These observations have led to the frequently expressed notion that tuberculosis naturally occurs in waves, thereby implying the development of some form of herd immunity. There is, in fact, little evidence that such herd immunity develops and it is more likely that the decline is largely brought about by improvements in socio-economic factors, such as better working conditions and less overcrowding, which reduce the contagion parameter. If this is the case, there is no guarantee that the high incidence of tuberculosis seen in many of the poorer countries will decline significantly unless socio-economic conditions improve or unless more effective health measures are adopted — itself an important aspect of socio-economic development.

Likewise, hopes that the incidence of tuberculosis would continue to fall in developed nations until it eventually disappeared have not been realised. For the reasons outlined above, many developed countries have experienced an upsurge in the incidence of the disease since the 1980s. Effective chemotherapy was introduced in the developed countries at a time when the incidence of tuberculosis was rapidly declining and this appears to have led to over-optimistic expectations that 'scientific' interventions alone would lead to the elimination of tuberculosis. This, in turn, has led to an underestimation of the impact of changing socio-economic conditions on the natural history of the disease. In the centenary year of the discovery of the tubercle bacillus by Robert Koch, Waaler (1982) wrote that "without Koch's discovery, the socio-economic character of tuberculosis would have been clearer, and a demand for redistribution of the wealth of the community would have become a much more important issue".

The Impact of Poverty on Tuberculosis

Poverty, malnutrition and overcrowding have long been recognised as being among the principal predisposing factors for tuberculosis. As discussed above, improved socio-economic conditions contributed more to the dramatic decline in the prevalence of this disease in the

industrialised countries from the late 19th century to the present time than medical interventions.

There is a clear association between socio-economic factors and the incidence of tuberculosis in England and Wales where, between 1980 and 1992, the incidence showed a 35% increase among the poorest 10% of the population, a 13% increase among the next poorest 20%, but no increase among the remaining, relatively more affluent, 70% (Bhatti *et al.*, 1995). In the city of Liverpool, the incidence of tuberculosis was significantly related to the Townsend and Jarman deprivation indices (Spence *et al.*, 1993), while in various districts of London the notification rate increased by 12% for each 1% rise in the numbers of persons living under overcrowded conditions, irrespective of their ethnic origin (Mangtani *et al.*, 1995). In the major cities of the UK and other industrialised countries there is a particularly high incidence of tuberculosis among single homeless people, among whom there is also a high incidence of alcoholism. This group of patients is notoriously difficult to treat as they have little confidence or trust in health workers and often abscond (Moore-Gillon, 1998; Citron, 1997).

In the USA, the recent epidemics of tuberculosis have principally affected the socio-economically underprivileged. In New York City, the incidence of tuberculosis, AIDS and death from all causes is much higher among those who receive welfare support and abuse drugs, alcohol or both (Friedman *et al.*, 1996). During an eight year study commencing in 1984, of 858 such persons aged between 18 and 64 years, 47 (5.5%) developed tuberculosis and 84 (9.8%) developed AIDS. A fifth of this population were dead by the end of the study period and infectious disease, principally tuberculosis, AIDS and pneumonia was the cause of 57% of these deaths. The annual incidence of tuberculosis in this group was almost 15 times that in the general population of New York City, and 70 times higher than the rate for the entire USA. Also, at an incidence of 744 per 100,000 of the population, it was very much higher than that encountered in most parts of the developing world. The high prevalence of tuberculosis in this disadvantaged population

may be associated with a high prevalence of AIDS, which was ten times higher than in the general population.

The Impact of the HIV/AIDS Pandemic on Tuberculosis

Infection by HIV is now by far the most important of the factors predisposing to the development of overt tuberculosis in those infected by the tubercle bacillus. For this reason, Chretien (1990) referred to the combination of these pathogens as 'The Cursed Duet'.

The HIV pandemic is having three major effects on the behaviour of tuberculosis. It markedly increases the chance of an infected person developing overt disease, it leads to a considerable reduction in the time interval between infection and the manifestation of such disease and it modifies the clinical features of the disease, notably in the more profoundly immunosuppressed. A fourth, indirect and delayed effect is the increased transmission of the infection in the general population as a result of the greater numbers of source cases (Lienhardt and Rodrigues, 1997).

As described above, people infected by the tubercle bacillus have about a 10% chance of developing overt tuberculosis at some period later in their lives. If infected when young, this risk is spread over several decades. If, on the other hand, the infected person is also HIV positive, the chance of developing active tuberculosis is increased to 50% over what is often a considerably shortened life span. The *annual* risk of developing active tuberculosis in HIV-positive persons is between 8 and 10%, which is over 40 times greater than the risk in HIV-negative persons (Dolin *et al.*, 1994; Antonucci *et al.*, 1995). Put another way, a co-infected person has roughly the same chance of developing tuberculosis in a single year as a person infected only by *M. tuberculosis* has in their entire lifetime.

HIV-related tuberculosis may be the result of either primary infection of a previously uninfected person, endogenous reactivation of latent disease or an exogenous reinfection. The proportion of these three