

The Epidemiology of Chronic Respiratory Disease

IAN T. T. HIGGINS

*University of Michigan, School of Public Health,
Ann Arbor, Michigan 48104*

Chronic respiratory disease (CRD) is usually understood to include chronic bronchitis, emphysema, asthma, and a number of other chronic bronchopulmonary diseases. This paper will concentrate on the first two. The epidemiology of asthma differs in many ways from that of chronic bronchitis and emphysema, for example, in its age and sex incidence, relationships to social class, allergy, and time trends. But many of the clinical features are common to all three diseases with the result that diagnosis may be difficult. Furthermore the extent to which asthma contributes to emphysema is as yet uncertain. While it would be desirable to consider the epidemiology of the components of CRD separately this is unfortunately not yet possible. A number of conferences and organizations (2,17,104) have suggested definitions and classifications of these diseases. The following definitions, essentially those of the American Thoracic Society, would probably be generally accepted.

Chronic bronchitis is a clinical condition characterized by excessive mucous secretion in the bronchi. It is manifested by chronic or recurrent productive cough not attributable to other lung or heart disease.

Emphysema refers to an anatomical alteration of the lung characterized by an abnormal enlargement of the air spaces distal to the terminal, nonrespiratory bronchiole, usually accompanied by destructive changes in the alveolar walls.

Asthma is a condition characterized by increased responsiveness of the trachea and bronchi to various stimuli. It is manifested by widespread narrowing of the airways which changes in severity either spontaneously or as a result of treatment.

During the past 20 years epidemiologists have developed standardized methods for studying CRD (3,35,36,65). Precisely worded questions about respiratory symptoms and chest illnesses have been formulated, simple tests of ventilatory lung function, notably the forced expiratory volume (FEV) and forced vital capacity (FVC), have been widely applied, various objective methods, such as measurement of the volume and quality of the morning sputum during the first hour or half hour after rising, have been recommended (93). Bronchial reactivity has occasionally been measured. More sophisticated testing of lung function and chest radiography have been included in some surveys. The result is a considerable body of knowledge on chronic bronchitis but rather less on emphysema and asthma.

TABLE I
NUMBERS OF DEATHS AND PERCENTAGE DISTRIBUTION FROM
CHRONIC RESPIRATORY DISEASE: UNITED STATES, 1967

	United States	
	Number of Deaths	Percentage Distribution
All Causes	1,851,323	100.0
Chronic Respiratory Disease	40,443	2.2
Asthma (241 ^a)	4137	0.2
Bronchitis—chronic & unqualified (501 & 502)	5306	0.3
Emphysema without mention of bronchitis (527.1)	20875	1.1
Pneumoconiosis (523, 524)	1640	0.1
Other chronic interstitial pneumonia (525)	4219	0.2
Bronchiectasis (526)	1476	0.1
Other (527.0 & 527.2)	2790	0.2
Cancer of trachea, bronchus, & lung (162 & 163)	54,407	2.9
Tuberculosis of respiratory system (001-008)	6,351	0.3

^a International Statistical Code.

SIZE OF THE PROBLEM IN THE UNITED STATES

Thirty to forty thousand people die each year in the United States from chronic respiratory disease (Table I). This is about 2% of all deaths. It can be compared with 3% of all deaths from cancer of the bronchus and lung and 0.3% from respiratory tuberculosis. Each year there are 50-60,000 additional deaths in which chronic respiratory disease is mentioned as contributing to death. The 30,000 deaths from chronic bronchitis, emphysema, and asthma now ranks them ninth among all causes of death. Over 15,000 middle aged Americans are disabled each year by these diseases. Social Security Administration records indicate that from 1959 to 1962 they were responsible for 10% of all disability awards. Approximately 10% of all disability of one week or more is attributed to CRD. Between July, 1964 and June, 1965 roughly 2% of men and women aged 17 years and over who were interviewed in the National Health Survey reported chronic bronchitis and/or emphysema. But this must be a gross underestimate since surveys of several communities have indicated a prevalence of up to 40% depending on age, sex, and definition of disease.

TIME TRENDS

During the early decades of this century death rates from CRD in the U. S. declined rapidly. They remained low during the 1930's and 1940's. But since 1949 there has been a steady rise in the death rates from emphysema (24). More recently there has also been a rise in death rates from chronic bronchitis

in men. The rise in the emphysema rates has been noted in all States and also in certain cities (22). It has affected both men and women and white and non-white. How much this trend is real and how much it reflects changes in diagnostic fashions is debatable. But it would seem reasonable to conclude that at least some of it is real.

In certain countries, though not in the United States, there was a marked increase in deaths due to asthma during the 1960's (86, 87). In England and Wales the 5-34 age group was mainly affected with particular emphasis on those aged 10-14 years. Among them asthma mortality increased nearly eight times in seven years and in 1966 it comprised 7% of all deaths. Increases were also noted in Australia (39), New Zealand, Ireland, various western European countries and Japan. The evidence suggests that the increased mortality was due to excessive use of bronchodilator aerosols (40,54,87). The recent demonstration that excess mortality depended both on nebulizer sales and licensing of a concentrated preparation of isoproterenol (88) provides additional support for this view.

PLACE DIFFERENCES

The remarkable international differences in bronchitis mortality, which received so much attention 10 to 15 years ago are now realized to have been due largely to different diagnostic practices. A more valid assessment of these differences is obtained by combining bronchitis mortality with mortality from other diseases of the respiratory system, which excludes acute infections and upper respiratory conditions. When this is done a forty-fold difference in bronchitis between the United States and the United Kingdom is reduced to a more reasonable three- or four-fold difference in chronic respiratory disease mortality.

Within the United States there are considerable differences in chronic respiratory disease mortality. High rates occur in Arizona, Wyoming, New Hampshire, Vermont, and Florida. Differences in age distribution in these states can explain some of the difference. But climate, air pollution, occupational exposures, and migratory patterns may also contribute.

In England and Wales a close association between density of population and bronchitis mortality has long been demonstrable. But in the United States differences in mortality from chronic respiratory disease between urban and rural areas are less striking (62). They have been found for men but not for women.

FACTORS RELATED TO CRD

Age and Sex

In nearly all countries age-adjusted and age-specific mortality rates for CRD are higher in men than in women. In adult life mortality rates increase with age in both sexes. In many countries the rate of increase is greater in men than in women. In the U. S. for example the sex ratio of mortality for chronic bronchitis and emphysema increases from two to one at ages 35-44 to nine to one at ages 65-74, while at all ages combined it is five to one. Most morbidity

surveys have shown a higher prevalence of cough, sputum and wheeze among men compared with women. Much of this excess is probably due to differences in smoking habits (74). Chest illnesses during the past three years appear to be equally common in both sexes, and breathlessness is commoner in women (75).

The prevalence of breathlessness increases consistently with age but that of the other symptoms is less consistently related to age. Cough and sputum prevalence increases in some areas but not in others. It may be determined to some extent by air pollution as well as by smoking. Wheezing in the chest and chest illnesses during the past three years have been found to be inconsistently related to age.

Many studies have shown that ventilatory lung function declines with age (75,89,92). Thus from about the age of 25 there is a fairly uniform, linear decline in the forced expiratory volume (FEV) which appears to be rather similar in different countries. The regression of the one second FEV on age and height based on a number of surveys in different countries given by Cotes *et al.* (21) is

$$FEV_{1.0} = 3.62 \text{ height (meters)} - 0.031 \text{ age (years)} - 1.41 \text{ (S.D. 0.5) lBTPS}$$

The FEV in women is roughly 75% of that found in men of the same age. Most of the difference can be explained by differences in height, but some residual differences may be due to smoking or occupational exposures.

Socio-economic Circumstances

In England and Wales for many years there has been a striking gradient of increasing mortality from bronchitis with decreasing social class leading to a six-fold difference between the highest (most privileged) and the lowest classes (90). More recently in Britain a similar pronounced morbidity gradient with social class has been demonstrated (19,69). The cause of this gradient is not clear. Poor housing, domestic overcrowding with greater opportunity for cross infection, infections early in life, occupational exposures, too early return to work (especially heavy work) after respiratory infections, residence of the poor in more polluted parts of cities, differences in the quality of medical care and social drift whereby those with CRD tend to descend in the social scale are some of the suggestions which have been offered. In the U. S. the association between socio-economic level and CRD appears to be much less striking than in Britain. There is only a roughly two-fold difference in mortality between professional and unskilled workers. An inverse relationship between income level and respiratory disease has sometimes been found. Several recent surveys have also suggested that the prevalence of respiratory symptoms and chronic bronchitis may be higher and the level of ventilatory lung function may be lower in the less well-educated.

Heredity

The prevalence of chronic bronchitis is higher in the first degree relatives of bronchitic patients than in the relatives of controls (50,73,90). Estimates vary with the age and sex of the group under consideration but prevalence has

usually been about twice as high in the relatives of persons with bronchitis. In Tecumseh in addition to familial aggregation of bronchitis, levels of FEV have also been shown to be correlated in index persons and their first degree relatives. Some of this family resemblance is due to environmental rather than to genetic factors. It has been shown for example that there is familial aggregation of smoking habits (50).

More recently support for a hereditary factor has come from twin studies. Cederlof and his colleagues (13-15) have shown that concordance rates for cough, persistent cough, and bronchitis among nonsmokers are significantly higher among monozygotic than among dizygotic twins indicating a genetic influence. By comparing the prevalence of these conditions in monozygotic twin pairs, which differ in respect to such factors as smoking or urban/rural residence, with the appropriate values for the general population, an estimate of the relative importance of heredity and environment can be made. Such analyses have indicated that smoking is a more important cause of cough and bronchitis than hereditary predisposition, but that this may be important in some persons.

Deficiency of the serum protein alpha-1-antitrypsin may result in the development of emphysema at a relatively early age (29,57). The deficiency is found in less than 2% of all cases of severe emphysema. It seems unlikely therefore to be a very important factor in most cases. But it has been suggested that 7.5% of Americans or more may be heterozygous carriers of the deficiency gene. If heterozygosity is associated with an increased susceptibility to emphysema such a high frequency could be very significant. There is at present uncertainty on the point. Some studies have suggested an increased incidence of CRD among heterozygotes (60); others (80) have failed to support the suggestion. Clearly further work is needed. The importance of detecting those who are hereditarily predisposed to CRD is that particular attention can be given to them to avoid environmental determinants.

Smoking

Many surveys in many countries have shown that smoking is an important factor in CRD. These have been reviewed in considerable detail in the Health Consequences of Smoking, A Report to the Surgeon General: 1971 (92). Only a brief summary of the findings will be presented here.

Studies in which respiratory symptoms have been recorded in a standardized manner have shown that the prevalence of such symptoms is higher in cigarette smokers than in non-smokers both in men and in women. Smoking is particularly closely related to the frequency of cough and sputum, which increase progressively with increasing tobacco consumption. An effect of smoking on these symptoms has been noted at young ages (77,82), even in school children. In fact the prevalence of cough and sputum among teenagers who smoke five or more cigarettes a day may be little less than that of adult smokers (82). Wheezing in the chest usually shows a similar, though less marked, trend with cigarette consumption. The prevalence of chest illnesses and of breathlessness, while usually higher in smokers than in nonsmokers

have not been found to increase consistently with tobacco consumption. In ex-smokers the prevalence of these symptoms has usually been found to be closer to that of nonsmokers than of smokers. This is often particularly striking for cough and sputum. A lower prevalence of symptoms has also usually been found in pipe or cigar smokers.

Many studies have shown that smoking is associated with reduced lung function (1,92). In epidemiological surveys ventilatory lung function has most often been assessed, usually by means of the FEV, FVC, or peak expiratory flow. Characteristically the FEV is lower in cigarette smokers than in non- or ex-smokers, but there has often been only a small difference in the mean values between the light and heavy smokers. Usually there is little difference in the mean FVC between smokers and nonsmokers. Consequently the FEV/FVC% is especially reduced in smokers. The ventilatory capacity of ex-smokers and of pipe or cigar smokers is usually only slightly lower than that of nonsmokers. Lung volumes (7), airways resistance and transfer factor have been included in a few epidemiological surveys. They have shown that these other aspects of lung function are also lower in smokers than in nonsmokers. The lower lung function values found in smokers are mainly associated with the presence of respiratory symptoms; but even in those without symptoms smoking has sometimes been shown to be associated with lower ventilatory function (77,100).

Relatively few studies of trends in lung function over time according to smoking habits have been made (34,48,99). The findings from a nine year follow-up of a survey of a representative sample of men living in Staveley, Derbyshire, an English industrial town, are shown in Table II. The mean values at the initial survey in 1957 support the statements, which have already been made. The decline in FEV over the nine years is related to the smoking habits which were recorded at the first survey. There was a higher decline in smokers than in nonsmokers and in heavier compared with lighter smokers. The decline was intermediate in ex-smokers and in smokers of pipes or cigars. In this study there was also a somewhat higher attack and a lower remission rate of respiratory symptoms among smokers compared with non-smokers.

A wealth of information on smoking and mortality is available from the prospective studies which have been carried out on smoking and lung cancer (92). These show that death rates from bronchitis and emphysema rise with increasing cigarette consumption. Among British doctors (23) the mortality ratios for chronic bronchitis among those smoking 25 cigarettes a day and over were over 20 times as high as that of nonsmokers. Among U. S. veterans (56) ratios for chronic bronchitis were eight times and for emphysema 25 times as high in those who smoked 40 cigarettes or more daily as in nonsmokers. Relatively little information on smoking and CRD mortality is available for women. Hammond (47) found mortality ratios for emphysema in lighter and heavier smokers of nearly five and over seven times that for non-smokers respectively. But the number of deaths from emphysema among women was small. There is general agreement in all these studies that the mortality ratios for CRD in smokers of pipes or cigars are only slightly above those for non-smokers.

TABLE II
 MEAN FEV_{0.15} AND CHANGE IN FEV IN NINE YEARS IN THOSE FOLLOWED-UP IN STRATIFIED SAMPLE OF STAVELEY IN 1957
 MEN AGED 25-34 AND 55-64

Age in 1957	Smoking habits in 1957	Complete sample 1957				Those sampled in 1957 & seen in 1966				Mean FEV over the 9 years	Mean change in 9 years	Mean annual decline
		No.	Mean	SD	No.	1957		1966				
						Mean	SD	Mean	SD			
25-34	Nonsmokers	56	3.63	.60	49	3.66	.59	3.45	.56	3.56	.21	.023
	Exsmokers	34	3.49	.45	31	3.52	.46	3.26	.48	3.39	.25	.028
	Smokers: 1-14 cig/day	132	3.54	.62	125	3.52	.63	3.19	.67	3.35	.33	.037
	15 or more cig/day	133	3.37	.48	108	3.35	.43	3.00	.47	3.17	.35	.039
	Other	14	3.40	.45	13	3.37	.45	3.03	.48	3.20	.34	.038
55-64	Nonsmokers	30	2.54	.55	25	2.60	.45	2.32	.56	2.46	.28	.031
	Exsmokers	64	2.24	.59	49	2.35	.51	2.02	.60	2.18	.32	.036
	Smokers: 1-14 cig/day	126	2.05	.71	79	2.22	.61	1.78	.59	2.00	.44	.049
	15 or more cig/day	100	2.01	.60	61	2.07	.56	1.55	.66	1.81	.53	.059
	Other	67	2.23	.63	46	2.34	.58	1.97	.63	2.16	.37	.041

A lower mortality from CRD in those who gave up smoking has been noted in several of the prospective studies. In U. S. veterans the age-specific mortality ratios of ex-smokers were lower than those of continuing smokers of similar amounts. In British doctors a reduction in bronchitis mortality was observed only after a delay of some five years. In recent years British doctors have shown a more favorable trend in bronchitis mortality than other men aged 35-64 in the U. K. Between 1953-1957 and 1962-1965 their rates fell by 22% compared with a fall of only 4% in all men of this age.

The benefit of stopping smoking has been noted for many years by clinicians (55). Systematic observations have been rather limited (70,105). A reduction in cough and sputum has usually been the most striking change. But a reduction in the number of exacerbations and of time lost from work have also been reported. In younger persons lung function may return to normal. The prevalence of persistent sputum has been shown to be somewhat lower in those who smoke filtered than nonfiltered cigarettes. Furthermore a recent controlled trial has supported the conclusion that switching to an effectively filtered brand of cigarettes would be beneficial (38). There is also evidence to support the view that respiratory symptom prevalence is higher in those who inhale moderately or deeply than in those who do not inhale or who inhale only slightly.

Randomized trials of intensive antismoking advice have been started in Britain, but no results have yet been published. Similar trials are needed in this country to appraise the efficacy and value of these efforts.

Air Pollution

Evidence from a wide variety of epidemiological investigations (Table III) suggests that air pollution is a significant factor in CRD. There is no doubt about the effect of exceptional episodes of pollution such as those in the Meuse Valley in 1930 (32), Donora in 1948 (83), London in 1952 (67) and other years and New York City in 1953, 1963 (44), and 1966, on mortality and morbidity in general and from respiratory disease in particular. The effect does however seem to have been less in New York than in London. Much of the increase which occurred seems to have been in those who already had chronic disease of the heart or lungs (18,67). But involvement of young persons, even of infants, and of domestic and other animals, and the development of symptoms as pollution increased suggest that fog can initiate as well as exacerbate disease.

There has been more doubt about the effect of lower, more sustained levels of pollution on respiratory disease. In London daily concentrations of smoke and SO₂ were fairly highly correlated with mortality and morbidity from all causes and from certain respiratory diseases from 1958 to about 1962. But since then there has been much less evidence of any such correlation. This is no doubt due to the remarkable decline in the concentrations of smoke and SO₂ which have occurred during the past 12 years in London (81). Even in especially susceptible subjects, such as patients with bronchitis or emphysema, it is no longer possible as it used to be to correlate respiratory well being with daily pollutant levels (95-97). On the other hand in a study of men aged 30-59

TABLE III
 MAIN TYPES OF EPIDEMIOLOGICAL INVESTIGATIONS
 INTO THE EFFECTS ON HEALTH OF AIR POLLUTION

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1. Acute Episodes
 2. Follow-up of Donora, Pennsylvania
 3. Day to Day Variation in Mortality, Morbidity, and Lung Function
 - (i) General Community
 - (ii) Patients with Chronic Respiratory Disease
 4. Longitudinal Observations at Less Frequent Intervals
 5. Geographical Comparisons
 - (i) International
 - (ii) Within Countries by Region
 - (iii) Urban/Rural Differences
 - (iv) Uniform Occupational Groups
 - (v) Comparisons Between Towns
 - (vi) Comparisons Within Towns
 6. Studies of Children
 7. Miscellaneous Studies
 - (i) Migrants
 - (ii) Twins
 - (iii) Athletic Performance
-

in north London, Fletcher was able to show that the incidence of a number of respiratory illnesses was related to both smoke and SO_2 concentration (4). A decline in the volume of morning sputum was also noted; but this could have been due to a concurrent reduction in the tar content of cigarettes. In this country McCarroll and his colleagues (63) followed a segment of the population of Manhattan Island for two years. Complaints of cough and of eye irritation were found to be related to SO_2 concentrations. Among bronchitis patients in Chicago the severity of symptoms has been shown to depend to some extent on pollution levels but there is some doubt about the extent to which this can be attributed to SO_2 concentration (10,12).

In a study of Canadian veterans Bates (5,6) and his colleagues found that patients with chronic bronchitis in Winnipeg were less severely affected and had better lung function, which declined less during the four years of observation, than patients in Toronto, Montreal, and Halifax. Winnipeg is much the least polluted of the four cities in terms of dust fall and SO_2 levels.

Many geographical comparisons relating pollution to indices of respiratory disease have been carried out (43,51,52,59,69,71,72,76,78,79,98). Some of the international differences in CRD mortality can be explained by differences in death certification. But even when these are allowed for and differences in smoking habits are also considered death rates from CRD are appreciably higher in Britain than in the United States or Scandinavia (16,81). Carefully

standardized studies of morbidity have supported conclusions based on death rates (52, 71, 72, 79). Thus while the prevalence of cough and sputum in Berlin, N. H. was similar to that found in rural and urban Britain, bronchitis with recurrent illnesses and breathlessness was commoner in Britain, especially among older people in larger towns. In a carefully standardized comparison of CRD in unpolluted Chilliwack, B. C. and moderately polluted Berlin, N. H., Ferris and Anderson (30) noted that ventilatory capacity was consistently above expectation in both men and women for all smoking categories in Chilliwack. Mortality and morbidity from respiratory disease has often been related to levels of air pollution in Britain. Earlier studies often had to use indirect measures of pollution, such as fuel consumption per acre. More recent studies have more often used measurements of smoke or SO₂ collected through the National Air Sampling Network. Whatever the index of pollution used, these studies have nearly all shown a fairly close relationship between level of pollution and bronchitis mortality and morbidity. To get round the problem of the association of pollution levels with social class the uniform occupational group has sometimes been used. Fairbairn and Reid studied postmen (mailmen) who work in different parts of the country, which vary widely in levels of pollution. Premature retirement and death from bronchitis was highest among postmen in more polluted areas.

In this country extensive studies of air pollution have been made in Nashville, Tennessee (106–108) and Buffalo, New York (101–103). In Nashville respiratory disease mortality was related to sulphation levels and various indices of morbidity, such as frequency of asthmatic attacks, to sulphation and soiling. In Buffalo pollution was monitored extensively from 1961 to 1963. Levels of pollution were related to mortality from 1959 to 1961. Mortality from all causes in men aged 50 years and over and respiratory disease mortality in men aged 50–59 was correlated with suspended particulate concentrations (Table IV). There was also a correlation between sulphation levels and respiratory disease mortality in men aged 50 and over. In a subsequent study of women the prevalence of cough and sputum was shown to be related to suspended particulate concentrations.

Some of the most significant effects of pollution on the respiratory system have been demonstrated in children. A higher prevalence of sinus infection, chronic otitis media (94), respiratory symptoms and chest illnesses, and lower ventilatory lung function (61) as assessed by FEV, FVC, and peak flow rates, have been associated with higher levels of pollution. One of the most interesting studies was that conducted by Douglas and Waller (25). Illness experience was related to levels of lifetime pollution experienced by a national sample of children born in 1946 and studied intensively until they left school in 1961. The findings were clear and consistent. Lower respiratory illnesses were related to air pollution; upper respiratory illnesses were not. The lowest illness rates were found in those who had experienced the lowest levels of smoke and SO₂, namely an average annual concentration of 70 and 90 µg/m³. Higher illness rates were found in all higher pollution categories. The higher rates did not appear to be explainable on the basis of social class.

TABLE IV
MEAN ANNUAL DEATH RATES PER 100,000 POPULATION FROM ASTHMA,
BRONCHITIS OR EMPHYSEMA ANYWHERE ON THE DEATH CERTIFICATE
BY ECONOMIC STATUS AND POLLUTION LEVELS^a

Median family income for each census tract (\$)	Mean annual particulate concentration ($\mu\text{g}/\text{m}^3$)			
	<80	80–	100–	135+
3005–5007	–	126	271	392
5175–6004	136	154	172	199
6013–6614	–	74	110	128
6618–7347	70	80	177	–
7431–11,792	79	109	0 ^b	–

^a White Males Aged 50 to 69 Years in Buffalo, New York, 1959–1961. Winkelstein *et al.*, (1967).

^b Rate based on less than 5 deaths.

Evidence which permits one to recommend standards for particulate and SO_2 concentrations is scanty. This study suggests that the maximal permissible annual concentrations of smoke or of SO_2 should both be under $100 \mu\text{g}/\text{m}^3$. Support for this statement can be drawn from the nationwide Enquiry into the Incidence of Incapacity from Bronchitis and from the random sample of Britain studied by Lambert and Reid. In Buffalo the findings on mortality suggested that an annual concentration of suspended particulates of less than $80 \mu\text{g}/\text{m}^3$ would be desirable.

OCCUPATIONAL EXPOSURES

During the 19th century in Britain authorities on respiratory disease had no doubt about the importance of occupational exposures as a cause of bronchitis. Thackrah and Greenhow (45) stressed the importance of dusts, particularly textile dusts, while in this century Collis (20) considered that bronchitis was chief of the dust diseases. But both Collis and Haldane pointed out that dusts varied in their effect according to their nature (42). In the mortality statistics given by Collis, cotton strippers stood out with a nearly six-fold higher mortality rate for bronchitis than all men whereas none of the other occupations he cited even reached a two fold excess. Haldane (46) reviewing the same material noted high rates among tin and copper miners and sandstone workers as well as cotton strippers and grinders. Coal miners in England and Wales have had consistently higher death rates from bronchitis than other men since 1900. But the excess has not been large, approximately 40% in most censuses. Furthermore the rates in the different coal fields has varied from less than the national average for all men of working age to approximately twice the average.

In the United States occupational mortality statistics are available only for 1950 (27). The high standardized mortality ratios (SMR) for mine operatives

and labourers and metal moulders are striking. The much higher ratios for coal miners in the U. S. (nine times the average for all men aged 20-64) compared with coal miners in England and Wales (1.4 times the average) is largely due to the inclusion of pneumoconiosis in the American statistics. If deaths from this cause are excluded the excess chronic respiratory disease ratios become much more similar in the two countries. The interpretation of mortality statistics is difficult. Apart from questions about the accuracy of diagnoses, discrepancies between the census and death populations can lead to serious biases which make conclusions about the effect of probable levels of dustiness on mortality exceedingly hazardous. The higher mortalities from bronchitis among the wives of men with higher bronchitis SMR also suggests that some of the excess may be due to social rather than to occupational factors.

In Britain morbidity statistics (68) available since 1948 have indicated that miners and foundry workers have higher rates of sickness absence from bronchitis than other men. How much this can be explained on the basis of the heaviness of the job is debatable. A survey of a random sample of all workers carried out in 1961/1962 confirmed the high rates for attacks of bronchitis in miners. It also showed above average incidence of bronchitis in furnace, forge, foundry men, gas, coke and chemical workers, and various other groups; but the excess in these occupations was not striking.

Field surveys in which representative samples of the community, some of whom work or have worked in the occupation under investigation and others have not, have been carried out in various countries during the past 20 years (26,28,48,58,84,85). A summary of the findings in those which have used comparable methods is shown in Table V (42). The results are fairly consistent. In almost all surveys, there was a higher prevalence of bronchitis and a lower mean ventilatory capacity among the men who were exposed to dust. The differences between dust-exposed and nondust-exposed groups varies in the different surveys from 1:1 to 6:1, but is most often around 2:1. With few exceptions the differences in mean ventilatory capacity between the two groups support the view that those exposed to dust were more disabled. These differences cannot be explained on the basis of differences in smoking habits since they have been shown to persist after standardization for smoking. Their relation to dust exposure is not clear. Some studies have suggested that as lifetime dust dosage, assessed either by the number of years spent working underground or at the coal face, increases, the prevalence of CRD increases and the level of ventilatory capacity declines (53,58,91). But other studies have indicated that at least in some areas any such trends may be inconsistent and that often they cannot be demonstrated at all (49).

Another reason for concluding that lifetime dust exposure cannot be the whole explanation of the differences in CRD between dust-exposed and nondust-exposed persons is that in coal miners there is no close relationship between category of simple pneumoconiosis and either the prevalence of bronchitis or level of ventilatory capacity (42). On the other hand, a close relationship between X-ray category of simple pneumoconiosis and the total mass of coal dust which is found in the lung at autopsy has been clearly demonstrated.

TABLE V
POPULATION SURVEYS OF BRONCHITIS AND VENTILATORY CAPACITY OF MEN WORKING IN DUSTY OCCUPATIONS

Country	Author	Industry	Age	No. in samples				Bronchitis %				Ventilatory capacity		
				Dusty	Control	Dusty	Control	Dusty	Control	D/C	Ratio	Dusty	Control	Difference
														D-C
England Leigh	Higgins <i>et al.</i> (1956)	Coal mining	55-64	132	84	23.5	10.7	2.2	2.17	2.39	-0.22			
Staveley	Higgins <i>et al.</i> (1959)	Coal mining Foundry work	55-64	149	81	20.8	14.8	1.4	2.44	2.52	-0.12			
Staveley	Higgins (1970)	Coal mining Foundry work	25-74 ^a 25-74 ^a	179 97	159 159	7.8 17.4	14.8 2.7	0.5 6.4	2.36 2.56	2.52 2.84	-0.16 -0.28			
Great Britain	Lloyd Davies (1970)	Foundry work	35-64	1770	1730	10.6	7.2	1.5	?	?	-0.05 ^b			
Wales Rhondda	Higgins <i>et al.</i> (1961)	Coal mining	35-64 ^a	275	262	28.5	9.2	3.1	2.53	2.87	-0.34			
USA (West Va) Marion Co.	Higgins <i>et al.</i> (1968)	Coal mining	20-79 ^a	425	399	4.8	4.0	1.2	3.41	3.53	-0.12			
Mullens	Enterline (1967)	Coal mining	21-64 ^a	225	224	13.0 ^c	6.5 ^c	2.0	3.01	3.18	-0.17			
Richwood	Enterline (1967)	Coal mining	21-64	175	153	8.9	9.1	1.0	3.29	3.28	+0.01			
Canada	Parsons <i>et al.</i> (1964)	Fluorspar mining	20-70 ^a	301	56	21.3	4.0	5.3	2.86	3.09	-0.23			
South Africa	Sluis-Cremer <i>et al.</i> (1967)	Gold mining	35 & ^a over	546	263	10.6	3.8	2.8	3.12	3.27	-0.15			
Northern Ireland	Pemberton <i>et al.</i> (1968)	Flax workers	35 & ^a over	262	594	12.6	5.6	2.3	N.A.	N.A.	N.A.			

^a Age adjusted to the total population

^b Given by Gilson (1970)

^c Cough and Sputum for 3 months or more.

Exposure to certain vegetable dusts is perhaps more liable to cause CRD than exposure to coal dust. No doubt some of the high mortality rates noted in the past among flax hecklers and cottonstrippers was due to the specific disease byssinosis, but more recent work has shown fairly convincingly that such workers also experience an increased prevalence of bronchitis as well as of byssinosis (26). Exposure to cotton, hemp, and flax dust has been shown to be associated with a decline in ventilatory capacity during the working shift (8,9,11,41,64). The decline is greatest on Mondays and becomes progressively less during the week. It is also usually greater in persons with byssinosis, but it may also occur in those without symptoms. The extent to which an acute response to the dust leads to permanent disability is at present uncertain. In contrast to this well marked acute effect on lung function of vegetable dusts exposures, coal dust exposure does not as a rule lead to any comparable decline. From the preventive standpoint it would seem reasonable to conclude that while some occupational dust exposures may be more harmful than others no exposure is likely to be beneficial. Adequate control of dust at work is clearly desirable.

INFECTION

Differences between persons with chronic bronchitis and other people in the frequency of certain respiratory infections have sometimes been noted. Bronchitic subjects report a higher frequency of pneumonia, pleurisy and acute bronchitis in the past; a higher prevalence of stuffy nose and sinus infection at the time of interview and a higher frequency of head colds, particularly of headcolds which "go down on the chest." Some of these conditions appear to date back to childhood and it may well be true, as Reid has maintained, that the seeds of CRD are sown early in life.

The degree to which the characteristic exacerbations of established CRD are due to infective agents is not yet certain. Fisher and her colleagues (33) in Edinburgh, Scotland showed that while there was a significant increase in bacterial (particularly pneumococci and H-influenzae), viral and mycoplasmal pathogens during exacerbations compared with quiescent periods, infective agents could be isolated in less than half the exacerbations. It seemed to them therefore that viral and mycoplasmal infections could not play a large part in exacerbations of bronchitis. Rather surprisingly infection does not appear to have as large an influence on the natural history of CRD as was generally supposed ten years ago. In the longitudinal study of chronic bronchitis conducted by Fletcher (33) in London from 1962 to 1966 neither exacerbations, nor volume or purulence of sputum appeared to influence the rate of decline of the FEV.

Support for this conclusion comes from controlled prophylactic and therapeutic clinical trials of antibiotics in chronic bronchitis (89). Most of these have been carried out on advanced cases. But a few trials (37,66) have been specifically directed at early cases. Such a trial was that carried out in Britain by the Medical Research Council. The trial moreover lasted for five years and included patients from a wide area of the country. The design included an assessment of both prophylactic and therapeutic possibilities. While chemo-

therapy reduced the time lost from work by about a third and appeared to reduce the number of patients who had many exacerbations, it did not influence either the volume or the purulence of the sputum nor did it affect the rate of decline in the FEV.

LESSONS FROM EPIDEMIOLOGY FOR PREVENTION

Epidemiological studies have shown clearly the overwhelming importance of smoking, particularly cigarette smoking, in the causation of CRD. There is a consistency about the findings in respect to smoking which is not present for the other major environmental factors. Apart from the direct effect it may have on the respiratory tract, interaction, which has sometimes been noted between smoking and air pollution or smoking and occupational dust exposure, suggests that smoking may act indirectly to modify the effect produced by these other determinants. Clearly the major emphasis in any program of prevention of CRD must be on smoking. Don't take it up, give it up, switch to a pipe, to cigars, or to more adequately filtered cigarettes, don't inhale—these are the obvious recommendations in order of preference.

At the same time when so much is involved both economically and from the standpoint of personal pleasure it is important to make every effort to measure what can be accomplished by persuasion or exhortation. The only satisfactory way of doing this is by randomized controlled trials. A start has been made along these lines; but much more remains to be done.

Reduction in air pollution is obviously desirable on many grounds, not merely because of its effects on health. The remarkable reduction in air pollution, particularly of smoke pollution, which has been brought about in Britain by the implementation of the Clean Air Act shows what can be accomplished by legislative action. Strict enforcement of National Air Quality Standards proposed for the United States should go far towards eliminating any effect of air pollutants. Monitoring the effect of reduction in pollution on CRD is highly desirable. Here because of the general implementation of the law controlled trials seem to be impossible. Instead we shall have to be content with observation of mortality and morbidity before, during, and after pollution reduction, or with small scale observations based on repeated observations in communities, which have reduced pollution such as that carried out by Ferris and his colleagues (31). The Environmental Protection Agency has made a good start with its Community Health and Environmental Surveillance System (CHESS). The observations being made should permit a more accurate assessment of the effects of specific pollutants and of critical levels than has previously been possible. Occupational exposures will no doubt be much more stringently controlled in the future as a result of the Occupational Safety and Health and Mining Health and Safety Acts. The legislation that miners must be offered examinations by chest X-ray and lung function tests should provide the needed evidence on the efficacy and adequacy of dust standards and at the same time identify any man who may be unduly sensitive to dust. It is not hard to foresee the extension of these requirements to other potentially hazardous industries.

Prompt and adequate treatment of acute respiratory diseases is clearly indicated from the reduction in lost working time which has been shown to result. That such therapy has not appeared to influence the rate of decline in lung function and so presumably has no very large effect on the natural history of the disease is disappointing. It does however emphasize that prevention, rather than early detection and treatment, is the only effective approach to CRD.

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