

Socioeconomic status and chronic obstructive pulmonary disease

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Socioeconomic status, whether measured by education, income, or other indices of social class, has long been known to be associated with mortality from different diseases.¹⁻⁷ This knowledge for the major part stems from studies conducted in the UK where there is a tradition of interest in socioeconomic differences. A milestone within recent years was the "Black Report" prepared by a committee appointed by the UK Minister of Health in 1980.⁷ This report showed that there were still large socioeconomic differences in morbidity and mortality in men and women of all ages in England. Since then a number of epidemiological studies have been published, both in England and other European and North American countries, which confirm this relationship.⁸⁻¹²

Socioeconomic differences have been shown to exist for a number of diseases including ischaemic heart disease, many types of cancer, respiratory diseases and, in particular, mortality related to alcohol and violence. In addition, despite a general fall in mortality during recent decades, the relative disadvantage of low socioeconomic status continues to increase.³⁻⁷ The number of papers published on socioeconomic factors and morbidity and mortality is increasing sharply.¹³ The social gradient in ischaemic heart disease has been extensively studied but, although there are indicators that socioeconomic differences in respiratory diseases are greater still,² very few studies have focused on this disease entity.

Review of current literature

A social class gradient in the prevalence of respiratory symptoms in adults, in particular cough and sputum, was first observed in early studies in the UK.¹ This has later been confirmed in other studies, most of which date from the 1970s.^{2,14-26} The relationship between socioeconomic factors and symptoms, lung function parameters, and respiratory morbidity in studies in adults is summarised in table 1. Most of these studies have adjusted for smoking habits since smoking is related to both the disease and socioeconomic status.

Only a few studies have attempted to quantify the effect of socioeconomic status on indices of lung function. In a study comparing mortality in manual and non-manual workers in two British towns, respiratory symptoms and impairment of lung function was found to differ across social class.¹⁹ The difference in forced expiratory volume in one second (FEV₁) was 7-15% of FEV₁% predicted and was not affected by stratification by smoking history. In a study of 410 male non-smokers, Stebbings *et al*¹⁴ found that the difference in FEV₁ between the highest and lowest social class was

400 ml.¹⁴ A similar result was found in the Copenhagen City Heart Study in which the difference between the highest and lowest socioeconomic group was 400 ml for men and 259 ml for women after adjustment for smoking.²⁵ This difference was reflected in a threefold higher risk of admission to hospital for chronic obstructive pulmonary disease (COPD) in subjects of the lowest compared with the highest socioeconomic group, and was not affected by adjustment for smoking. Interestingly, similar risk ratios for death from respiratory disease were seen in the Whitehall study.²

Most of these studies have been conducted on middle aged adults and it is worthwhile to remember that lung function in this age group may be the result of several mechanisms.²⁷ A reduced lung function in middle age may result from a low maximally attained lung function due to either low lung function at birth or decreased growth of lung function during childhood and adolescence, a shortened plateau phase during early adult life, or an increased decline in lung function in later adult life. The important unanswered question remains: which risk factors linked to socioeconomic status are causally related to the occurrence of COPD? The link is likely to be multifactorial and includes prenatal exposures, more frequent lower respiratory tract illness in childhood, housing conditions, air pollution, environmental tobacco smoke, diet, and other lifestyle factors including smoking, in addition to possible genetic factors. As these factors may or may not be directly associated with socioeconomic status, they will be briefly discussed in more detail. A list of the factors of interest in this review is given in table 2.

As for prenatal exposure, the possibility that intrauterine growth of the fetus might be related to health in childhood and adult life has attracted considerable attention.²⁸⁻³⁵ Most studies concern ischaemic heart disease but a link between prenatal exposure and respiratory disease has also been suggested.³³⁻³⁶⁻³⁷ Low birth weight has been associated with reduced lung function, respiratory infections, and asthma in children and adults³⁸⁻⁴⁰ regardless of length of gestation⁴¹ and whether the child had respiratory complications at birth.⁴²⁻⁴³ This gap may continue to widen into adult life as suggested by Barker and co-workers³⁴ who reported that, among men born 60-70 years ago in Hertfordshire, UK, those who had a lower birth weight had a lower FEV₁ adjusted for age and height. The standardised mortality rate for COPD was also associated with birth weight in these men. This was corroborated by a study on Indian men and women in which

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Table 1 Studies of socioeconomic status (SES) and indices of COPD

Author/place	Year	Study population	Index of SES
Studies of adults that include measures of lung function			
Stebbins ¹⁴ (Hagerstown, Maryland)	1971	Non-smokers (410 men)*	Income, education
Cohen ¹⁶ (Baltimore, Maryland)	1977	Genetic-epidemiological (1232 men and women)*	Place of residence
Higgins ¹⁷ (Tecumseh, Michigan)	1977	Population sample (4699 men and women)*	Education, income
Lebowitz ¹⁵ (Tucson, Arizona)	1977	Population sample (3800 men and women)*	Education, income, occupation
Rasmussen ²⁶ (Copenhagen, Denmark)	1978	Non-occupationally exposed (218 men)*	Housing conditions
Krzyzanowski ¹⁸ (Cracow, Poland)	1986	Population sample (1864 men and women)**	Education
Burr ¹⁹ (Caerphilly/Bath, Scotland)	1987	Population sample (513 men)*	Manual, non-manual
Krzyzanowski ²⁰ (7 cities, France)	1988	Population sample (16 000 men and women)*	Social class, education
Bakke ²² (Bergen, Norway)	1995	Population sample (714 men and women)*	Education (3 levels)
Hole ²³ (Renfrew/Paisley, Scotland)	1996	Population sample (15 411 men and women)*	Social class (IV–V/I–III)
Prescott ²⁵ (Copenhagen, Denmark)	1998	Population sample (14 223 men and women)*	Income, education
Other studies of socioeconomic status and indices of COPD			
Marmot ² (Whitehall study, UK)	1984	Civil servants (17 530 men)**	Grade 1–4
Lebowitz ²¹ (Tucson, Arizona)	1990	583 children aged over 6*	Family income
Demissie ²⁴ (Montreal, Canada)	1996	989 children aged 6–11*	Parental SES score 1–5

*Cross sectional study; **longitudinal study.

mean FEV₁ fell by 0.09 litres with each pound decrease in birth weight in men and by 0.06 litres in women, independent of smoking.²⁸ These results are supported by animal experiments which show that lung structure may be permanently altered by calorie or protein deprivation in utero.^{44–46} Changes include reduction of elastin or collagen in the lung, enlargement of air spaces, and a reduction in elastic recoil compared with that seen in human emphysema.^{47–50}

The theory of “intrauterine programming” has been criticised.⁵¹ In addition, whereas intrauterine growth retardation because of malnourishment during pregnancy associated with low socioeconomic status may have been common more than 50 years ago, this is not likely to be a major problem in western countries today. However, today relative growth retardation may be seen in children of smoking mothers. In the Tucson studies it has been shown that children of mothers who smoke during pregnancy have impaired lung function in infancy and are also at increased risk of developing asthma,⁵² and Hanrahan *et al* have found several measures of infant lung function to be negatively affected by in utero exposure to tobacco smoke.⁵³ Whether differences continue and affect lung function into adult life remains to be seen.

Another factor related to socioeconomic status is respiratory infections during infancy. Childhood experience of respiratory disease was associated geographically with mortality from chronic bronchitis.³⁷ Early respiratory tract infections have been shown to be associated with reduced lung volumes independent of the effects of low birth weight.^{29 37 54 55} Studies of the association have consistently shown deficits of lung function of the order of 5–10%.⁵⁶ However, because of

methodological problems it is still not clear whether the relationship is causal or is merely an epiphenomenon in the mesh of associations between lung function at birth, respiratory tract infections with or without wheeze, atopy, asthma, and growth of lung function as recently discussed by Britton and Martinez.⁵⁶ Not only childhood respiratory infections but also infections in adulthood have been linked to impairment of lung function.^{57 58}

Poor housing conditions²⁶ and home dampness^{59 60} with increased house dust mites⁶¹ and gas stove usage^{62 63} are all associated with respiratory symptoms, reduced lung function, and lower socioeconomic status. Household crowding has been hypothesised to cause increased instances of respiratory infections and thus increased rates of respiratory disorders,^{32 64} although this was not confirmed in the Tucson study.²¹ Home dampness and respiratory symptoms are strongly associated, whereas the relationship between home dampness and pulmonary function is weaker in both children and adults.^{59 60 65} Children living in homes using gas for cooking are more likely to have had respiratory illnesses than those from homes using electricity in some^{66–68} but not all studies.^{69 70} In adults the use of gas for cooking was related to an increased frequency of respiratory symptoms and impaired ventilatory function among men but not among women in two studies.^{62 63} Exposure to community air pollution is an additional risk factor to the airways more often experienced by subjects of lower than of higher social class.

Smoking remains the most prominent cause of lung disease. Children from homes with smokers are more likely to have respiratory illnesses than those not exposed to tobacco smoke at home.^{71–74} Smoking in childhood and adolescence affects growth of lung function.⁷⁵ Gold *et al* clearly demonstrated this in the US Six Cities Study where a relatively modest amount of tobacco led to marked reductions in growth of lung function, particularly in teenage girls who in most western countries seem to constitute a group at high risk of persistent smoking. There is no doubt that there is a socioeconomic gradient in smoking behaviour and that this gradient is increasing. A higher

Table 2 Factors potentially associated with socioeconomic status and lung function

Intrauterine growth of lung function
Environmental exposures in childhood
Childhood respiratory infections
Smoking in childhood and adolescence
Smoking in adulthood
Occupation
Housing
Imbalance in protective factors

Outcome	Adjusted for	Result
FEV ₁ , symptoms		Association between SES and FEV ₁ but not symptoms
FEV ₁	Smoking, sex	22.3/7.2% in low/high quintile SES had poor FEV ₁ (p<0.001)
FEV ₁ , symptoms	Smoking	Weak associations with both symptoms and FEV ₁
FEV ₁ , symptoms	Smoking	All indices of SES were associated with both symptoms and FEV ₁
FEV ₁	Smoking	FEV ₁ reduced 10 ml per year without central heating
FEV ₁ , symptoms	Smoking, occupation	Inconsistent associations
FEV ₁ , FVC, symptoms	Smoking	200–300 ml difference in FEV ₁ , symptoms associated with SES
FEV ₁ , FVC, symptoms	Smoking, occupation	Significant association with FEV ₁ and some symptoms
FEV ₁ , FVC, symptoms	Smoking, sex, occupational exposure	COPD OR 4.2 Spirometric airflow limitation OR 6.3
FEV ₁ , quintile	Not adjusted	15/26% of low SES in low/high quintile of FEV ₁
FEV ₁ , hospital admission	Smoking	300–400 ml difference in FEV ₁ , hospital admission RR 3
Cause-specific mortality	Unadjusted	Chronic bronchitis RR 7.3; other respiratory RR 3.1
Chronic bronchitis		Income associated with chronic bronchitis
FEV ₁ , FVC	Multiple	Boys FEV ₁ and FVC 8% lower, girls no difference

success rate in quitting is also associated with higher social class.^{76 77}

Social class, level of education, and occupation are closely correlated, and some of the social gradient in respiratory diseases may be caused by occupational exposure. The extent of confounding by occupation in epidemiological studies will depend strongly on the spectre of occupational exposure in the population under study. However, several findings indicate that confounding by occupational exposure does not fully explain the association. Women generally do not hold jobs with major exposure to dusts and fumes that potentially cause respiratory disease, yet socioeconomic differences in lung function are found in both sexes,²⁵ although most studies indicate that they are smaller in women.^{22 78 79} Furthermore, socioeconomic differences seem to exist at all ages—that is, they are present before accumulated occupational exposure has had time to cause an impairment of lung function.^{22 25 80} Finally, it was elegantly shown in a Norwegian study that education remained a significant risk factor for symptoms of COPD after adjustment for occupational exposure.²² In this study occupational airborne exposure status was assessed by self-reported past and present exposure to dust or gas as well as by classification of jobs held since leaving school. After adjustment for occupational and smoking exposure, the odds ratio of obstructive lung disease in primary educated versus university educated subjects was 2.9 (1.3–6.5) and the corresponding odds ratio for spirometric airflow limitation (dichotomised) was 5.2 (2.0–13.4).

Damage of alveolar tissue and small airways seems to be mediated by oxidants. Smoking profoundly affects the oxidant/antioxidant balance and increases oxidative stress, but antioxidants in fresh fruit and fish oils may offer some protection against such damage.^{81–89} This may help to explain some of the increased susceptibility related to social class. Antioxidants in wine could hypothetically have a protective effect on the lungs, judging from its effects on overall mortality and lung cancer.^{90 91} However, this association, which would influence the association between socioeconomic status and lung disease, has not so far been addressed in

detail. In fact, a risk factor of marginal importance is heavy alcohol consumption which is associated with airflow limitation.⁹²

Other possible contributory explanations that need consideration include selective drift—that is, that subjects with chronic lung diseases are downwardly mobile. In recent years a number of studies have attempted to clarify these causal relations.^{13 93} The socioeconomic gradient seems to be only slightly affected by selective drift or access to health care.^{94 95} In addition, a gradient is also found for education which, in contrast to income and social class, precedes morbidity.

Conclusion

The socioeconomic gradient in COPD is as great, if not greater, than in any other disease. The impact of socioeconomic status on symptoms, lung function, and other indices of COPD morbidity and mortality is second only to smoking. The association is important because it represents risk factors that are at least partly modifiable. With increasing socioeconomic differences it is becoming increasingly important to treat socioeconomic status in COPD not just as a “nuisance” parameter to adjust for, but as an independent risk factor whose constituents need to be disentangled.

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