Emphysema must of necessity be related to alteration of this skeleton. The pathogenesis of emphysema therefore must be much more complex than postulated by Laenniac. It was not until later that severe α1 antitrypsin deficiency and its relationship to emphysema was recognized. This observation, together with the finding that experimental emphysema in animals could be produced by elastase, led to the current theory of pathogenesis.

Looking back on the conference, it seems remarkable that the planners focused on etiology and pathogenesis. It is apparent that the answer to the problem of emphysema lies in prevention or early treatment. Current therapy is extremely costly and does little to halt the progress of the disease. It is reminiscent of the therapy for tuberculosis prior to the development of chemotherapy. There is no reason to believe that the incidence or mortality from the disease is decreasing. Control of the disease will depend on dealing with cigarette smoking both as a personal addiction and as a huge source of income for tobacco growers and cigarette manufacturers. This poses social, psychologic, political, and economic problems with which most of us are unfamiliar. It is as though we had climbed a mountain and believed we had reached the summit, but instead were faced with a massive barrier of snow and ice that must be ascended before reaching the top.

Session 1

Epidemiology of COPD
State of the Art

Millicent Higgins, M.D., D.P.H., FCCP*

The main purpose of epidemiology is to identify causes and risk factors associated with the development of disease. Other purposes are: to measure the frequency and describe the distribution, range of severity, and course of disease; to estimate risk for subgroups within the population and to identify persons at highest risk. This information is relevant to preventing disease, to screening and early detection, to predicting onset, course, and outcome of disease, and to planning and evaluating preventive and therapeutic measures.

This brief review will include information on the size and severity of the problem of chronic obstructive pulmonary disease (COPD) in the United States and on recent trends in mortality, but its main emphasis will be on known and suspected causes of COPD and on estimating risk of developing disease from risk factor profiles.

DEFINITIONS

Sometimes COPD is used as a diagnostic category to refer collectively to chronic bronchitis, emphysema or asthma with persistent obstruction of airflow, and sometimes as a specific diagnostic label. The use of widely accepted definitions and diagnostic criteria and the development and validation of standardized procedures for collecting questionnaire information and measuring lung function have led to better communication and more comparability in epidemiologic studies. Nevertheless, there is still variability and controversy surrounding the use of specific diagnoses and broader categories such as COPD or COLD. Chronic bronchitis is diagnosed in epidemiologic studies when a productive cough is present for at least 3 consecutive months per year for at least 2 years, provided it is not due to other lung or heart disease. Obstructive Airways Disease (OAD) refers to the condition in which the FEV1 is less than 65% of predicted and the FEV1/FVC ratio is less than 80%; this definition is useful in epidemiologic studies, whereas a definition of emphysema requiring knowledge of morbid anatomy is not. Asthma will not be discussed in this article, except as included in statistical data. Diagnoses from mortality records and death certificates must be accepted at face value, and inferences and comparisons must be made with caution.

MAGNITUDE OF THE PROBLEM

Data from the National Health Interview Survey provide estimates that there are about 7.5 million Americans with chronic bronchitis, 2.1 million with emphysema, and 6.4 million with asthma. Some people have more than one condition, but the numbers affected with each condition are underestimated, since only reported conditions are included in these figures. Data from the Tecumseh Community Health Study of over 9,000 males and females of all ages show that about 14% of adult males and 5% of adult females have chronic bronchitis, obstructive Airways disease, or both. Disability assessed as days of restricted activity per year is substantial and in the national data amounted to 12 days on the average for patients with chronic bronchitis, 68 days for patients with emphysema, and 17 days for asthmatics. Since emphysema is predominantly a disease of old age, days lost from work were fewest for this disease (0.1 per patient per year). Days lost were about the same for chronic bronchitis and asthma (1.0 and 1.1 per patient per year, respectively).

In 1981 there were nearly 60,000 deaths from COPD and allied conditions, the underlying cause of 3% of all deaths and the fifth leading cause in the United States. Death was attributed to emphysema in 13,600 decedents, to bronchitis in 3,900, to asthma in 3,100, and to other COPD and allied conditions in 39,230. Recently published information indicates that COPD is cited on death certificates as a contributory cause about 1/4 times as often as an underlying cause. Thus, an estimate of 150,000 deaths from and with COPD would be reasonable for 1981. The extent to which COPD is omitted entirely from death certificates of affected patients is unknown.4

Deaths from COPD are increasing; the age-adjusted death rate rose 28% between 1968 and 1978, during which time the death rate from all causes declined by 22%, and rates for heart disease and cerebrovascular disease declined by 23% and 37%, respectively. The increase for COPD death rates ranged from 97% for white females to 17% for white males. Trends over time are different for components of the COPD group of diseases. Downward trends have been apparent for

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emphysema and chronic bronchitis since 1968 and for asthma since the 1950s. COPD was not used as a death certificate code before 1968, but the trend for it and for the combined set of conditions has been upward since then. Thus, a misleading impression is gained, unless the increasing use of COPD is recognized and the combined rate considered.4

The estimated economic cost of COPD in 1979 was $6.5 billion. Of this amount, $2.3 billion was for health care and the remainder for indirect costs of morbidity and premature mortality. On the basis of illness, disability, and death rates, as well as social and economic costs, COPD is clearly an important public health problem.

DISTRIBUTION OF DISEASE

Prevalence, incidence, and mortality rates for COPD, chronic bronchitis, and emphysema increase with age and are higher in males than females and in whites than non-whites.4 The ratio of males to females is higher for emphysema and COPD than for chronic bronchitis, and the differences between the sexes increase with age. Death rates for emphysema and COPD rise more sharply with increasing age than do rates for chronic bronchitis. Incidence rates in Tecumseh for the 10-year interval from 1968 to 1978 were about the same in the two sexes below age 45, but they were about twice as high in males as in females at older ages.

Morbidity and mortality are inversely related to socioeconomic status and generally are higher in blue-collar workers than white-collar workers and in those with fewer years of formal education. COPD aggregates in families. Prevalence rates are higher in offspring of affected parents and in brothers and sisters of affected siblings. There are statistically significant correlations between measures of lung function among biologic relatives. For example, correlations between age- and height-adjusted values of FEV1 were generally between 0.2 and 0.3 for parents and children and between 0.2 and 0.4 for young adult siblings in Tecumseh.5 Studies of monozygotic and dizygotic twins show a greater similarity in respiratory symptoms and pulmonary function between MZ than DZ pairs. Results of the NHLBI twin study suggest that variation in FEV1 can be attributed to genetic and environmental determinants as well as to height, other constitutional factors, and cigarette smoking.6

CAUSES AND RISK FACTORS

Extensive data from many sources confirm that age and male sex are associated with increased morbidity and mortality from COPD.

CIGARETTE SMOKING

Cigarette smoking is also firmly established as a major risk factor for COPD (Table 1). Data from longitudinal, cross-sectional, and case-control studies show that in comparison with nonsmokers, cigarette smokers have higher death rates for chronic bronchitis and emphysema; higher prevalence and incidence rates for chronic bronchitis, emphysema, and obstructive airways disease; and higher frequencies of respiratory symptoms and lung function abnormalities. They also have a greater average annual rate of decline in FEV1. Differences between cigarette smokers and nonsmokers increase as cigarette consumption increases. Pipe and cigar smokers have higher morbidity and mortality rates for COPD than nonsmokers, but lower rates than cigarette smokers. There is also ample evidence to show that stopping smoking is beneficial. The prevalence of symptoms, chest illnesses, and disease is generally less in exsmokers than in continuing smokers, and levels of lung function are higher and rates of decline lower in exsmokers than continuing smokers. However, it appears that lost lung function is not regained.7,8 Length of time since stopping, age at stopping, amount smoked, and stage of disease at time of stopping all influence the outcome. As might be expected, exsmokers resemble nonsmokers more closely when they give up smoking at an early age or after a short period of smoking only a few cigarettes. Prognosis is less hopeful if cessation occurs at older ages, among heavy smokers, and if disease is already advanced.4,8

Although the importance of cigarette smoking as a cause of COPD is firmly established, some facets of the problem are not fully understood. For example, the health effects of cigarette smoking are not of equal frequency or severity in all smokers. Cigarette smoke does not act in isolation on uniformly susceptible people, but rather in a variable context in which personal factors and exposures to other environmental hazards, as well as the composition and dose of cigarette smoke, influence the onset and course of disease.

Cigarettes in current use differ substantially from cigarettes in use at the start of the epidemiologic studies which implicated smoking as a health hazard. The tar and nicotine contents of sales-weighted average cigarettes are now less than half their former levels, and other changes in the quality and character of cigarette smoke have resulted from the use of filters, changes in tobacco and manufacturing processes, and use of new natural and synthetic products. Current smokers also differ in several ways from smokers of 20 years ago; eg, a higher proportion are women, and the percentage of smokers smoking 25 or more cigarettes a day has increased.4,9

The health effects of cigarettes on the market today are uncertain. Recent evidence indicates that low-nicotine cigarettes do not deliver a lower dose of nicotine to smokers.10

Table 1—Risk Factors for COPD

<table>
<thead>
<tr>
<th>Established</th>
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<tbody>
<tr>
<td>Age</td>
<td></td>
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<tr>
<td>Male sex</td>
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<tr>
<td>Cigarette smoking</td>
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<tr>
<td>Reduced lung function</td>
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<td>Occupational exposures</td>
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<td>Air pollution</td>
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<tr>
<td>α1-AT-deficient phenotypes (PiZ, PiSZ)</td>
<td></td>
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<tr>
<td>Probable and Possible</td>
<td></td>
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<tr>
<td>Infections of the respiratory tract</td>
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<tr>
<td>Allergic conditions</td>
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<tr>
<td>Bronchial reactivity</td>
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<td>Leaness</td>
<td></td>
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<tr>
<td>Socioeconomic circumstances</td>
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<tr>
<td>Alcohol intake</td>
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<tr>
<td>Diet and nutrition</td>
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<tr>
<td>ABO, ABH secretor, kell phenotypes</td>
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<tr>
<td>Impaired immune defenses</td>
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<td>Hormonal factors</td>
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<td>Familial factors</td>
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<td>Climate</td>
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</table>
and one study showed that tar content of cigarettes was unrelated to pulmonary function at baseline or 5 years later, although there was a significant relationship between the number of cigarettes smoked per day and FEV\textsubscript{1} and FVC.\textsuperscript{11} Effects of involuntary exposure to cigarette smoke have also attracted attention recently. Mean levels of ventilatory lung function are significantly lower and prevalence rates of respiratory symptoms and diseases higher in nonsmoking wives of smoking husbands, and in nonsmoking children of smoking parents.\textsuperscript{12,13} Although the effect of passive smoking appears to be small, it is important because of the frequency and involuntary nature of the exposure.

**Reduced Lung Function**

People with obstructive airways disease (OAD) may have experienced excessive decline in lung function from levels which were previously normal, or they may have started out with levels at the low end of the normal range and experienced average declines with age. The former pattern has been referred to as the horse-racing effect.\textsuperscript{7} The latter pattern, known as tracking, is a situation in which individuals tend to maintain their relative position in rank in the distribution over a period of time. In practice, both phenomena are probably important, and a combination of excessive decline plus low normal lung function to start with will obviously result in clinical impairment at a relatively young age. Incidence rates of obstructive airways disease were related inversely to initial levels of FEV\textsubscript{1}, FVC, and FEF\textsubscript{25-75} in 10- and 15-year follow-up studies of the population of Tecumseh (see below).\textsuperscript{12,13} Incidence rates were also related to other indices of lung function, such as the nitrogen index of uneven ventilation. In men but not in women, incidence rates of OAD were significantly higher in those who had the biggest increase in FEV\textsubscript{1} or FVC after inhalation of isoproterenol.

**Occupational Exposures**

Death rates for chronic respiratory diseases are higher than expected among men in certain occupations and industries. Standardized mortality ratios (SMRs) were published for 1950 in the United States, and in these and more recent data for England and Wales, ratios were high for workers in coal and other types of mining and quarrying, metal molding, manufacture of stone, glass, and clay products, and in laborers. Epidemiologic surveys have shown that work in cotton, flax, hemp, or grain dust; fire-fighting; and work involving exposure to asbestos or coke oven emissions is associated with respiratory symptoms and/or reduced lung function. Inception rates for chronic bronchitis were high in the United Kingdom for miners and quarry men, laborers, drivers of stationary engines, furnace, forge, foundry and rolling mill workers, as well as for men who worked with gas, coke, or chemicals, or who were glass or ceramic makers. Higher morbidity and mortality rates in these occupations and industries and in blue-collar workers generally are not necessarily due to harmful exposures in the workplace. In the U.K., SMRs for bronchitis tend to be high for wives as well as for the men themselves, which suggests that other factors, such as exposure to general and domestic air pollution and general standard of living, may be involved. In addition, cigarette smoking is more prevalent among blue-collar than white-collar workers. Comparisons of prevalence rates of respiratory symptoms and mean levels of FEV\textsubscript{1} in miners and nonminers in West Virginia showed that respiratory function was poorest in miners who smoked and best in nonsmoking nonminers.\textsuperscript{15} It is difficult, if not impossible, to attribute nonspecific symptoms or reduced levels of pulmonary function to specific causes or to determine the contribution which each of several factors makes to impairment of the respiratory system. Interaction between cigarette smoking and certain occupational hazards results in increased rates of COPD, and some studies have suggested that exposure to asbestos, cotton dust, and crystalline silica is more harmful to cigarette smokers than to nonsmokers. Relationships between certain occupational exposures and chronic airways obstruction were discussed by Weill at this conference.

**Air Pollution**

High levels of air pollution are harmful, especially to persons with heart or lung disease. The severity of reactions ranges from minor symptoms to premature death. But extremely high levels of pollution are rarely encountered, and the more important and unsettled question is whether long-term exposure to low levels of pollutants has a significant effect on health. Greater emphasis has been given to indoor pollutants recently because their levels may be higher than outdoor levels and because Americans spend more time indoors than outdoors. The use of new materials in the construction or renovation of buildings and increased interest in reducing ventilation to conserve energy have contributed to the potential for adverse effects of indoor pollution. Measurement of exposure to pollutants is not easy, and many studies rely on residential histories or responses to questions about exposure, or on monitoring a few pollutants at a few sites, which may not reflect exposures experienced by the human population. Some recent studies are monitoring personal or domestic levels of exposure to a few pollutants, but more studies are needed to ascertain the effect of air pollution alone and in combination with exposure to cigarette smoke or occupational hazards. More information also is needed about the effects of specific pollutants and combinations of pollutants, including active and passive smoking, emissions from gas stoves as well as from other domestic and industrial sources, and emissions from automotive vehicles. Most studies indicate clearly that cigarette smoking is a more important cause of COPD than air pollution, but there is some evidence suggesting that pollution at current levels has a small deleterious influence on respiratory symptoms and lung function; this effect may be greater on smokers than nonsmokers.

**Familial and Host Factors**

Familial and host factors may be genetic or environmental; they include simply inherited Mendelian characteristics such as protease inhibitor (Pi) type; polygenic and multifactorial traits and attributes such as pulmonary function and allergic conditions; personal behavior such as cigarette smoking; and constitutional effects of present or past illnesses and environmental exposures. The role of these factors, whether inherited or acquired, in increasing resistance or susceptibility to environmental hazards is potentially important but inadequately determined. The inherited Pi phenotype (Z), associated with severe α, antitrypsin deficiency, is implicated...
in the development of a small proportion of cases of emphysema. Heterozygotes for PIZ and other variant alleles do not seem to be at increased risk of developing COPD. The role of other genetic markers is controversial. Certain ABO, ABH secretor, and Kell phenotypes have been associated with chronic bronchitis or impaired pulmonary function in some studies but not in others. Evidence that some allergic conditions and bronchial reactivity are related to increased prevalence or incidence of COPD, respiratory symptoms or impaired pulmonary function is accumulating and warrants prospective study in several populations in which relationships with more firmly established risk factors are also being investigated. Methods suitable for use in population-based epidemiologic studies are needed for measuring these and other familial and host characteristics, including levels of immune defenses, hormonal, and other factors.

Infections of the Respiratory Tract

Patients with COPD are more susceptible to respiratory tract infections and experience higher rates of morbidity and mortality during influenza epidemics than unaffected persons. The extent to which respiratory infections contribute to the initiation of COPD is less certain, but several studies have shown that childhood respiratory illnesses may be associated with reduced lung function at older ages. Incidence rates of obstructive airways disease and chronic bronchitis were higher in those with a history of respiratory tract infections in Tecumseh; the association with chronic bronchitis was stronger. A history of "lung trouble" in childhood was associated with impaired pulmonary function in Tucson.

Other Risk Factors

Other risk factors which may be associated with COPD include leanness, nutrition, alcohol consumption, and climate. However, evidence currently available indicates that these factors are less important than the established risk factors discussed above.

Risk factors, whether major or minor, must be considered in combination as well as one at a time. Statistical methods have been used recently to identify those risk factors which have the most important effect on the incidence of disease, to assess their combined effects, and to estimate probabilities of developing disease for subgroups in the population.

Estimates of Risk From Risk Factor Profiles

Characteristics associated with increased rates of developing obstructive airways disease were identified for males and females observed over periods of 10 and 15 years in Tecumseh. Multiple logistic regression models were used to determine the extent to which onset of disease could be predicted from knowledge of a few risk factors measured at the beginning of the observation period. Variables included in the 10-year models are shown in Table 2. In both the 10- and 15-year models, age, smoking habits, and levels of pulmonary function were the most important predictors of disease. Other factors which made small but significant improvements to predicting OAD in one or both sexes, include leanness, respiratory symptoms and illnesses, socioeconomic circumstances, blood group, and respiratory disease in the family. If risk factor profiles are to be useful in practical settings such as doctors' offices and screening facilities, they should include a few risk factors which can be measured easily, accurately, and inexpensively. The profiles must also, of course, identify a large proportion of the future cases and place them in the subgroup of high-risk persons; the number of false positives should be as small as possible. The percentages of cases which were concentrated in the top deciles of the risk distribution are shown in Table 2 for some combinations of risk factors. The models which use the fewest risk factors to identify a high proportion of future cases are models 4 for men and 2 for women. Sixty-one % of male and 64% of female incidence cases will develop in the 10% of the population with the highest risk scores if age, FEV,,% predicted, and cigarettes smoked per day are used in the risk equation; 84% of male and 86% of female cases will occur in the 20% of the population at highest risk. Changes in cigarette consumption during the follow-up period also determine probability of developing OAD. If no measurement of FEV is available, prediction is substantially worse, as can be seen in models 6 and 7 for men and 3 and 4 for women. In the absence of a measurement of lung function, only 30-39% of cases will be included in the top decile of risk.

Estimates of the probability of developing OAD within 10 years can be obtained from Table 3, where points are given for each of the 3 risk factors, age, cigarettes per day, and FEV,,% predicted. The sum of these points is converted to risk of OAD in the right-hand column; this level of risk obtains if smoking habits remain unchanged. Risk (and therefore

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**Table 2—Models for Estimating Risk of Obstructive Airways Disease Within 10 Years For Men and Women Aged 25-64**

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>Models for Men</th>
<th>Models for Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Age</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Cigarettes/day</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Change in cigarettes/day</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>FEV,,% predicted</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>FVC Response to isoproterenol</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>N,%</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Weight/height,°</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Cough</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>% of Cases in top decile</td>
<td>66</td>
<td>66</td>
</tr>
<tr>
<td>% of Cases in top 2 deciles</td>
<td>84</td>
<td>79</td>
</tr>
</tbody>
</table>

Model: $p = [1 + \exp(-a - Xb)]^{-1}$

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points) will be reduced by stopping or reducing cigarette smoking and increased by increasing cigarette consumption. Thus, a range of risks (or probabilities) may be calculated and the benefits of stopping smoking demonstrated. Figure 1 illustrates the situation for 40-year-old women.

The Tecumseh index of risk has been tested and validated in other populations, but its usefulness in clinical settings remains to be determined. Risk equations do not quantify risks for individuals precisely, nor do they provide comprehensive assessments of risk for all types of smoking or FEV related morbidity or mortality. Estimates of risk apply to groups of people on the average, and are useful for ranking subgroups of the population according to their need for preventive measures. The purpose of the Tecumseh index of risk is to facilitate recognition of those at highest risk of developing COPD. Smokers should be encouraged to reduce their level of risk by stopping smoking.

In conclusion, substantial progress has been made toward meeting the "need for more studies regarding the epidemiology of emphysema," which was noted in the summary of the First Aspen Emphysema Conference. There has been progress in defining and measuring the frequency and

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### Table 3—Tecumseh Index of Risk for Obstructive Airways Disease (OAD) Within 10 Years (Expected Number of Cases Per 100 Men)

<table>
<thead>
<tr>
<th>Age (Yr)</th>
<th>Cig/Day</th>
<th>FEV₁</th>
<th>Change in Cig/Day</th>
<th>Risk of OAD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>#</td>
<td>%Pred</td>
<td>#</td>
<td>Total Points</td>
</tr>
<tr>
<td>25</td>
<td>0</td>
<td>0</td>
<td>121</td>
<td>-14</td>
</tr>
<tr>
<td>27</td>
<td>1</td>
<td>0</td>
<td>118</td>
<td>-12</td>
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<tr>
<td>29</td>
<td>2</td>
<td>0</td>
<td>115</td>
<td>-10</td>
</tr>
<tr>
<td>31</td>
<td>3</td>
<td>0</td>
<td>112</td>
<td>-8</td>
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<td>33</td>
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<td>-5</td>
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<td>35</td>
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<td>106</td>
<td>-4</td>
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<td>37</td>
<td>6</td>
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<td>-2</td>
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<td>7</td>
<td>0</td>
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<td>8</td>
<td>9</td>
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<td>19</td>
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<td></td>
<td></td>
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<tr>
<td>65</td>
<td>20</td>
<td>9</td>
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**Figure 1.** Probabilities of developing COPD within 10 years for 45-year-old men.
severity of emphysema and other members of the group of
diseases we refer to now as COPD and allied conditions. But
morbidity and mortality statistics and epidemiologic studies
do not provide enough reliable information about the fre-
quency or distribution of these diseases in persons of differ-
ent ages, sexes, races, or socioeconomic circumstances.
Knowledge of trends in morbidity and mortality over time is
inadequate; the apparent continuing rise in mortality re-
mains more attention and further investigation to determine
to what extent the rise is real and to what extent it is due
to changing fashions and practices in diagnosis and death
certification. Repeated measurements of prevalence are
needed to monitor secular changes in morbidity. Substantial
progress has also been made in identifying causes and risk
factors for COPD and in recognizing at an early stage those
who are most likely to experience illness, disability, or
premature death. The potential for applying knowledge of
risk factor profiles to prevention of COPD remains to be
realized and evaluated.

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Longitudinal Analysis of the Effects of Cigarette Smoking in
Children*
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Cigarette smoking among teenagers continues to be fre-
quent despite attempts to reduce the numbers of such
individuals who take up this habit. Although many teenagers
who begin to smoke may stop after a few years, there are few
data on the effect of such smoking on the growth of lung
infection. As part of a longitudinal study of early life risk
predictors for obstructive airways disease, we have investi-
gated the effect of short-term exposure to cigarette smoke on
the evolution of FEV1 in children aged 6–19 years.

We performed FVC maneuvers on an 8-L, water-filled
spirometer in 674 children who completed standardized
respiratory illness questionnaires for at least 2 consecutive
visits in years 4 through 7 of the study. FEV1 was obtained
using standard methods. At any given year, 85–90% of
children were <14 years old, and approximately 52% were
male. Frequency of current smoking increased from 5.7% in
year 4 to 9.9% in year 7. A Markov-type, autoregression
model was used to analyze the data. This model permits
analysis of time-dependent (eg, height, smoking) as well as
fixed (eg, sex) variables that may effect change of FEV1 over
time. Based on this model, personal smoking by the children
significantly (p<0.001) decreased the rate of increase of FEV1,
after adjustment for height in any year, annual change in
height, sex, age, and level of FEV1 in the previous year. In
particular, the level of FEV1 achieved by a smoking child after
2.5 and 5 years was predicted to be lower by 10% and 15%,
respectively, compared to that achieved by a comparable
nonsmoking child. At age 20, a child who began smoking at
age 15 would be expected to have an FEV1, 0.6 L lower than
that of a comparable nonsmoker. Data concerning the valida-
tion of the model are forthcoming.

These data indicate that relatively short periods of ciga-
rette smoking in children may have profound effects on the
rate of increase of FEV1.

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