# Epidemiology of Environmental Lung Disease

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### INTRODUCTION

The health hazards posed by gases and dust in the air we breathe have become a matter of widespread public interest and concern. This, in turn, has led to farreaching attempts to protect people from these hazards. Federal as well as state agencies are deeply involved in efforts to clean the air outdoors and in factories, through programs which have considerable impact on our economy and on our personal lives. Recently, even smoking has become a topic for laws and regulations, introduced by politicians whose main decisions used to be made in proverbial smokefilled rooms.

We performed epidemiological studies of incipient and overt lung disease associated with some of these inhaled toxic agents. Although inhalation of foreign matter is a crucial factor in the pathogenesis of infectious lung disease and of common forms of lung cancer, the scope of our survey is limited to disorders such as chronic bronchitis and asthma, which lead to changes in lung function and to persistent respiratory symptoms.

Our aim was to establish baseline data for lung function in healthy persons not exposed to known environmental risks, and, next, to examine how certain common risks affect lung function and lead to respiratory symptoms in the general population, and in a population with a specific occupational risk. On the basis of these data, we attempt to provide a perspective on the relative risks posed by cigarette smoking, community air pollution, and occupational exposure to cotton textile dust.

## POPULATIONS AND METHODS

To establish the prevalence of respiratory symptoms and to measure the function of the lungs, we used well-known methods suitable for use in large groups of people. With a mobile laboratory, we brought these methods to the communities where our subjects lived. The size of our populations-altogether some 8,000 people were studied-posed data collection problems of quality control, storage, and analysis, which we minimized with computer techniques. In addition to studying the populations, we obtained data on their environment outdoors, as well as some data on indoor pollution in homes.

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This is the fourth of a series dealing with the impact of environmental issues on medicine. Henry R. Black, M.D., and Hallie Black, M.F.S., are guest editors.

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#### Populations

Residents of rural, urban, and semi-rural communities were studied. We chose Lebanon, Connecticut, as a rural town, remote from urban centers, with only light traffic, and without factories. The urban community we studied was Ansonia, Connecticut, a typical small factory town near New Haven. Reviews of air pollution reports (1966-1972) showed that Ansonia had total particulate levels which were among the highest in the state (mean annual particulate levels up to 152  $\mu$ g/m<sup>3</sup>); presumably, sulfur dioxide concentrations have been high as well in the recent past. No previous pollution data were available for Lebanon, nor for Winnsboro, South Carolina, which we chose as a small, semi-rural town with a different climate and a large black population group. Winnsboro was also suitable for comparisons with our results obtained in a separate study of cotton textile workers in nearby Columbia, South Carolina. Winnsboro has several industries, but, since the neighboring textile mills have spun only synthetic fibers since the 1940s, few Winnsboro residents have had significant exposure to cotton textile dust.

In each of these three towns, we studied all persons aged <sup>7</sup> years and older who consented to participate and who lived in <sup>a</sup> geographically defined area. The population of each area was enumerated in a private census, and samples of nonresponders were visited at home to determine whether or not they differed in important respects from the participants. The organization of the surveys, demographic data, and comparisons between participants and nonrespondents have been published in detail for Lebanon and Ansonia [1]. Table <sup>1</sup> summarizes data on the Connecticut populations and adds details for Winnsboro. In Winnsboro, the school system admits students from surrounding Fairfield County as well as from the town itself. By including the total student population in our study we increased the numbers of subjects in the age range 7-24 by more than 1,200; two-thirds of the added group were black. Comparisons of lung function in students in the Winnsboro school system showed no differences between those from the town and from the county, by sex, race, age, height, and weight; we therefore pooled their data in the further analyses. The adult population included 200 persons aged 25 and over (17.8% of total) who lived outside the city limits but worked in town (teachers, city employees). We considered them part of the total town population.

As a group at risk of occupational dust exposure, we studied active and retired, male and female white cotton textile workers aged <sup>45</sup> years and older. On the average they had worked 35 years in carding, spinning, yarn preparing, weaving and other jobs in cotton mills in Columbia, South Carolina. Details on this population group have been published elsewhere [2].

#### Response Rates

Within the town of Winnsboro, we performed <sup>a</sup> private census and determined response rates by race, sex, and age (Table 2). As in Lebanon and Ansonia [1], response rates were higher for women than for men (data not shown), and they were higher for whites than for blacks, and lower for adults than children. We studied over  $90\%$  of the students in the school system, and about 60% of the white adults; among black adults, response rates were low. To establish whether those we studied differed in important ways from those not seen, we conducted door-to-door surveys of nonrespondents in geographically defined parts of the town. With a simplified questionnaire, we determined the prevalence of respiratory symptoms and the smoking habits of the nonrespondents. We were able to account for close to 100% of





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all residents in these areas (Table 2) so that we could compare respondents and nonrespondents.

Table 3 gives an example of the comparison of respondents and nonrespondents; there were no consistent trends toward differences in prevalence of symptoms. We have concluded that in all three communities, the respondents are a representative sample of the general population.

### Questionnaire

To record respiratory symptoms, smoking habits, occupational and other environmental exposures, previous residence, and histories of lung disease, we used a standard questionnaire published in detail elsewhere [3]. Cough and sputum production were recorded according to persistence and duration. Wheezing was qualified according to severity, frequency, and periodicity; factors which aggravated or precipitated wheezing or chest tightness were also recorded. Dyspnea was recorded by grade, in persons who were not disabled from other conditions such as heart disease or arthritis (see Definitions). To assist the interviewers, all questions were prompted by computer; the answers were recorded in computer memory [4]. Thus, no questions could be omitted and each subject's record was complete.

#### Maximum Expiratory Flow- Volume (MEFV) Curves

We selected this method to assess differences in lung function between subgroups in our population because of its sensitivity to slight degrees of airway obstruction, its reproducibility in individuals, and its simplicity [3]. The MEFV curves were recorded on-line with a computer system described in detail elsewhere [4]. Five or more curves were obtained in each subject; the data reported here are the means of the values derived from the two curves with the highest  $FEV_{1.0}$ .

Since our studies required MEFV curve recordings in different people in different places and at different times, special care was taken to ensure technical comparability of recordings at all times. For precise calibration, <sup>a</sup> standard MEFV curve produced by a motor-driven pump [5] was recorded every two hours throughout all study days. The curves and the function values for each person were recorded and calculated online [4] so that human error was excluded to the maximum extent feasible.

The MEFV curve data from all healthy, lifetime nonsmokers in the three

	Comparison of while respondents and nonrespondents in winnsport (25–64 years) <sup>-</sup>						
		Females					
Subgroups:		Н	Ш		Н	ш	
n	149	71	42	235	114	33	
Usual cough, $\%$	21	17	26	14	14	12	
Usual phlegm, %	17	22	10	9	11		
Frequent wheeze, %	13	15			6	o	
Current cigarette smokers, %	40	54	62	32	44	33	

TABLE <sup>3</sup> Comparison of white respondents and nonrespondents in Winnsboro (25-64 years)\*

\*Percentage positive responses to specific questions by sex, in three sub-groups: 1. All subjects seen in the mobile laboratory, residing in areas outside of those of the door-to-door survey; II. All subjects seen in the mobile laboratory, residing in the areas of the door-to-door survey; and III. All subjects not seen in the mobile laboratory, but questioned at home, residing in the area of the door-to-door survey. None of the differences in symptom prevalences between subgroups 1, <sup>11</sup> and III, by sex, are significant. There were more female smokers in group II than in Group I (I vs. II,  $p = 0.03$ ) but smoking habits did not differ significantly between males or females seen in the mobile laboratory (11) and those interviewed at home (111).

communities have been used to derive regression equations (with age, height, and weight variables) which describe the data optimally according to objective statistical criteria [6]. They predict normal lung function with equal confidence regardless of age, height, and weight. Separate equations are used for men (age 18+), women (age 15+), boys (age 7-17) and girls (age 7-14), and for blacks and whites in these four groups by sex and age. The population groups used to develop the equations exclude all those who have ever smoked tobacco in any form, and also those with significant (more than one year) occupational exposures in known-risk industries. In one subgroup (black adult males) we included healthy smokers and ex-smokers as well as nonsmokers to obtain a group large enough for analysis.

#### Interviewers

We considered self-administration of questionnaires by the subjects but abandoned this as too time-consuming, impractical in young children, and prone to errors of interpretation. Therefore, we used specially trained interviewers who were given detailed instructions on the questions, their interpretation, and on all common problems that arise during these interviews. Each interviewer's performance was monitored by a supervising staff member, who also spot-checked individual records in repeat interviews and who identified persons with clinically significant symptoms (e.g., recent hemoptysis).

The use of locally hired and trained interviewers for each survey poses problems of observer error and comparability of results. In each town, eight interviewers performed most of the interviews and function tests. For 14 key questionnaire data, we examined the extent of variation by interviewer in each survey  $(x^2$  contingency analysis), by age, sex, and race of their subjects. In Lebanon and Ansonia, responses to the questions used in this paper showed no excess variation. In Winnsboro, the only significant finding was under-reporting of symptoms by 45-64 year-old white males to three black interviewers; the data from these 49 men were excluded from analysis. To compare performance of the lung function test among interviewers, we used the measurement of peak flow rate (PEF) which is highly dependent on the subject's effort. In Lebanon and Ansonia, no single interviewer differed significantly from all others (analysis of covariance, taking sex, age, and height into account). In Winnsboro, three interviewers recorded slightly lower PEF values in some groups but not in others, and the significance of the differences was marginal. Thus, we have concluded that interviewer, variation is not an important factor in the results presented in this paper.

#### Air Pollution

We monitored the air pollutants listed in Table 4 in each of the three communities, using commonly accepted methods [7]. In Lebanon and Ansonia, sampling was conducted on a regular schedule (every sixth or twelfth day) from November 1972 to December 1973; in Winnsboro, air samples were taken on <sup>11</sup> consecutive days in each of the four seasons, between October 1973 and July 1974. All sampling locations were in built-up areas where many subjects lived. Details and a summary of the average results are included in Table 4, together with demographic data on the three towns. Throughout most of the monitoring period, Ansonia had higher concentrations of total suspended particulates, nitrogen dioxide, sulfates, and nitrates than either Lebanon or Winnsboro. With respect to oxidant (ozone) concentrations, the three towns were remarkably similar. Sulfur dioxide concentrations were low in all three towns, with Winnsboro having the lowest values. Air pollutant concentrations varied



as a function of season and weather conditions [8]. Our data in Connecticut confirmed the importance of pollutant transport from the New York metropolitan area [9] and added the suggestion that much of the sulfate ions in the air over Connecticut may be derived from seawater spray.

In separate studies we monitored indoor air pollution (respirable particulates,  $SO_2$ , and  $NO<sub>2</sub>$ ) in homes in the three towns. Indoor pollutant concentrations, in particular of respirable particulates, were often higher indoors than outdoors, especially in homes of smokers [10]. There were also indications that homes equipped with gas stoves had higher  $NO<sub>2</sub>$  concentrations than electrically equipped homes [11]. Detailed dust measurements in the cotton textile mills where our Columbia, South Carolina, workers were employed are not available. However, dust concentrations up to 2.05  $mg/m<sup>3</sup>$  (area samples) and up to 4.33 mg/m<sup>3</sup> (personal samples) were measured in one of these mills (South Carolina Department of Labor, April 9, 1975).

### **Definitions**

Residence For comparisons between urban and rural residents, we examined four categories in Ansonia and Lebanon:

- 1. Lifelong rural residents (LR): Persons who lived in Lebanon all their lives or who had moved there, coming from another rural area.
- 2. Previous urban residents (PU): Persons who lived in Lebanon but who had previously lived in urban areas.
- 3. Lifelong urban residents (LU): Persons who lived in Ansonia all their lives, or who had moved there, coming from another urban area.
- 4. Previous rural residents (PR): Persons who had moved to Ansonia, coming from a rural area.

Since PU residents constituted  $41\%$  of the number of subjects studied in Lebanon, this category needed to be examined separately. In contrast, there were only 73 previous rural residents in Ansonia (both sexes, whites, all ages); these were omitted from analysis.

We did not categcrize Winnsboro residents according to residence; most of them were lifelong rural dwellers. In Lebanon and Ansonia, we excluded those whose history of prior residence (urban or rural) was unclear. For most analyses, we excluded those who had been exposed to known occupational hazards of lung disease for more than one year, to avoid confounding effects of this variable.

Smoking habits were grouped into three categories:

- 1. Lifelong nonsmokers (NS): Those who had never smoked tobacco in any form.
- 2. Ex-smokers (XS): Those who had previously smoked cigarettes but no longer did so at the time of the survey.
- 3. Smokers (S): Those who currently smoked cigarettes.

Smokers of pipes and cigars only were excluded since they were too few for analysis. If they also smoked, or had smoked cigarettes, they were included according to their cigarette smoking habits.

Symptoms and symptom complexes in this paper are defined as follows: Usual cough or phlegm: cough or phlegm on most days, at least <sup>3</sup> months/year. Frequent wheezing: wheezing or chest tightness at least a few times each week.

Chronic bronchitis: usual cough and usual phlegm on most days, at least three months/year during at least the past two years.

Dyspnea:

Grade 1: Shortness of breath when hurrying on ground level or walking up a slight hill.

Grade 2: Shortness of breath walking with other people of one's own age on level ground, or worse.

Grade 3: Shortness of breath walking a quarter mile on level ground in about 15 minutes or worse.

Abbreviations of lung function values are as follows: FVC <sup>=</sup> forced vital capacity  $FEV<sub>1.0</sub>$  = forced expiratory volume in 1 second PEF <sup>=</sup> peak expiratory flow MEF50% = instantaneous maximum expiratory flow at 50% of FVC MEF25% <sup>=</sup> similar, at 25% of FVC (= with 25% of FVC remaining to be expired)

### RESULTS

We shall first consider the prevalence of respiratory symptoms, chronic bronchitis, and asthma in the four main population groups: Lebanon, Ansonia, Winnsboro, and the group of cotton textile workers in Columbia, SC. Next, smoking, occupational exposure, and air pollution will be considered separately as risk factors, using appropriate subgroups for comparisons of people at different degrees of risk.

#### Prevalence of Chronic Bronchitis

The overall prevalence of chronic bronchitis in all four populations is summarized in Fig. 1. The syndrome is rare among the young, and more common among men than women. There is a significant increase of prevalence with age, among men as well as women, largely because the syndrome is rare among the 7-24 year olds. Among blacks, older women (45+ years) have less chronic bronchitis than white women. There are no significant differences between the prevalences in the three community populations, but cotton textile workers (active or retired) have a much higher prevalence than those in the same age group in the three communities. Chronic bronchitis is relatively rare among nonsmokers (Fig. 2), especially women, but nonsmoking cotton textile workers have a significantly higher prevalence. Exsmokers (data not shown) have prevalences close to those of lifetime nonsmokers, for all groups by sex and age. Cotton textile workers (nonsmokers, smokers, and exsmokers) have higher prevalences than community residents in the same smoking categories. Further subdivision of the Lebanon and Ansonia populations in LR, PU, and LU residents (see Definitions) does not show consistent prevalence patterns which might suggest an "urban factor" in chronic bronchitis.

### Prevalence of a History of Asthma

A positive answer to the question, "Have you ever had asthma?" was most common among young Winnsboro white males and least common among older urban men and women (Fig. 3). Among men, prevalence decreased with age, but among women there were no significant trends with age. The difference between urban and rural males persisted when lifetime urban and rural residents in Connecticut were compared, but the higher prevalence among rural residents was no longer significant. The differences among the three communities persisted when only lifetime nonsmokers were compared. By  $X^2$  analysis, a history of asthma was not significantly related either to smoking habits or to race.

### Prevalence of Cough, Phlegm and Other Symptoms:

Comparisons of the prevalences of two principal symptoms, i.e., usual cough and frequent wheezing, are shown in Table 5. Most subgroups of cotton textile workers



FIG. 1. Percent prevalence of chronic bronchitis (= usual cough and usual phlegm 3 months per year, 2 or more years). All smoking categories are included. Since residence (Lebanon, Ansonia, or Winnsboro) was not a significant variable among whites, the residential categories were combined for most analyses. The data given for blacks include those who live in Ansonia and in Winnsboro. The increase of prevalence with age is highly significant for white males and females, and for black males ( $\chi^2$  test;  $p < 0.01$ ). Black women (45+ years) have significantly ( $p < 0.01$ ) less chronic bronchitis than white women of the same age in Winnsboro, where most blacks lived. White males aged 25-44 years and 45+ have significantly more chronic bronchitis than white females at the same age; the same is true for black males vs. black females age 45+ (all  $p < 0.01$ ). White cotton workers ( $n = 645$ ) by sex, have a significantly higher prevalence than white community residents of the same age (see also [2]).

have significantly higher prevalences of both symptoms than the respondents in the three communities combined  $(X^2$  test) [2]. For usual cough, Ansonia residents have slightly higher prevalences than Lebanon residents. There are no consistent differences between Ansonia and Winnsboro, but the numbers of subjects, especially males, in the Winnsboro group are small.

The graphs of Fig. 4 include four variables that might affect symptom prevalence: sex, age, residence, and smoking, for the same symptoms as in Table 5. To determine the contribution of each to the variation in prevalence, we performed a weightedleast-squares analysis among 15-64 year old whites in Lebanon and Ansonia for these two and other symptoms [12]. The results show:

1. Smoking is significantly associated with the prevalence of all five symptoms (cough, phlegm, recent or frequent wheeze, dyspnea).

2. Prevalences increase significantly with age for all symptoms except frequent wheezing; for usual cough, the 25-44 year olds have a higher prevalence than those younger or older.

3. Sex is significant for usual phlegm (more common among men) and for dyspnea (more common among women).

In view of the complex relations between sex, age, smoking, and residence, in their

**Chronic Bronchitis in Lifetime Nonsmokers (Whites)** 



FIG. 2. Prevalence of chronic bronchitis among lifetime nonsmokers (whites). Numbers of subjects: 875 males and 943 females aged 7-24 years; 228 males and 794 females aged 25+ (Lebanon, Ansonia, Winnsboro); 50 male and 234 female cotton textile workers.



FIG. 3. Prevalence of positive answers to question: "Have you ever had bronchial asthma?", by race, residence, age, and sex (all smoking groups combined). X<sup>2</sup> analysis: whites (all residential groups) vs. blacks, by sex and smoking habits, not significant. Males vs. females: significantly higher prevalence among 7-24 year old white males vs. 7-24 year old white females ( $\chi^2$  = 17.35,  $p < 0.01$ ); other differences not significant. Nonsmokers vs. all smoking groups by sex, race, and age group: no significant differences. Residence (white only):  $*_p$  < 0.05, \*\*p < 0.01 for differences between Lebanon or Winnsboro residents and Ansonia residents, by sex. Age: significantly higher prevalence in 7-24 year old white males vs. 25-44 year olds ( $p < 0.05$ ); not significant in females.

		Usual cough				Frequent wheezing				
	L	A	W	$\mathbf{C}\mathbf{W}$	L	A	W	cw		
Males										
NS $%$	8	10	15	17	3	$\bf{0}$	23	19		
n	(37)	(19)	(13)	(42)	(37)	(19)	(13)	(42)		
$S \%$	27	32	34	59	21	17	34	42		
n	(66)	(47)	(29)	(76)	(66)	(47)	(29)	(76)		
Females										
NS $%$	7	12	9	23	5	7	8	26		
$\boldsymbol{n}$	(97)	(94)	(75)	(168)	(97)	(94)	(75)	(168)		
$S \%$	13	17	24	33	18	4	13	33		
n	(63)	(47)	(54)	(61)	(63)	(47)	(54)	(61)		

TABLE <sup>5</sup> Prevalence of respiratory symptoms in four populations (whites, 45-64 years) by sex and smoking habits

Percent prevalence of two common symptoms;  $n =$  no. of subjects. L = Lebanon, A = Ansonia, W = Winnsboro, CW = Cotton textile workers. L, A, and W subjects exclude those with  $> 1$  year work in cotton textile mills. Differences between L, A, and W not significant. Cotton workers, by sex and smoking group, have significantly higher prevalences of each symptom than L, A, and W subjects combined except among the small group of male nonsmokers. Differences between nonsmokers and smokers are significant for cough and wheezing in males (p  $< 0.01$ ) and for cough in females ( $p < 0.02$ ) in L, A, and W groups combined.



FIG. 4. Prevalence of two symptoms in white LR, PU and LU residents (see Definitions), white Winnsboro residents (W) and black residents of Lebanon and Winnsboro combined (B). Usual cough = cough on most days, more than 3 months per year; frequent wheezing at least a few times each week. \*Absence of bar means zero prevalence.

Smokers have higher prevalences than nonsmokers for both symptoms in all subgroups by sex, age, race and residence, except for wheezing among black adult males (29 nonsmokers; 36 smokers). No residence effect is evident among smokers in any sex and age group. An urban-rural gradient  $(LR < PU < LU)$  is present for usual cough in nonsmoking white adults, but not in 15-24 year olds nor for wheezing in any group.

association with symptom prevalences, examination of these prevalences with simpler analyses such as the  $X^2$  test in groups with a limited age range, or of one sex only, may not allow conclusions about the general population. We were able to detect a significant association between residence and the prevalence of some symptoms largely because our population groups include many nonsmokers, especially women.

In Winnsboro, the prevalence of most symptoms, by sex, age, and smoking habits, is similar to that among comparable Lebanon residents (Fig. 4). In this sense, the Winnsboro residents should probably be considered a rural population group. In contrast, the cotton textile workers have higher prevalences than any one of the community population groups of the same age (Figs. <sup>1</sup> and 2; Table 5; [2]). Symptom prevalences among rural (Lebanon and Winnsboro) blacks are in most respects similar to those of whites, by smoking, sex, and residence.

### Air Pollution

There are significant differences in mean concentrations of some air pollutants between the three communities where we studied the residents (Table 4). Urban Ansonia had higher particulate,  $NO_2$ ,  $SO_4$ , and  $NO_3$  levels than rural Lebanon. In comparing urban and rural residents to detect possible associations between residential history and respiratory symptoms or lung function, we focused on the two Connecticut populations.

A weighted-least-squares analysis showed [12] that residence variables were only significant among nonsmokers, and only for usual cough (Fig. 4) and usual phlegm. In addition, dyspnea was associated with residence, but this association was complicated by interactions with smoking and with sex. For example, lifetime rural nonsmokers had less dyspnea than lifetime urban nonsmokers, but among smoking men and women lifetime rural or urban residents had less dyspnea than rural residents who had previously lived in cities. This analysis was limited to whites aged 15-64 years. The groups of urban blacks and of 65 year and older subjects were too small for comparison by sex, age, and smoking habits. Among children (7-14 years) the prevalence of symptoms was too low for meaningful analysis.

Thus, urban residence in Ansonia may be associated with a slight excess of some respiratory symptoms among nonsmokers. Among smokers, any urban-rural difference in symptom prevalence appears to be swamped by the excess of symptoms associated with smoking. However, the differences in symptom prevalences between urban and rural dwellers are not accompanied by any significant differences in lung function. To examine these differences, we used prediction equations for lung function values in relation to sex, race, age, height, and weight [6] as a reference. Lung function residuals(= observed minus predicted values) based on these equations are independent of age, height, and weight within each subgroup by age, sex, and race. Figure <sup>5</sup> shows average residuals for MEF50% in the three residential groups in Connecticut and in Winnsboro, by race, sex, age group, and smoking status. The only significant differences are between smokers and nonsmokers in comparable subgroups.

Because of the limited degree of air pollution contrast between Ansonia and Lebanon in the present study (Table 4), we have also compared our data to those of other investigators who have recorded MEFV curves in persons living in more polluted areas. For example, Cohen et al. [13] studied nonsmoking men and women living in Los Angeles and San Diego; their  $FEV_{1,0}$  and MEF50% values are similar to ours when age, sex, and height are taken into account [12]. Fig. <sup>6</sup> compares our data



FIG. 5. Average residual MEF50% (see text for definition) by sex, age group, smoking habits, residence, and race. For children, only data on nonsmokers are shown. Among nonsmokers, the residuals do not differ significantly from zero or from each other. Among smokers, residuals are significantly more negative than among corresponding nonsmokers except for black males.



FIG. 6. A, Lung function in 10-11 year old boys and girls in the U.S.A. and Czechoslovakia. Average values for FEV<sub>Ia</sub> and MEF50% (SD indicated within each bar) adjusted for slight differences in predicted values (+ or - 0.07 1 or I/sec, or less) according to Schoenberg et al. [6]. Numbers of subjects in each group are shown in bars at left. Sequence of bars from left to right: lifetime rural, previous urban, and lifetime urban residents of Lebanon and Ansonia; residents of "clean" and "polluted" towns in Czechoslovakia (data from Zapletal et al., [ 14]). Right-hand graph (B) shows mean SO<sub>2</sub> and particulate concentrations in Lebanon (L) and Ansonia (A) (data from Hosein et al., [7]) and in "clean" (C) and "polluted" (P) towns in Czechoslovakia (data from Jech and Kaspar, [ 15]). The Czech data may underestimate TSP by as much as  $100 \mu g/m^3$  because of methodological differences.

in 10- and 11-year-old children with those obtained by Zapletal in children living in a highly polluted town [14]. Again, after differences in height are taken into account, the groups do not differ. Around  $1962$ , SO<sub>2</sub> and particulate concentrations in Manhattan (with methods similar to ours) were high—about 500  $\mu$ g/m<sup>3</sup> and 250  $\mu$ g/m<sup>3</sup> [16]—yet, FEV<sub>1.0</sub> values of male nonsmokers working in outdoor jobs in New York City (blacks and whites) in 1961-1962 [17,18] are nearly identical to those in our subjects in 1973 when age, race, and height are taken into account. The New York City data concern 1,187 white and 467 black male nonsmokers, aged 30-59 years, representing more than 80% of a defined population of post office and transit workers. As far as black subjects are concerned, there were only minimal differences in lung function between urban residents of Ansonia and residents of Winnsboro [6]. The pooled data for FVC and  $FEV_{1,0}$  in our black subjects from both towns are very similar to those obtained by Huizinga and Glanville in a primitive village in Upper Volta (178 adults, 97 children) [19], taking age, sex, and height into account. Cars, factories, and other modern sources of outdoor pollution were absent in this village, which is situated away from cities and other potential sources of pollutant emissions.

Thus, we do not find any clear indications that current levels of either oxidant pollution, as in Los Angeles, or  $SO_2$ -particulate pollution, as in New York City in 1961-1962 and in the Czech studies of Zapletal et al., are associated with a measurable loss of lung function among white subjects. The fact that our black subjects in Ansonia, and black males in New York City [18], have lung function values similar to those of blacks living in rural Africa suggests that, for blacks as well, urban air pollution is not a demonstrable risk factor with respect to lung function.

Although respirable particulate levels were often high indoors in homes of smokers [10], and high  $NO<sub>2</sub>$  levels were observed in homes with gas appliances [11], analysis of lung function results gave no indication that indoor air pollution in the range of our observations (up to about 150  $\mu$ g/m<sup>3</sup> respirable particulates, and up to 500  $\mu$ g/m<sup>3</sup>  $NO<sub>2</sub>$ , 24 hr averages) is a risk factor in the development of lung function loss.

### Smoking and Lung Function

To examine associations between cigarette smoking and loss of lung function we used lung function residuals (see Air Pollution). Table 6 presents average residuals for white males and females in different smoking groups, by number of packyears (I packyear = 20 cigarettes per day for <sup>1</sup> year). In white adults the average decrement of lung function-in comparison with healthy, lifetime nonsmokers on whose data the prediction equations are based-increases with the amount smoked, among males as well as females. This dose-response relation is particularly clear among those who still smoked at the time of our surveys, but linear trends of dose vs. response are significant for ex-smokers and smokers and both for  $FEV_{1,0}$  and MEF50% (Table 6).

Among blacks (data not shown), the dose-response relations between amount smoked and lung function loss are less clear. This may be due to the small number of heavy smokers. Among the 1,635 whites in Table 6, 31.8% have smoked 20 packyears or more; only about 10% of the blacks have smoked as much. Among white heavy smokers (packyears 40+), the dose-response relation no longer holds (data not shown). This may be due to self-selection; only people with relatively good lung function may continue to smoke heavily.

Further analysis of lung function residuals (FEV<sub>1.0</sub>, MEF50%, MEF25%) by age shows that white male current smokers aged 18-24 years (average 19.3 cig/day;  $n =$ 73) have a positive average residual; only those 25 years and over have negative average residuals. Among children, white male current smokers (7-17 years) also

			Males $18+$			Females 15+			
	Packyears	n	Residual <b>MEF50%</b> (liters/sec)	Residual $FEV_{L0}$ (liters)	n	Residual <b>MEF50%</b> (liters/sec)	Residual $FEV_{1.0}$ (liters)		
<b>Smokers</b>	$0 - 9$	116	.10	.02	313	$-.18$	$-.08$		
	$10 - 19$	129	$-.27$	$-.22$	158	$-.56$	$-.23$		
	$20 - 29$	78	$-.44$	$-.42$	71	$-.61$	$-.26$		
	$30 - 39$	59	$-.73$	$-.40$	40	$-.82$	$-.38$		
	$40+$	76	$-1.13$	$-.58$	22	$-.90$	$-.33$		
Ex-smokers	$0 - 9$	96	.02	$-.01$	171	$-.14$	$-.06$		
	$10 - 19$	89	$-.20$	$-.07$	43	$-.16$	$-.10$		
	$20 - 29$	57	$-.36$	$-.26$	23	$-.25$	$-.03$		
	$30 - 39$	29	$-.65$	$-.29$	8	$-.93$	$-.36$		
	$40+$	49	$-.77$	$-.33$	8	$-.27$	$-.36$		

TABLE <sup>6</sup> Residual lung functions in smokers and ex-smokers (white adults) by packyears and by sex

have slightly positive average residuals (average <sup>8</sup> cig/ day). Among these young smokers, 49 boys who smoked <sup>1</sup> packyear or more have negative average residuals for MEF50% and MEF25%. Among girls (7-14 years), the average residuals for these flow rates are negative regardless of the amounts smoked. Thus, there are indications that young male smokers, in particular light smokers, have slightly better lung function than healthy lifetime nonsmokers at the same age. This may reflect a selection process, with persons with better lung function more likely to smoke. However, heavier smokers among the males, and also smoking girls, already have slight decrements of flows on the MEFV curve (MEF50% and MEF25%). This confirms previous findings from our laboratory [20] which showed that smoking boys and girls ( $> 15$  or 10 cigarettes per day, respectively; age 15–19 years) had significantly lower values of MEF50% and MEF25% than comparable lifetime nonsmokers.

With few exceptions, ex-smokers in Table 6 have better lung function than current smokers in the same packyear category. This may reflect partial reversibility of the effect of smoking, once the habit is abandoned. There are no indications that women are less sensitive than men to the effects of smoking on the airways.

#### Occupational Exposure to Cotton Dust

Data on respiratory symptoms and lung function in 645 white male and female cotton textile workers, aged 45 years and older, have been described in detail elsewhere [2]. Comparisons of symptom prevalences in this group and in community populations are included in Figs. <sup>1</sup> and 2 and in Table 5. Lung function results confirm the impression of a significant excess of chronic lung disease among textile workers of both sexes. Figure 7 compares average residuals for  $FEV_{1,0}$  in textile workers and community residents, by sex and smoking habits. Comparisons of FVC and flow rates (MEF50%, MEF25%) gave similar results [2]. In each sex and smoking category, the cotton workers have a greater average function loss than the control subjects from the community populations; the differences are highly significant in five out of the six groups (there were only 37 ex-smoking female cotton workers in the group which is the exception). Moreover, the function deficit associated with cotton dust exposure is similar in all smoking categories; the effects of smoking and of cotton dust exposure on lung function appear to be additive.



FIG. 7. Average residual  $FEV_{10}$  in cotton workers and controls (45+) by sex and by smoking habits. p values apply to differences between cotton workers and control subjects in each of the six subgroups by sex and smoking status.

The increased loss of lung function among cotton textile workers probably results from an increased decrement of function with age, in comparison with the control subjects. Direct evidence for this has been obtained in a longitudinal study of hemp workers, in whom lung function continued to decrease at an increased rate, even in the absence of further exposure to dust [21]. If lung function decreases more rapidly with age, more cotton textile workers are expected to reach low levels of lung function, incompatible with significant exercise capability, at pre-retirement age. To make a conservative estimate of the number of disabled persons, we assumed that an  $FEV<sub>1,0</sub>$  of 1.20 liters or less in males, and 1.00 or less in females, is associated with disability due to lack of ventilatory reserves. Under this definition, 6.2% of the male and 5.4% of the female textile workers were disabled (2.2% and 2.7% in the controls). Among textile workers 45-64 years of age, 21/481 (4.4%) were disabled, vs. 16/1009 (1.6%) among the controls ( $x^2 = 10.52$ ,  $p < 0.005$ ). Hence, significantly more textile workers than control subjects were disabled at pre-retirement age.

#### DISCUSSION

In the present study, we have used data on over 3,000 healthy, lifetime nonsmoking men, women, and children (age 7+) to establish reference values for the growth and decay of lung function in whites and blacks. The equations which describe these physiological changes in lung function [6] predict  $FVC$ ,  $FEV_{1,0}$ , and flow rates on MEFV curves with equal confidence regardless of age, height, and weight. We have used these reference data to examine lung function among more than 4,000 smokers, ex-smokers, and persons with histories of chronic bronchitis, respiratory symptoms, and/or asthma. In addition, we examined environmental variables which might

affect prevalences of respiratory symptoms and of histories of asthma. Here we briefly summarize the main results concerning the associations between lung function and symptoms on the one hand, and three important environmental exposures on the other hand: cigarette smoking, outdoor air pollution, and occupational exposures to textile dust.

A history of chronic bronchitis was uncommon among the young and among older nonsmokers. An excess prevalence occurred, in accordance with many previous studies, among older smokers (compare Figs. <sup>1</sup> and 2); a history of chronic bronchitis appears to be a late sequel of exposures to cigarette smoke and to textile dust.

The distribution of the prevalence of asthma, as determined from a questionnaire response, differs from that of chronic bronchitis when examined by age and sex (Fig. 3). A history of asthma was most prevalent among young males, in particular residents of the two rural towns. Among women, the highest prevalence occurred in the middle age group (25-44 years) and there was no significant association with residence. The data of Figs. 1-3 suggest that the prevalences of histories of asthma and of chronic bronchitis differ with respect to their distribution by sex, age, and smoking habits. Thus, it seems incorrect to lump these histories together under some general term like chronic obstructive lung disease.

The analysis of lung function shows significant decrements of maximum expiratory flow rates among current and past cigarette smokers and among cotton textile workers. Among the latter, the effects of smoking and of textile dust exposure on lung function appear to be additive. In contrast, we have not found any association between outdoor air pollution in urban Ansonia and decrements of lung function. In addition, a comparison with data from other areas where either oxidant or particulate-SO<sub>2</sub> pollution were more marked did not show detectable differences in lung function between residents of those areas and residents of the towns in our study. Thus, we have concluded that present air pollution levels outdoors, and probably also the somewhat higher levels that occurred in the past, have no demonstrable effect on lung function. There was a slight excess of cough and phlegm among urban nonsmokers, compared to rural nonsmokers. However, it is not clear whether this excess of minor symptoms (in contrast with the lack of excess prevalence of chronic bronchitis) means that urban air pollution has a deleterious effect on health. A slight increase in cough and phlegm might also be interpreted as the result of useful adaptive mechanisms which help to clear pollutants from the lungs.

From <sup>a</sup> public health point of view, it seems evident that cigarette smoking and textile dust exposure are far more deleterious to respiratory health than outdoor air pollution under present conditions in the U.S. Even the much higher levels of urban particulate- $SO<sub>2</sub>$  pollution that prevailed in many cities in the recent past, as well as the high oxidant concentrations that are still common in Los Angeles, do not appear to have perceptible effects on lung function. This finding contradicts the common belief that air pollution represents a continuing hazard to the respiratory health of urban residents. Our conclusion also contrasts with that of Lave and Seskin [22] whose mortality statistics suggest that reduction of air pollution would decrease the total, unadjusted mortality rate by 7 percent or more. This is difficult to explain if there is in fact not much excess illness due to air pollution. In our studies we carefully measured air pollution at the time we studied the residents, and we took smoking, migration, and race fully into account. Lave and Seskin used existing air pollution data, incomplete in several respects, and they ignore effects of smoking. Others as well [23,24] have concluded that the data base used by Lave and Seskin is too poor to allow reliable conclusions. The present study provides additional support for the

conclusion that "while there are many compelling reasons to reduce pollution, ill health is not foremost among them" [25]. It seems clear that no measurable benefits can be expected from air pollution control as far as the prevalence of asthma, chronic bronchitis, and loss of lung function is concerned. On the other hand, considerable benefits to health could accrue from systematic efforts to reduce occupational exposures to textile dust and other inhalant risks in industry [26]. The recently announced federal standard for cotton dust [27] prescribes engineering controls to limit dust exposures as well as medical surveillance of workers. The proposed measures, if adequately implemented, can largely eliminate health hazards from cotton dust exposure.

The long lead time in the development of chronic bronchitis and lung function loss among cigarette smokers (Fig. 1; Table 6) is likely to remain an important obstacle for programs to decrease the impact of this unhealthy personal habit. In addition, only <sup>a</sup> minority of smokers becomes disabled from chronic airway obstruction. We have recently (1978) re-studied more than 1,300 residents (age 12+) of Lebanon, six years after the initial study. The analysis of changes in lung function during this follow-up period may provide clues about the development of chronic bronchitis and airway obstruction among smokers.

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