratory symptoms, a finding that runs counter to studies finding fewer smoking-related health effects in pipe or cigar smokers.[17] Our study suggests that pipe and cigar smokers are getting enough doses of smoke to cause lung damage, but our findings may also reflect, in part, former cigarette smoking (in our study, 69.3% of pipe and cigar smokers were former cigarette smokers)." In addition, our finding of decreased lung function in pipe and cigar smokers demands intervening in this group.

The results of our analysis also confirmed previously known associations between aging and increased airway obstruction." The prevalence of low lung function increased with increasing age (Figure 2) except in the oldest group, which may be related to either differential mortality or inability to do pulmonary function testing. While it is certainly known that the individuals with low lung function die young,[13,20,21] our data also suggest that people over age 45 years may have significantly decreased lung function that remains undiagnosed (Figure 2 and Figure 5). The patterns we found of reported OLD also were consistent with reports of COPD increasing with age' (Figure 1). The racial differences we observed, of generally lower levels of reported COPD in blacks compared with whites, is consistent with other observations?

There have been numerous, but much smaller regional prevalence studies done in the United States, most notably in Berlin, NH[22]; Tecumseh, Mich[23]; and Glenwood Springs, Colo.[24] These studies used different degrees of airflow obstruction and had different definitions of the diseases characterized by airflow obstruction. In Berlin, NH, the prevalence of all chronic nonspecific respiratory diseases ranged from 15.4% to 39.1% among men and from 15.2% to 19.8% among women, and low levels of lung function were found among 3.1% to 21.7% of men and 6.7% to 13.9% of women.[22] In Tecumseh, Mich, about 14% of adult men and 8% of adult women had chronic bronchitis, obstructive airway disease, or both.[23] In Glenwood Springs, Colo. about 13% of adult men and 10% of adult women had chronic bronchitis and 13% and 4%, respectively, had low lung function." The above-noted findings can be contrasted with our own findings of 8.5% of adults reporting current OLD and 6.8% having low lung function.

Our data also showed a large degree of overlap between asthma and COPD. Subjects with both diseases had lower lung function and more respiratory symptoms than subjects with just one or the other disease (Table 3). Asthma is a potentially reversible disease when diagnosed and treated early in its course with bronchoactive and anti-inflammatory medications.[25,26] By contrast, COPD

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Table 2. Characteristics of Participants With Low Lung Function, Stratified by Race, Sex, and Smoking Status*

Race/Sex	Smoking Status	Estimated Population	No. of Participants With Low Lung Function	Low Lung Function, %†	Cough, %†	Phlegm, %†	Wheezing, %†	Shortness of Breath, %†	No. of Participants With Any Symptom	Any Symptom, %†
Black/F	Current smoker	2 854 000	184 000	8.6	8.0	9.0	18.9	33.1	1 269 000	43.5
	Former smoker	1 281 000	69 000	4.3	2.8	6.6	14.3	28.6	472 000	36.7
	Never smoker	6 823 000	160 000	2.6	4.5	5.0	10.5	25.8	2 000 000	31.8
Black/M	Current smoker	3 337 000	273 000	11.6	10.9	9.7	19.0	21.7	1 190 000	38.1
	Former smoker	1 559 000	150 000	8.4	4.4	5.6	16.8	14.2	409 000	24.3
	Pipe or cigar smoker	260 000	32 000	7.1	6.4	3.7	15.0	28.9	124 000	40.2
	Never smoker	3 771 000	116 000	4.8	4.1	4.3	8.8	14.5	701 000	21.5
White/F	Current smoker	19 770 000	2 187 000	13.6	20.6	16.0	31.1	38.7	11 360 000	57.4
	Former smoker	16 260 000	1 520 000	6.8	6.5	4.1	15.3	25.7	6 240 000	35.5
	Never smoker	41 120 000	1 396 000	3.1	5.0	4.3	11.7	20.4	13 250 000	31.3
White/M	Current smoker	22 140 000	2 267 000	14.2	24.0	20.5	32.2	31.7	12 150 000	57.6
	Former smoker	21 440 000	2 195 000	6.9	4.7	6.1	15.8	17.3	7 003 000	29.8
	Pipe or cigar smoker	2 627 000	291 000	9.9	13.3	17.5	16.7	9.8	749 000	37.6
	Never smoker	26 010 000	690 000	3.3	4.0	5.3	9.4	10.6	5 355 000	22.8
Total		169 352 000	11 520 000	6.8	9.3	8.3	17.6	22.9	62 272 000	36.8

* Low lung function is a forced expiratory volume (FEV_1) in 1 second–forced vital capacity (FVC) ratio of less then 0.70 and an FEV_1 of less than 80% of the predicted value. AU relative SEs am less than 35%. Date from the Third National Health and Nutrition Examination Survey, 1988-94.[8]
† Age adjusted to all study participants.

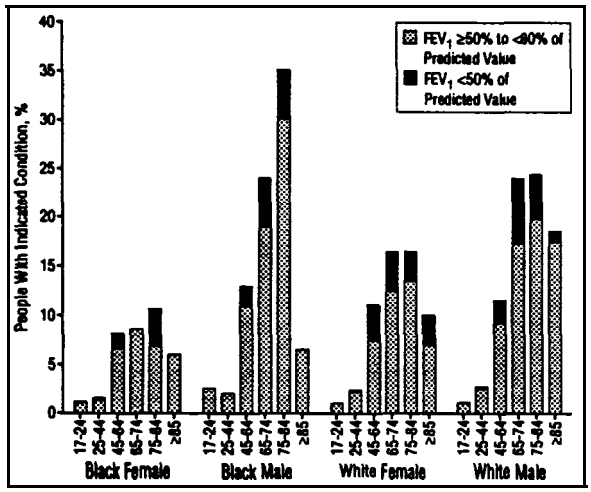

Figure 2. The age-specific percentage of people (numbers an, age ranges in years), stratified by race and sex, with a forced expiratory volume in 1 second (FEV_1)–forced vital capacity (FVC) ratio of less than 0.70 and an FEV_1 50% to 80% or baa then 50% of the expected value. From the Third National Health and Nutrition Examination Survey, 1988-94.[8]

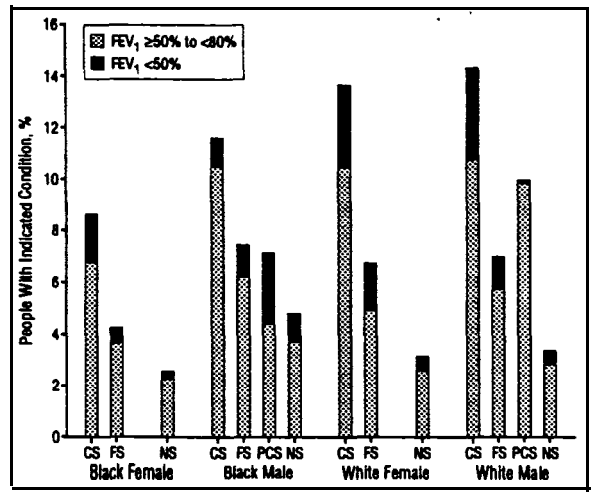

Figure 3. The age-adjusted percentage of people, stratified by race, sex, and smoking status (current smokers [CS], former smokers [FS], pipe or cigar smokers [PCS] and never smokers [NS]), with a forced expiratory volume in 1 second (FEV_1)–forced vital capacity (FVC) ratio of less than 0.70 and an FEV_1 50% to 80% or less than 50% of the expected value. From the Third National Health and Nutrition Examination Survey, 1988-94.[8]

tends to be a progressive disease, with relentless decline in lung function and resultant morbidity and mortality beginning in the fifth or sixth decade of life.[25] Preventing or forestalling this burden of disease may be possible through aggressive smoking cessation and, perhaps, medications that may improve baseline lung function. The search for other drugs to modulate the inflammatory processes incident to the pathogenesis of COPD is also ongoing.[27-34]

This survey is subject to several limitations that affect the interpretation of these results. The entire medical history, including the previous and current diagnoses, the respiratory symptoms, and the smoking histories, were entirely self-reported. It is possible that subjects may have underreported or overreported in any of these categories. For instance, we found that only 33.9% and 25.6% of subjects with COPD reported cough and phlegm, respectively. While the criteria for a positive response to these questions were stringent, this may indicate that either symptom or disease reporting was inaccurate. Another limitation was that current smokers who either smoked irregularly or had just stopped smoking could be misclassified as former smokers. In our analyses, however, many of our findings were consistent with known relationships between respiratory disease, respiratory symptoms, and lung function, suggesting that any

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Table 3. Characteristics of Participants With Low Lung Function, Stratified by Reported Lung Disease and Level of Lung Function*

Characteristic	Estimated Population	No. of Participants With Low Lung Function	Low Lung Function, %	Cough, %	Phlegm, %	Wheezing, %	Shortness of Breath, %	No. of Participants With Any Symptom	Any Symptom, %
Reported Lung Disease Category									
COPD† and asthma	2 302 000	939 000	29.9	50.8	46.7	80.6	66.0	2 221 000	94.5
COPD only	5 989 000	1 791 000	17.4	33.9	25.6	57.8	48.3	5 110 000	83.8
Asthma only	6 073 000	1 002 000	19.2	13.9	11.1	66.9	45.5	4 928 000	80.6
Chronic bronchitis or asthma in the past	7 280 000	584 000	8.9	13.7	11.2	29.5	32.7	3 740 000	52.2
No COPD or Asthma	147 700 000	7 204 000	5.1	7.3	6.9	12.9	19.6	46 280 000	31.6
Lung Function Category									
FEV_1/FVC <0.70 and FEV_1 <50% of predicted value	2 456 000	2 456 000‡	100.0‡	41.2	32.7	63.6	65.4	2 175 000	79.0
FEV_1/FVC <0.70 and FEV_1 50% to 85% of predicted value	9 066 000	9 066 000‡	100.0‡	14.6	15.4	42.8	40.0	5 492 000	60.1
FEV_1/FVC <0.70 and FEV_1 ≥80% of predicted value	12 170 000	0‡	0.0‡	17.5	12.2	28.8	25.6	5 681 000	50.8
FEV_1/FVC >0.70	145 600 000	0‡	0.0‡	7.9	7.2	15.2	21.6	48 940 000	34.4
Total§	169 352 000	11 521 000	6.8	9.3	8.3	17.6	22.9	62 272 000	36.8

*Low lung function is a forced expiratory volume (FEV_1) in 1 sewn&forced vital capacity (FVC) ratio of less than 0.70 and an FEV_1 of less than 80% of the predicted value. All relative SEs are less than 35%. Unless otherwise indicated, all data are age adjusted to all study participants. Data from the Third National Health and Nutrition Examination Survey,1988-94.[8]

†COPD indicates chronic obstructive pulmonary disease.

‡By definition.

§Totals may not be exact because of rounding.

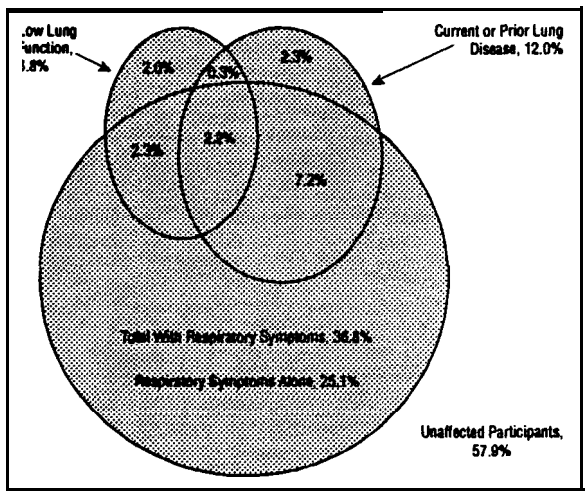

Figure 4. A proportional Venn diagram of the prevalence of low lung function (forced expiratory volume in one second [FEV_1]–forced vital capacity [FVC] ratio of less then 0.70 and an FEV_1 less than 80% of the expected value), current or pest diagnosis of obstructive lung disease, and the presence of 1 or mom respiratory symptoms From the Third National Health and Nutrition Examination Survey, 1988-94.[8]

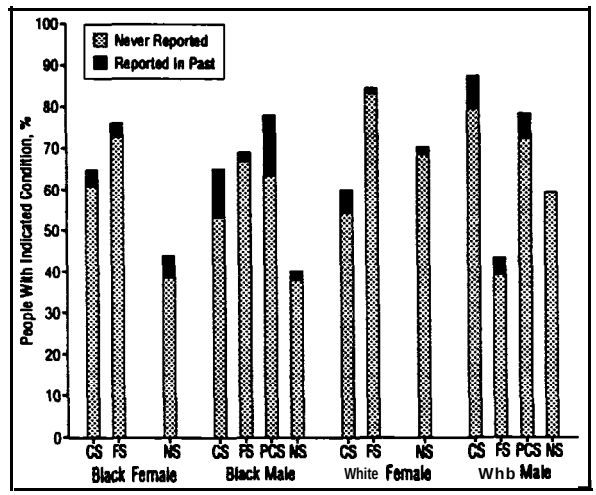

Figure 5. The age-adjusted percentage of people with a forced expiratory volume in 1 second (FEV_1)–forced vital capacity (FVC) ratio of less than 0.70 and an less than 80% of the expected value and either no diagnosis or a past diagnosis of obstructive lung disease, stratified by race, sex, and smoking status (current smokers [CS], former smokers [FS], pipe or cigar smokers [PCS], and never smokers [NS]). From the Third National Health and Nutrition Examination Survey,1988-94.[8]

misreporting bias potentially present did not have a large effect on the results.

Another potential bias of this study design is that not everyone completed pulmonary function testing. It is possible that subjects unable to do pulmonary function testing were also more likely to have low levels of lung function, thus resulting in an underestimate in the burden of the prevalence of low lung function in the United States. Selection was limited to a noninstitutionalized, nonmilitary population, so it was not truly randomized to a general population. Only patients who were ambulatory and able to participate were studied, which might have created a selection bias.

Pulmonary function testing may provide an interventional opportunity in the United States. Since early intervention may alter the course and prognosis of OLD, it is important that all clinicians who encounter smokers and any patient with respiratory symptoms, particularly those over age 45 years, be able to make an accurate diagnosis. Previous studies have shown, though, that

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symptoms alone are not adequate to diagnose COPD,[35,36] and that spirometry may need to be more widely used to detect early disease.'

The National Lung Health Education Program is a new health care initiative aimed at involving primary care physicians in the early identification and treatment of obstructive lung disease.' The National Lung Health Education Program is promoting the widespread use of simple office spirometry to measure the FEV_1, FVC, and FEV_1-FVC ratio. The present study demonstrates that a significant proportion of the US population older than 45 years has low lung function, which may represent unrecognized and potentially treatable OLD. Spirometry remains the most efficient means of identifying and treating this population.

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Reprints: David M. *Mannino*, MD, National Center *for Environmental* Health, Centers *for* Disease Control and Prevention, 1600 Clifton Rd, MS E-17, Atlanta, GA *30341*-3724 (e-mail: *dmm6@cdc.gov*).

REFERENCES

1. Hoyert DL. Kochan KD, Murphy SL. *Death: Final Data for 1997 National Vital Statistics Reports 1999;* Hyattsville, Md: National Center for Health Statistics; 1999. US Dept ol Health and Human-publication PHS 99-1120.
2. Centers for Disease Control and Prevention. Mortality patterns: preliminary data, United States, 1996. *MMWR Morb Mortal Wkly Rep.* 1997;46:941-944.
3. Adams PF, Hendershoot GE, Marano MA. *Currente Estimates From the National Health Interview Survey, 1996.* Hyattsville, Md: National Canter for Health Statistics; 1999. US Dept of Health and Human Services publication PHS 99-1528.
4. The National Lung Health Education Pmgram (NLHEP). Strategies in preserving lung health and preventing COPD and associated diseases. *Chest.* 1998;113 (suppl):123S-163S.
5. Mannino DM, Homa OM. Pertowski CA, et al. Surveillance for asthma: United States, 1960-1995. *MMWR Morb Mortal Wkly Rep.* 1998;47:1-27.
6. Webs KB, Gergen PJ, Hodgson TA. An economic evaluation of asthma in the United States. *N Engl J Med.* 1992;326:862-866.
7. Anthonisen NR, Connett JE. Kiby JP. et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1: the Lung Health Study. *JAMA.* 1994;272:1497-1505.
a. National Center tor Health Statistics. *Plan* and *operation of the* mid *National Health and Nutrition Examination Survey, 1988-94;* 1994. US Dept of Health and Human Services publication PHS 94-1308.
9. American Thoracic Society. Standardization of spirometry—1987 update: statement of the American Thoracic Society. *Am Rev Respir Dis.* 1987;136:1285-1298.
10. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general US. population. Am *J Respir Crit Care Med.* 1999;159: 179-187.
11. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 1995;152 (suppl):S77-S121.
12. Shah BV, Barnwell BG. Bbbr GS. *SUDAAN User's Manual, Release 7.0.* Research Triangle Park, NC; Research Triangle Institute; 1996.
13. Peto R, Speizer FE, Cochrane AL, et al. The relevance in adults of air-flow obstruction, but not of mucus hypersecretion, to mortality from chmnic lung disease: results from 20 years of prospective observation. *Am Rev Respir Dis.* 1983: 128:491-500.
14. Higgins M. Epidemiology of COPD: state of the art. Chest 1984;85(suppl):3S-8S.
15. Burrows B, Lebowitz MD, Camilli AE, Knudson RJ. Longitudinal changes in forced expiratory volume in one second in adults: methodologic considerations and findings in healthy nonsmokers. Am *Rev Respir Dis.* 1986;133:974-980.
18. Rennard SI. COPD: overview of definitions, epidemiology, and factors influencing its development *Chest.* 1998;113(suppl):235S-241S.
17. Wald NJ, Watt HC. Prospective study 01 effect of switching from cigarettes to pipes or cigars on mortality from thrw smoking-related diseases. *BMJ.* 1997: 314:1860-1863.
18. Nelson DE, Davis RM, Chrismon JH, Giovino GA. Pipe smoking in the United States, 1965-1991: prevalence and attributable mortality. *Prev Med.* 1996;25:91-99.
19. Wise RA. Changing smoking patterns and mortality from chronic obstructive pulmonary disease. *Prev Med.* 1997;26:418-421.
20. Weiss ST. Segal MR. Sparrow D, Wager C. Relation of FEV1 and peripheral Mood leukocyte count *to total* mortality: the *normative* aging *study. Am J Epidemiol.* 1995;142:493-498.
21. Patty TL, Pin OJ. Dick NP, Hudson LO, Walker SH. Follow-up evaluation 01 a prevalence study for chmnic bronchitis and chronic airway obstruction. Am *Rev Respir Dis.* 1976;114:881-890.
22. Ferris BG Jr, Anderson 00. The prevalence of chmnic respiratory disease in a New Hampshire town. *Am Rev Respir Dis.* 1962;86:165-177.
23. Hiiins MW, Keller JB. Landii JR. et al. Rii of chronic obstructive pulmonary disease: collaborative assessment of the valiiity of the Tecumseh index of risk. *Am Rev Respir Dis.* 1984;130:380-385.
24. Mueller RE, Keble OL. Plummer J. Walker SH. The prevalence of chronic bronchitis, chronic airway obstruction, and respiratory symptoms in a Colorado city. Am *Rev Respir Dis.* 1971;103:209-228.
25. Burrows B, Bloom JW. Traver GA, Cline MG. The COUM and prognosis 01 different forms of chronic airways obstruction in a sample from the general population. *N Engl J Med.* 1987;317:1309-1314.
26. Burrows B. The course and prognosis of different types of chmnic airflow limbtation in a general population sample from Arizona: comparison with the Chiigo 'COW' series. Am *Rev Respir Dis.* 1989;140(suppl):S92-S94.
27. Anthonisen NR. OM-88V for COPD. *Am J Respir Crit Care Med.* 1997;156:1713-1714.
28. Collet JP, Shapiro P, Ernst P, Renzi T, Ducruet T, Robinson A. Effects of an immunostimulating agent on acute exacerbations and hospiiiis in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Cam Med.* 1997: 156:1719-1724.
29. Tomee JF, Koeter GH. Hiemstra PS. Kautlman HF. Secretory leukoprotease inhibitor: a native antimicrobial protein presenting a new therapeutic option? *Thorax.* 1998;53:114-116.
30. van Schayck CP, van Grunsven PM, Dekhuijzen PN. Do patients with COPD benefit from treatment with inhaled corticosteroids? *Eur Respir J.* 1996;9:1969-1972.
31. Dompeling E, van Schayck CP, van Grunsven PM, et al. Slowing the deterioration of asthma and chronic obstructive oulmonarv disease observed durino bronchodilator therapy by adding inhaled corticosteroids: a 4-year prospective study. *Ann Intern Med.* 1993;118:770-778.
32. Kerstjens HA, Brand PL, Hughes MD, et al. A comparison of bronchodilator therapy with or without inhaled corticustaroii therapy for obstructive airways disease. N *Engl J Med.* 1992;327:1413-1419.
33. Confalonieri M, Mainardi E. Della Porta R, et al. Inhaled corticosteroids reduce neutrophilic bronchial inflammation in patients with chronic obstructiw pulmonary disease. *Thorax.* 1998;53:583-585.
34. Shahar E. Folsom AR. Melnick SL, et al. Dietary n-3 polyunsaturated fatty acids and smoking-related chronic obstructive pulmonary disease. *N Engl J Med.* 1994; 331:228-233.
35. Mannino OM. Etzel RA, Flanders WO. 00 the medical history and physical examination predict *low* lung *function? Arch Intern Med.* 1993;153:1892-1897.
38. Badgett RG, Tanaka OJ, Hunt OK, et al. Can moderate chmnic obstructive pulmonary disease be diagnosed by historical and physical findings alone? Am J Med. 1993;94:188-196.

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Reprints: David M. Mannino, MD, National *Center for* Environmental *Health, Centersfor Disease Control and Prevention, 1600 Clifton Rd, MS E-l 7, Atlanta, GA 30341-3724* (e-mail: *dmm6@cdc.gov*).

REFERENCES

1. Hoyert DL, Kochan KD, Murphy SL. *Death: Final Data for 1997 National Vital Statistics Reports 1999;* Hyattsville, Md: National Center for Health Statistics; 1999. US Dept of Health and Human Services publication PHS 991126.
2. Centers for Disease Control and Prevention. Mortality patterns: preliminary data, United States, 1996. *MMWR Morb Mortal Wkly Rep.* 1997;46:941-944.
3. Adams PF, Hendershoot GE, Marano MA. *Currente Estimates From the National Health Interview Survey, 1996.* Hyattsville, Md: National Center for Health Statistics; 1999. US Dept of Health and Human Services publication PHS 99-1528.
4. The National Lung Health Education Program (NLHEP). Strategies in preserving lung health and preventing COPD and associated diseases. *Chest.* 1998;113 (suppl):123S-163S.
5. Mambo DM, Homa OM. Pertowski CA, et al. Surveillance for asthma: United saw, 1960-1995. *MMWR Morb Mortal Wkly Rep.* 1998;47:1-27.
6. Wabs KB, Gergen PJ. Hodgson TA. An economic evaluation 01 asthma in the United States. *N Engl J Med.* 1992;326:862-866.
7. Anthonisen NR. Connett JE, Kiley JP. et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1: the Lung Health Study. *JAMA.* 1994;272:1497-1505.
6. National Center for Health Statistics. *Plan* and *operation of the Third* Nation& *Health and Nutrition Examination Survey, 1988-94;* 1994. US Dept of Health and Human Services publication PHS 94-1308.
9. American Thoracic Society. Standardization of spirometry—1987 update: statement of the American Thoracic Society. *Am Rev Respir Dis.* 1987;136:1285-1298.
10. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference value from a sample cd the general U.S. population. Am *J Respir Crit Care Med.* 1999;159: 179-187.
11. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Am *J Respir Crit Care Med.* 1995;152 (suppl):S77-S121.
12 Shah BV. Barnwell BG. Biir GS. *SUDAAN User's Manual, Release 7.0.* Research Triangle Park, NC: Research Triangle Institute; 1996.
13. Peto R, Speizer FE. Cochrane AL, et al. The relevance in adults of air-flow obstruction, but not ol mucus hypersecretion, to mortality from chronic lung disease: results from 20 years of prospective observation. *Am Rev Respir Dis.* 1983; 128:491-500.
14. Higgins *M.* Epidemiology of COPD: state of the *an Chest.* 1984;85(suppl):3S-8S.
16. Burrows B, Lebowitz MD, Camilli AE, Knudson RJ. Longitudinal changes in forced expiratory volume in one second in adults: methodologic considerations and findings in healthy nonsmokers. Am *Rev Respir Dis.* 1986;133:974-980.
16. Rennard SI. COPD: overview ol definitions, epidemiology, and factors influencing its development. *Chest.* 1998;113(suppl):235S-241S.
17. Wald NJ. Watt HC. Prospective study of effect ol switching from cigarettes to pipes or cigars on mortality from three smoking-related diseases. *BMJ.* 1997: 314:1860-1863.
18. Nelson DE, Davis RM, Chrismon JH, Giovino GA. Pipe smoking in the United States, 1965-1991: prevalence and attributable mortality. *Prev Med.* 1998;25:91-99.
Ig. Wise RA. Changing smoking patterns and mortality from chronic obstructive pulmonary disease. *Prev Med.* 1997;26:418-421.
20. Webs ST, Segal MN, Sparrow D, Wager C. Relation of FEV1 and peripheral blood leukocyte count to total mortality: the normative aging study. *Am J Epidemiol.* 1995;142:493-498.
21. Petty TL, Pierson DJ, Okk NP, Hudson LO. Walker SH. Follow-up evaluation of a prevalence study for chronic bronchitis and chronic airway obstruction. Am *Rev Respir Dis.* 1976;114:881-890.
22. Ferris BG Jr, Anderson 00. The prevalence of chronic respiratory disease in a New Hampshire town. *Am Rev Respir Dis.* 1962;86:165-177.
23. Higgins MW, Keller JB, Landis JR. et al. Risk of chronic obstructive pulmonary disease: collaborative assessment of the validity of tha Tecumseh index of risk. *Am Rev Respir Dis.* 1984;130:380-385.
24. Mueller RE, Keble DL, Plummer J. Walker SH. The prevalence of chronic bronchitis, chronic airway obstruction, and respiratory symptoms in a Colorado city. *Am Rev Respir Dis.* 1971;103:209-228.
26. Burrows B, Bloom JW. Traver GA, Cline MG. The course and prognosis of different forms of chronic airways obstruction in a sample from the general population. *N Engl J Med.* 1987;317:1309-1314.
26. Burrows B. The course and prognosis of different types of chronic airflow limitatbn in a general population sample from Arizona: comparison with the Chicago "COPD" series. *Am RN Respir Dis.* 1989;140(suppl):S92-S94.
27. Anthonisen NR. OM-88V for COPD. *Am J Respir Crit Care Med.* 1997;156:1713-1714.
26. Collet JP. Shapii P. Ernst P, Renzi T, Ducruet T, Robinson A Effects of an immunostimulating agant on acute exacerbations and hospitalizations in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 1997; 156:1719-1724.
29. Tomee JF. Koeter GH. Hiemstra PS. Kauffman HF. Secretory leukoprotease inhibitor: a native antimicrobial protein presenting a new therapeutic option? *Thorax.* 1998;53:114-116.
30. van Schayck CP, van Grunsven PM, Dekhuijzen PN. Do patients with COPD benefit from treatment with inhaled corticosteroids? *Eur Respir J.* 1996;9:1969-1972.
31. Dompeling E, van Schayck CP. van Grunsven PM. et al. Slowing the deterioration of asthma and chronic obstructive pulmonary disease observed during bronchodilator therapy by adding inhaled corticosteroids: a 4-year prospective study. *Ann Intern Med.* 1993;118:770-778.
3 2 Kerstjens HA, Brand PL, Hughes MD, et al. A comparison of bronchodilator therapy with or without inhaled corticosteroid therapy for obstructive airways disease. *N Engl J Med.* 1992;327:1413-1419.
33. Conlabniwi M, Mainardi E. Della Porta R, et al. Inhaled corticosteroids reduce neutrophilic bronchial inflammation in patients with chmnii obstructive pulmonary disease. *Thorax.* 1998;53:583-585.
34. Shahar E, Folsom AR, Melnick SL, et al. Dietary n-3 polyunsaturated fatty acids and smoking-related chronic obstructive pulmonary disease. *N Engl J Med.* 1994; 331:228-233.
35. Mannino OM, Etzel RA, Flanders WD. Do the medical history and physical examination predict tow lung function? *Arch Intern Med.* 1993;153:1892-1897.
36. Badgett RG, Tanaka DJ, Hunt OK, at al. Can moderate chronic obstructive pulmonary disease ba diagnosed by historical and physical findings alone? *Am J* A&d 1993;94:188-196.

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