



TUBERCULOSIS MORTALITY IN ENGLAND AND WALES DURING 1982–1992: ITS ASSOCIATION WITH POVERTY, ETHNICITY AND AIDS

FRANCES ELENDER,^{1*} GRAHAM BENTHAM¹ and IAN LANGFORD²

¹School of Environmental Sciences, University of East Anglia, Norwich, Norfolk NR4 7TJ, U.K., and

²Centre for Social and Economic Research into the Global Environment (CSERGE), University of East Anglia, Norwich, Norfolk NR4 7TJ, U.K.

Abstract—This paper seeks to establish the strength of association between contemporary tuberculosis (TB) in England and Wales and several potential aetiological factors. It presents an ecological analysis of standardised annual TB mortality rates for the 403 local authority districts between 1982 and 1992, disaggregated by age and sex. Social, demographic and ethnicity measures from the 1981 and 1991 censuses and standardised annual AIDS-related mortality rates for young men are used to calculate Poisson regression models. A strong association was found between all TB mortality groups and overcrowding at the household level. For women, no other measures improved the explanatory power of the models. In multiple regressions, both poverty and AIDS-related mortality explained additional variation in the model for younger men. The link between ethnicity and tuberculosis notifications was not reflected in this analysis of mortality. For all groups no evidence of a positive relationship with ethnicity was found, once overcrowding had been accounted for. The significance of household as opposed to district level crowding suggests that prolonged contact is required for disease transmission. Regression analysis indicates that it is the overcrowding and poverty among ethnic populations that is significant for their tuberculosis mortality. The fact that the relationship between AIDS and TB is confined to the group most typical of AIDS patients provides evidence that AIDS has little influence on the level of tuberculosis mortality in the wider population. Explanations for the observed relationship include preferential certification, migration for treatment and shortcomings in health care provision. © 1998 Elsevier Science Ltd. All rights reserved

Key words—tuberculosis, overcrowding, poverty, ethnicity, AIDS, Poisson regression

INTRODUCTION

In common with many other countries, England and Wales faces a resurgence of tuberculosis (Kochi, 1991; Medical Research Council, 1988). After declining for more than a century, notification rates began to increase in the mid 1980s and the long-term downward trend in mortality also shows signs of leveling off at about 400–500 deaths annually (Nisar and Davies, 1991). Several factors may have contributed to these trends, including increases in poverty, immigration from high risk areas and the epidemic of HIV and AIDS.

Tuberculosis is both a respiratory and infectious disease that is strongly associated with material deprivation, including poor housing conditions (Smith, 1989). This association can be explained in terms of two broad causal mechanisms: increased exposure to infection and increased susceptibility to disease. Virtually all transmission of infection occurs in enclosed environments as exposure to ultraviolet radiation is known to kill the bacterium (Snider, 1994). Thus, tuberculosis is associated with

crowded living conditions and high incidence rates have been reported in prisons, shelters and nursing homes (Snider and Hutton, 1989). However, whilst crowding increases the risk of exposure to infection this does not invariably lead to disease. It is known that immunological status is instrumental in whether or not an individual develops tuberculosis. Impoverished housing conditions, inadequate diet, physical hardship and psychological stress are some frequent consequences of poverty and all are associated with suppressed immunity (Khansari *et al.*, 1990; McMurray and Bartow, 1992; Halpern, 1995). It is unsurprising, therefore, that a number of studies find high rates of active tuberculosis amongst the homeless, with levels between 150 and 300 times that of the general population (Imperato, 1992; Neims *et al.*, 1992; Ramsden *et al.*, 1988). There is also evidence that the increase in tuberculosis notifications is predominantly associated with the poorest areas of the country and that individuals living in such areas are at greater risk of developing the disease (Bhatti *et al.*, 1995; Mangtani *et al.*, 1995; Cundall and Pearson, 1988; Goldman *et al.*, 1994).

*Author for correspondence.

Whilst epidemiological evidence suggests that susceptibility to tuberculosis is under some genetic control (Motulsky, 1979; Skamene, 1986; Stead *et al.*, 1990), in countries where the disease is common the large pool of infective individuals presents a risk to all genotypes. However, in countries where tuberculosis is relatively rare, ethnic group can be a strong influence on its epidemiology. England and Wales has large numbers of immigrants from the Indian subcontinent (ISC) where incidence of the disease is much higher. Surveys have established that this group experiences tuberculosis rates up to 30 times higher than are seen in the white population (Medical Research Council, 1988). Research has shown that the relationship between ethnicity and tuberculosis is multifaceted and includes cultural, dietary and socio-economic components. Whilst the recent increase in tuberculosis is not matched by an equivalent increase in immigration from high incidence countries, it is known that individuals visiting their country of origin can "bring back" tuberculosis on their return (McCarthy, 1984). There is also evidence that not all immigrants from the ISC are at equal risk as Hindus experience a higher level of tubercular disease than Muslims (Chanarin and Stephenson, 1988; Finch *et al.*, 1991). It has been suggested that vitamin D deficiency amongst the predominantly vegetarian Hindus has an immunosuppressive effect, resulting in a greater vulnerability to infection. The question as to whether the poverty of certain ethnic groups is the predominant factor in relation to their high tuberculosis levels, has generated much debate. Individual level studies have found very similar tuberculosis rates for "white" and ISC groups in areas that are particularly deprived (Cundall and Pearson, 1988; Goldman *et al.*, 1994). This suggests that, in some situations, poverty is the predominant causal mechanism.

It is well established that the impairment of the immune system as a result of HIV infection predisposes an individual to tuberculosis and the disease is now regarded as a "sentinel" manifestation of the progression from HIV to AIDS (Festenstein and Grange, 1991; Watson and Gill, 1990). During the past 15 years, alongside the dramatic rise in HIV, Africa has experienced a concomitant increase in the tuberculosis rate (Kochi, 1991). In the United States, HIV infection has also been implicated in the rapid increase in tuberculosis notifications for young men. However, material deprivation, immigration and the dismantling of the tuberculosis control programme have also been demonstrated to be causative factors (Brudney and Dobkin, 1991). The extent to which HIV is implicated in the resurgence of tuberculosis in England and Wales will depend on the overlap between the populations infected with these two conditions. This overlap is small. In the United Kingdom the majority of individuals with tuberculosis are elderly or of ISC origin whilst HIV is most common amongst young, white men.

As yet, ecological studies have found little evidence of any interaction between HIV/AIDS and tuberculosis (Davies, 1989; Nisar *et al.*, 1992). At the individual level, the most recent national survey reports a prevalence of 2% for HIV amongst notified cases of tuberculosis (Watson, 1996). However, there is some evidence that in certain areas of London the prevalence of dual infection may be double this figure and that the trend is an increasing one (Foley and Miller, 1993; Tomlinson *et al.*, 1992).

Tuberculosis can arise in two different ways: either from a recent infection with *Mycobacterium tuberculosis*; or from the reactivation of dormant tubercle bacilli years or decades after initial infection which can then give rise to tubercular disease. Thus, the present level of tuberculosis in England and Wales comprises both individuals with "new" exogenous infections and those with a reactivation of "old" endogenous disease. The annual risk of developing pulmonary tuberculosis following a recent primary infection is estimated to be 300 times greater than the risk of disease from endogenous reactivation (Sutherland and Svandova, 1972). However, older people having lived through a period of high tuberculosis incidence, are very likely to have been infected with *M. tuberculosis* and now comprise a large and growing population group. In contrast, younger people who have acquired primary infections have done so during a period of much lower incidence and consequently comprise a much smaller subgroup. It is generally assumed, therefore, that disease in the elderly largely consists of endogenous reactivation whilst most tuberculosis in younger people is the result of exogenous infection. Unfortunately, the two types of disease are not clinically distinguishable and DNA analysis is the only definitive means of establishing whether infections are from the same strain of *M. tuberculosis* (Godfrey-Faussett, 1994). For both exogenous and endogenous disease, immunological status is a key factor. In the case of the latter, dwindling immunocompetence associated with old age is thought to be instrumental. Adverse contemporary circumstances may interact with this natural propensity for reduced immunity, thus increasing the risk of tuberculosis. Alternatively, it may be that "the die is cast" much earlier in life and that historical factors are of greater importance for development of tubercular disease.

Previous ecological studies have examined the relationship between measures of poverty and ethnicity on notification rates in England and Wales (Bhatti *et al.*, 1995; Mangtani *et al.*, 1995). However, notification data at the district level are not routinely available disaggregated by age which precludes any investigation of the relative effect of potential causative factors on endogenous and exogenous disease. Mortality data do not suffer from this limitation. Whilst acknowledging that tuberculosis deaths are a weak signal for morbidity (ap-

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