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Editorials

Tuberculosis Control and Social Change

Tuberculosis and its control are manifestations of social and economic development. During the industrial revolution, crowding and other factors caused the number of tuberculosis cases to increase. Tuberculosis rates fell steadily in most developed countries in this century, except during periods of social stress such as wartime, with higher rates persisting among immigrants from high-prevalence countries.[1] The discovery of antituberculosis medications accelerated this decline and brought into focus the possibility of eliminating tuberculosis in the United States.[2]

During the past 8 years, cases of active tuberculosis have increased substantially in the United States and other industrialized countries.[3,4] This increase was caused by the following: the spread of tuberculosis in such congregate settings as hospitals,[5] shelters,[6] and correctional facilities[7]; a declining public health infrastructure[8]; immigration from areas of the world where tuberculosis remains endemic[9]; and the human immunodeficiency virus (HIV) epidemic.[10]

Tuberculosis is caused by the bacteria from the *Mycobacterium tuberculosis complex*. Without treatment, approximately 1 in 10 persons with tuberculosis infection will develop tuberculosis disease at some point in their lives; most of the remaining persons will never have symptoms nor develop active disease or spread the infection to others.[11] Persons who are infected with both HIV and *M tuberculosis* are at much higher risk, perhaps 7% to 10% per year, for developing active disease.[12] HIV-infected persons newly infected with *M tuberculosis* face an even higher risk for active disease.[13] Antibiotics, which must be taken for at least 6 months, cure active tuberculosis in 98% of patients with drug-susceptible disease and prevent tuberculosis infection from progressing to active disease in 60% to 90% of infected persons, even those who are HIV-infected.[14]

Based on these data, the principles of tuberculosis control are (1) to identify all persons with active disease and ensure their complete treatment and (2) to identify high-risk persons with tuberculosis infection and provide them with complete preventive treatment. Groups at highest risk for tuberculosis infection and disease are HIV-infected persons, close contacts of persons with active tuberculosis, persons from countries where tuberculosis remains common, and patients whose chest radiographs show scarring due to incompletely treated tuberculosis. There is little or no public health value in identifying persons who have tuberculosis infection or disease if complete treatment cannot be offered. Although socioeconomic improvements can decrease tuberculosis incidence, effective tuberculosis control programs can reduce tuberculosis incidence even faster.[15]

Four articles in this issue of the Journal illustrate the challenges and priorities of modern tuberculosis control. Buskin and colleagues reviewed risk factors for active tuberculosis among patients in King County, Washington.[16] The authors found that HIV infection is the strongest risk factor for tuberculosis disease and that increasing age, male sex, non-White race/ethnicity, birth in a foreign country, and several medical conditions (e.g., partial gastrectomy and low body weight) are associated with tuberculosis disease. The authors also found that smoking and alcohol consumption increase the risk for

Editor's Note. See related articles by Buskin et al. (p 1750), Leonhardt et al. (p 1834), and Ciesielski et al. (p 1836) and commentary by Comstock (p 1729) in this issue.

active tuberculosis, although they did not control for the potentially confounding effects of HIV infection and socioeconomic status. As the authors note, these data provide the framework for targeting control efforts.

Tuberculosis can affect all sectors of society, but it disproportionately affects socially and economically disadvantaged communities, which require expanded outreach and services. Many tuberculosis cases in the United States went undetected 50 years ago, and it is estimated that half of tuberculosis cases are undetected in developing countries. In contrast, today in the United States, even in underserved communities, most active tuberculosis cases are known; therefore, rather than directing our efforts at case finding, we should direct our efforts at ensuring that patients with active disease complete their treatment and at finding high-risk infected persons and ensuring that they complete preventive therapy.

Leonhardt and colleagues[17] describe a cluster of tuberculosis among persons associated with a high-risk group—crack cocaine users. Of 89 identified contacts of a single source case, 46 (52%) were tuberculin positive. The authors report that 13 (15%) of these contacts had active disease, including 5 of 6 children (83%) who were 5 years old or younger and 6 of 14 children (43%) who were 6 to 18 years old. This study confirms other investigations demonstrating that a single individual can infect dozens or hundreds of people.[18]

Although young children are known to be at increased risk for tuberculosis disease if infected, the very high rate of active disease among children suggests either a dose-response relationship between exposure and active disease or an overdiagnosis of tuberculosis disease in children, in whom standard diagnostic criteria are difficult to apply. A dose-response relationship has not been convincingly documented as being a risk factor for the development of active disease in exposed persons. It is intriguing that Leonhardt et al. documented a 57% tuberculin positivity rate among contacts considered to have a high exposure level to the source case, compared with a 9% positivity rate among contacts who had less exposure. It would be interesting to control for HIV status and compare the rates of disease progression between these two groups of infected contacts to explore the possibility of a dose-response relationship.

What is most impressive about the report by Leonhardt and colleagues is the authors' successful outreach into a difficult-to-reach population. With persistence, sensitivity, and a mobile van, public health workers gained the trust and participation of patients and their social network. This was essential in identifying and testing contacts and in helping 74% of infected contacts complete isoniazid preventive therapy. An even more rapid public health response may have prevented 12 secondary cases believed to have been part of the cluster.

Although the incidence of tuberculosis disease has been well characterized in the United States, there is limited information on the incidence of tuberculosis infection. Case rates do not necessarily reflect infection rates. Ciesielski and colleagues present provocative data on a medically underserved community—migrant agricultural workers.[19] In 1988, the authors tested 543 workers and found a tuberculin positivity rate of more than 40%.[20] To estimate the rate of new tuberculosis infection, in 1991 the authors, using a design somewhat analogous to the "capture-recapture" method,[21] attempted to retest workers who were tuberculin negative in 1988. Forty-six such individuals were retested. Of these, 14 (30%) were tuberculin positive or had active tuberculosis.

These data suggest that the annual risk of infection in this community could be as high as 10%. Small sample size, potential selection bias, inaccuracies of tuberculin testing, and the confounding effect of tuberculin boosting, which may occur in Bacillus Calmette-Guérin (BCG-vaccinated individuals,[22] make these data difficult to interpret. It is clear, however, that tuberculosis in migrant farmworkers warrants further investigation and intervention. The report by Ciesielski et al. also highlights the importance of improved coordination and communication among health care workers, public health programs, clinics, and other agencies in serving difficult-to-reach populations.

Finally, Dr George Comstock, a pioneer in tuberculosis control, reviews past and prospective strategies for controlling the disease.[23] Physicians once debated whether tuberculosis was caused by heredity, the environment, or infection. Comstock astutely notes that although tuberculosis in an individual is caused by infection with *M tuberculosis*, tuberculosis in society and its control depend on environmental factors and that even genetic characteristics may be important and amenable to therapeutic intervention.[24] Although many recommended measures are complex, Comstock notes that "simplicity is likely to be the key to success."

Comstock calls for renewed investigation of the epidemiology of tuberculosis. Coming from an investigator who, as much as any other, defined our understanding of tuberculosis epidemiology,[25] this call is particularly compelling. There are many unanswered questions in tuberculosis epidemiology: Where does most transmission occur? Why are some patients and organisms so effective at spreading infection and disease? How can risk of reactivation best be predicted? Are immunosuppressed patients at increased risk for infection?

In 1980, many people incorrectly assumed that tuberculosis in the United States had been controlled; the case rate of tuberculosis in Central Harlem in New York City was 50 per 100 000 persons. In 1991, there was widespread concern about tuberculosis in New York City—the case rate for all of New York City was 50 per 100 000, and Central Harlem's rate had increased to 221 per 100 000. If we had been as concerned about Central Harlem in 1980 as we were about all of New York City in 1991, much of the city's epidemic might have been avoided. Similarly, if we were as concerned today about tuberculosis in the developing world as we are about tuberculosis in the United States, we could prevent cases here and abroad for decades to come. The tuberculosis bacterium infects approximately 1.7 billion people, causing about 8 million cases and 2 million to 3 million deaths annually worldwide, more than any other infectious agent.[26] With nearly 1 in 3 US tuberculosis cases occurring in foreign-born persons, we cannot afford to continue our policy of public health isolationism.

In the 1970s and 1980s, tuberculosis declined, and the programs that had been established in the United States for its control were disbanded.[8] As a result, we have seen a dramatic increase in tuberculosis and drug resistance in recent years. In response, federal, state, and local efforts have begun to reestablish effective tuberculosis control programs. As the disease once again begins to decline in the United States and leaves the front pages, our challenge will be to persevere. We must expand effective outreach programs such as those described by Leonhardt et al., provide services to underserved populations such as those described by Ciesiel-

ski et al., target services to groups identified by epidemiological studies such as the study by Buskin et al., conduct the epidemiologic investigations called for by Comstock, and work to improve the social and economic environment that provides the substrate for the tuberculosis epidemic in the United States and abroad. □

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Toward an Epidemiology of Disablement

Finding out how many people with disabilities live in a population, what the preventable or intractable determinants of disability are, and what problems and unmet needs exist is not a straightforward task. The epidemiology of disablement, a global term covering impairment, disability, and handicap,[1] has developed slowly from multiple origins and within multiple disciplines. The articles devoted to disablement in this issue of the Journal illustrate the use of large data sets and epidemiological surveys to investigate its prevalence, distribution in the population, and consequences.[2–5] National surveys and large population catchment-area studies provide the data for analyses. In some instances,[4] other data sources provide contextual variables reflecting the social and economic conditions of the population.

Not surprisingly, these researchers have found a pattern for the distribution of disability or the restrictions in activity that is consistent with previous research on correlates of mortality and other indicators of health status.[6] Disability, measured as rates of activity limitation, increases with age and worsening socioeconomic conditions, and is unequally distributed by race and sex. Specific types of impairments, such as back pain, hand discomfort, and dermatitis, are more prevalent in some occupations than in others.[2] Specific combinations of disabilities and impairments, such as hip fracture in association with cognitive impairment, are associated with higher mortality.[5]

These findings prompt increased attention to health promotion and disease prevention focused on the needs of persons with disabilities.[7] Primary prevention activities are targeted to the occurrence of the disabling condition itself. Persons already affected by the condition, as illustrated in the articles in this issue,

Editor's Note. See related articles by Behrens et al. (p 1780), Bruce et al. (p 1796), Lafata et al. (p 1813), Marottoli et al. (p 1807), and Wagner et al. (p 1800) in this issue.