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# Tuberculosis: an Anthropological Perspective

Tuberculosis is a contagious and destructive disease that has plagued humans and their domesticated animals for at least 6000 years (Morse, Brothwell, and Ucko 1964), and continues to be a serious disease worldwide. Like most infectious diseases, the virulence of tuberculosis constantly fluctuates as changes occur in the host, the pathogen, and the environment. For example, in the United States, cases of tuberculosis are on an increase after a century of decline. A new population of homeless, intravenous drug users, and AIDS victims has provided an environment favorable for the comeback of tuberculosis in the United States. The spread of tuberculosis is tied very closely to human cultural practices and human biology. Living conditions, nutritional status, occupation, health, age, sex, group affiliation, and many other factors play a role in the susceptibility of individuals to tuberculosis. The possibilities for research on tuberculosis are endless. Because of the steady decrease in mortality until the last decade, researchers have almost ignored tuberculosis (Weiss 1992). This paper provides a basic understanding of the disease process involved in tuberculosis and addresses some of the major research currently being dealt with by researchers. It is not the purpose of this paper to exhaust any one of these topics but to provide an overview of some of the more important ones.

## Classification of Tubercle Bacilli

Tuberculosis is caused by a group of parasitic bacteria form in the genus *Mycobacterium*. This genus is composed of numerous species of acid-fast, nonmotile, nonsporulating rod bacteria ranging from saprophytes to obligated parasites. The common tie of all *Mycobacterium* species is that they resembles fungi (*myco* means "fungus") in their slow growth and method of toxicity much more closely than they do other bacteria (Stead 1973). Of the parasitic *Mycobacterium* species, humans are generally only infected by the tubercle bacterium and the leprosy bacterium (i.e., *M. leprae)*. Several other *Mycobacterium* species are occasional human pathogens or "atypical mycobacteria" and cause chronic progressive pulmonary diseases similar to tuberculosis (National Tuberculosis and Respiratory Disease Association, NTRDA, 1969).

Human tuberculosis is primarily caused by the tubercle bacillus *M. tuberculosis* (human type), occasionally by *M. bovis* (bovine type), and rarely by *M. avium* (avian type). The three types or forms, however, can only be distinguished by their cultural appearance and virulence in rabbits, guinea pigs, and other animals. *Mycobacterium tuberculosis* and *M. bovis* have similar cultural appearances and are both highly pathogenic for guinea pigs. *Mycobacterium bovis* is also highly pathogenic for rabbits while *M. tuberculosis* causes only mild infection in rabbits. *Mycobacterium avium* has a distinctly different cultural appearance than the human or bovine type and is not pathogenic for guinea pigs.

## Pathogenesis of Tuberculosis

### Route of Infection

In humans there are two primary means of entry for the tubercle bacillus: inhalation and ingestion. On rare occasions humans can be inoculated cutaneously by directly handling contaminated animal products or congenitally via a tuberculous uterus. The National Tuberculosis and Respiratory Disease Association (1969) also reports that tuberculosis has been acquired by the genital route and during mouth-to-mouth resuscitation in a few rare cases. Inhalation of airborne tubercle bacilli, contained in droplets ejected by persons with active pulmonary tuberculosis or in contaminated dust, is the most common route of infection by *M. tuberculosis*. The bovine tubercle bacillus, on the other hand, is most commonly acquired by ingestion of milk from tuberculous cattle. In most countries today pasteurization of milk has eliminated this danger, but cattle ranchers, dairy

farmers and other people working closely with cattle are still at risk of being inoculated by the bovine type tubercle bacillus though an aerial route. The avian bacillus is also typically acquired by ingestion. Raw or incompletely cooked eggs provide the best means of transfer.

The lungs are the most affected organ in human tuberculosis, but all organs are susceptible. Once entry is gained, tubercle bacilli obtained by aerial route primarily implant in the alveoli and bronchioles--generally in the best ventilated areas of the lung. They may also implant into the tonsils, cervical lymph glands, or the mediastinal lymph glands after gaining entry via macrophages in the lymphatic system (Willis and Willis 1972). Ingested tubercle bacilli typically implant in the intestine and mesenteric lymph glands (Willis and Willis 1972).

**Primary Tuberculosis**

The first time a person is infected by tubercle bacilli, there is generally no illness and primary lesions heal without notice. A healthy body is able to encapsulate the tubercle bacilli within a few weeks and render it inactive. The body is generally not successful at killing the bacilli, however, and encapsulated bacilli remaining alive frequently reactivate later when the infected persons immune system is stressed (e.g., malnutrition, trauma, other diseases). Only in individuals with an impaired immune system do primary infections lead to chronic tuberculosis without passing through a dormant stage.

In some cases the bacilli can escape the primary tubercle and enter the circulatory system before the immune system is able to get control over them. Once in the blood stream, bacilli are transported to other areas of the body and frequently set up secondary foci. These secondary foci may be within the lungs or any other organ or tissue where oxygen tension is relatively high (Lurie 1964). The secondary foci are usually brought under control immediately by the immune system. But, if resistance is low, hematogenous military tuberculosis (i.e., acute generalized) or tuberculosis meningitis (inflammation of the membranes of the spinal cord and brain) may develop.

**Chronic Tuberculosis**

For many years it was believed that chronic or secondary tuberculosis was the result of a person being reinfected after surviving the primary infection. Today, it is well known that most cases of chronic tuberculosis do not result from reinfection but from primary hematogenous foci lesions becoming reactivated. The chronic stage of tuberculosis is the most serious and contagious. The infected person often develops a chronic cough productive with yellow or greenish sputum and sometimes blood. Weight loss, fatigue, fever, and night sweats are common. Today, about five percent of patients with chronic tuberculosis will die from the disease.

**Extrapulmonary Tuberculosis**

Extrapulmonary tuberculosis is the result of direct contamination of the bronchi, larynx, mouth, and intestine, or hematogenous contamination of any organ. Tuberculosis of the larynx, bronchi and mouth generally results from implantation of tubercle bacilli in excretions from pulmonary lesions (NTRDA 1969). Superficial ulcers will frequently form and are maintained by constant reinfection. Intestinal tuberculosis occurs after a primary infection from drinking raw milk or other contaminated dairy products. Tubercular ulcers form most readily in the ileum and cecum around the ileocecal junction. Hemorrhage, peritonitis, fistula formation, and intestinal obstruction are common complications resulting from intestinal tuberculosis.

Hematogenous infection can spread to any organ, but the bacilli accumulate in higher concentrations and survive for longer periods of time in the lung, kidney, and splenic corpuscles (Lurie 1964). Bone marrow, synovial joints, and splenic pulp are also highly susceptible. Tuberculosis of the bones is rare but occurs most frequently in children where it commonly develops in the vertebrae (primarily T8-L2) and epiphyseal ends of the hip, knee, and ankle (Willis and Willis 1972).

## Age, Sex, and Group Affiliation Influences on Susceptibility

**Age and Sex**

Tuberculosis thrives when the infected person's immune system is stressed; occurring more frequently in infants, young adults, and the elderly. Infancy and old age are the times that humans are most susceptible to

almost all infectious diseases. Infants frequently do not have the acquired immunity to fight off a primary invasion of bacilli, and as a result tuberculosis is extremely dangerous to infants under one year of age (Rich 1944). Elderly people, on the other hand, frequently become victims of reactivated tubercle bacilli that they obtained earlier in life. As their immune system weakens due to age and other complications, it becomes unable to keep the bacilli inactive. Young adults normally have a fairly strong immune system, but it is frequently stressed by the responsibilities of providing for a growing family. Physical exertion, malnutrition, insufficient sleep, mental stress and other factors all reduce an individuals resistance to tuberculosis, and primary infections acquired during childhood can become reactivated.

During childhood and middle age, stress levels are at their lowest. Responsibilities, physical exhaustion, and mental stress are low while sleep and acquired immunity are at there greatest. For these reasons the rate of mortality from tuberculosis drops during childhood and again after the fourth decade. Although children between the ages of 5 to 14 years are especially resistant to tuberculosis (Anderson and Scotti 1976) the danger of infection is still high (Rich 1944).

There is little difference in the mortality rate of males and females from birth to puberty. During the reproductive years mortality in women exceeds males, but by age 40 the mortality rate of women drops below men and remains lower throughout life (Rich 1944; Anderson and Scotti 1976). The reason for an increased rate of female mortality during the reproductive years is probably directly related to the stress of menstruation, child birth, and child rearing (Rich 1944). By the fourth decade of life the mortality rate decreases for both males and females, but the mortality rate for females becomes lower than males. There are probably numerous reasons for the larger mortality rate decrease seen in women. In the United States, middle aged males have a higher occupational hazard effect and greater prevalence of alcoholism (Rich 1944). Furthermore, as Rich (1944:202) points out, the greater decrease in female mortality may result from the most susceptible females dying during the reproductive years. The stress on males during young adulthood is frequently not as great as it is in females, resulting in some very susceptible individuals surviving into their forties.

**Group Affiliation**

In the United States, between 1985 and 1987, there was a 6.3 percent increase in the number of cases among Blacks and a 12.7 percent increase among Hispanics (Snider, Salinas, and Kelly 1989). During the same time period, there was a 4.8 percent decrease in the cases of tuberculosis among non-Hispanic whites. Are these racial differences truly constitutional or merely a reflection of environmental influences? Differences in resistance to tuberculosis between racial groups has been demonstrated and discussed by numerous authors (Black 1975; CDC 1987a; CDC 1987b; CDC 1987c; Rich 1944; Rieder 1989; Snider, Salinas and Kelly 1989 and others), but relatively few studies have been conducted to eliminate environmental factors associated with the different races.

Rich (1944) argues that although there may be numerous environmental influences that can explain some of the racial differences seen in resistance to tuberculosis, environmental factors are inadequate to explain the anatomical and clinical differences encountered by researchers. He argues that there is a definite difference in the way different racial groups respond to the tubercle bacilli, and that these differences are indicative of difference in native resistance. As an argument, Rich (1944) explains that adult blacks frequently show symptoms of both "childhood" (primary) and "adult" (chronic) tuberculosis while white adults rarely exhibit "childhood" tuberculosis. Furthermore, pulmonary lesions tend to spread much more rapidly in blacks than in whites. Rich (1944) argues that the lesions spread much slower in whites because of a constitutive ability to inhibit the growth of tubercle bacilli.

Katz and Kunofsky (1960) and Stead et al. (1990) are two studies that have tried to eliminate the environmental influences. Katz and Kunofsky (1960) conducted a study of the difference in susceptibility between black and white patients in hospitals of the New York State Department of Mental Hygiene. Based on their data, Katz and Kunofsky (1960) concluded that under similar environmental conditions there was no significant difference in the rate of tuberculosis development between the racial groups. Stead et al. (1990) conducted similar studies examining the rate of infection among patients in an Arkansas nursing homes and prisoners in Arkansas and Minnesota prisons. Their research, however, concluded that black patients are almost

twice as likely to become infected by tubercle bacilli as white patients.

Although the studies of Katz and Kunofsky (1960) and Stead et al. (1990) appear to contradict one another, it must be kept in mind that Stead et al (1990) were looking at the rate of infection while Katz and Kunofsky (1960) were looking at the rate of disease development. However, Stead et al. (1990:426) suggested that "there are individual variations in the threshold that an inoculum of *M. tuberculosis* must exceed to establish an infection and that this threshold is significantly higher in whites than in blacks."

**Cultural Influence on Susceptibility**

Cultural practices play a major role in the transmission of tubercle bacilli and the morbidity of tuberculosis. Nutrition, occupation, living arrangements, hygiene, religion, economics, education, medical practices, and numerous other cultural influences have a direct effect on the incidence of tuberculosis.

Living arrangements especially affect the spread of tuberculosis. Tuberculosis is primarily transmitted person-to-person by aerial route, and crowded conditions resulting in numerous people breathing the same unfiltered air provides a perfect atmosphere for tubercle bacilli to be transmitted. The crowed conditions of urbanized areas are especially favorable for the rapid spread of tuberculosis (Hunter and Thomas 1984), but this does not mean that tuberculosis could not persist in small groups (Black 1975; Buikstra 1981). The low rate of mortality associated with tuberculosis and the ability of tubercle bacilli to lay dormant provides a means for the survival of tuberculosis in small populations. However, even within small populations, cultural housing practices can play a major role. Chen (1988) reports a greater incidence of tuberculosis among Sarawak natives living in longhouses than those living in individual dwellings.

Crowded conditions, poor hygiene, and poor nutrition associated with the economically deprived also provide conditions for the rapid morbidity of tuberculosis. Poor hygiene can help promote the spread of tuberculosis. Tubercle bacilli are relatively resistant to a large number of disinfectants and can survive for long periods of time in dried sputum as long as they are not exposed to direct sunlight. Young children, especially infants, become exposed to large doses of tubercle bacilli if a tuberculous individual is present and hygiene is poor.

Nutritional deficiency frequently associated with the poor also jeopardize the immune system and provides opportunities for tuberculosis to get "out-of-hand".

Occupational hazards can often weaken the immune system resulting in reactivation of old lesions and formation of new ones. Cattle farmers, along with butchers and veterinarians are at especially high risk of exposure to *M. bovis*. Mine workers exposed to silicon dust are much more susceptible to pulmonary tuberculosis than most people. Likewise, individuals working in jobs that are physically or mentally demanding frequently are more susceptible because their immune system is stressed.

Education, or the lack of it, promotes the spread of tuberculosis today. Noncompliance to treatment is common among less educated tuberculosis victims. Antibiotic treatment often brings relief to tuberculosis suffers within weeks, but it takes months for the antibiotic to kill the bacteria. Many tuberculous people today discontinue their medication once symptoms subside. As a result, the next time their immune response is weakened, inactive tubercle bacilli become active once again, and the surviving tubercle bacilli are frequently drug resistant (Weiss 1992).

Drug addiction is also associated with high incidence of tuberculosis. Drug addiction frequently results in some of the most favorable conditions for tuberculosis. Intravenous drug users frequently live in crowded and unsanitary areas, and their immune systems are likely to be jeopardized by the drugs themselves and the poor nutrition associated with drug use. Snider et al. (1989) associate intravenous drug use, along with noncompliance, HIV infection, and tuberculosis in correctional facilities, as a major problem in the treatment and spread of tuberculosis in the United States today, especially among poor minorities.

## Tuberculosis in Antiquity

Tuberculosis seemingly has plagued humankind for many millennia. Early literature from India, China, Babylonia, and Assyria describe tuberculosis-like symptoms between 2700 B.C. and 675 B.C. (Morse 1967). Several cases of tuberculosis in mummies and skeletal remains from the Old World provide further evidence of

its antiquity. Morse et al. (1964) report 31 cases of skeletal and mummy pathology resembling tuberculosis in Egypt between 3700 to 1000 B.C. Formicola et al. (1987) attributed skeletal lesions in a fourth millennium Italian Neolithic burial site to tuberculosis.

Pre-contact American Indian art forms of hump-backs may also indicate tuberculosis cases in America, but Morse (1967) argues that they can not be used as evidence of tuberculosis. He theorizes that persons in advanced states of tuberculosis would not be lying on their backs playing the flute as represented by the Kokopoli pictographs from Arizona. However, Morse (1967) accepts evidence from early Mesopotamian medical texts that sufferers of tuberculosis have respirations that 'sound like a flute.' It is possible that the playing of the flute by Kokopolo figures is symbolic of the wheezing associated with advanced states of pulmonary tuberculosis. In the New World, skeletal evidence of tuberculosis has also raised controversy. Morse (1967) argues that skeletal evidence does not provide sufficient evidence of tuberculosis in the New World prior to European contact.

However, numerous other authors (Allison et al., 1973; El-Najjar, 1979; Lichtor and Lichtor, 1957; Pfeiffer, 1984; Richie, 1952 and others) have contributed skeletal lesions in pre-contact Native Americans to tuberculosis. Recently, Arriaza et al. (1995) have isolated a segment of DNA they argue is unique to *M. tuberculosis* from a vertebral lesion in a pre-contact Northern Chile population.

## Possible Origins of Human Tuberculosis

The origin of the human-type tubercle bacillus has been speculated on by several authors but little research has been directed at solving the dilemma. The two species *M. tuberculosis* and *M. bovis* are so similar that many authors, including Van der Hoeden (1964) and the National Tuberculosis and Respiratory Disease Association (1969), include them as the same species. Some researchers even suggest that the difference between the human type and bovine type of tubercle bacillus could be the route of entry (Francis 1958). Francis (1958) suggests the possibility that tubercle bacilli are able to transform between the bovine and human forms and that atypical species represent intermediate forms. Rich (1944) points out that bovine tuberculosis can cause all forms of tuberculosis that the human type can and visa versa. However, he argues that bovine tubercle bacilli retain their individual characters even after years of implantation in human tissue and therefore should not be considered the same species as the human bacillus.

Hare (1967) theorizes that the close resemblance between bovine and human tubercle bacilli suggests that one is derived from the other. Hare also theorizes that pulmonary tuberculosis, which is chiefly a disease of human type bacilli, did not occur until the second millennium B.C. From this he speculates that the human tubercle bacillus is probably derived from the bovine type. Clark et al. (1987) agree with this sequence, stating that the fact that humans are susceptible to bovine tubercle bacilli but cattle are only mildly susceptible to human tubercle bacilli provides evidence for the mutation of the bovine type tubercle bacillus to the human type. Francis (1958) also points out that because the bovine tubercle bacillus produces disease in a wider variety of animals than does the human tubercle bacillus, the former is probably more primitive than the latter.

Cockburn (1963) suggests that the *Mycobacterium* that causes tuberculosis in both humans and cattle is derived from avian tubercle bacilli that were spread in droppings indiscriminately. Cockburn also theorizes that neither humans nor cattle acquired tuberculosis at any significant rate until humans began domesticating cattle. He bases this on the observation that wild animals do not generally develop tuberculosis unless confined for a period of time. But, no evidence is present as to whether the tuberculosis results from new infection or reactivation of dormant tuberculli. Buikstra (1981) proposes that if Cockburn (1963) is correct, then animals such as llamas, turkeys, dogs, and even bison may have served as reservoirs for the development of human tubercle bacilli in the Americas. Dogs are susceptible to both human and bovine tubercle bacilli and once exposed can serve as a source of infection to humans, especially children (Rich 1944). The likelihood of humans within the prehistoric Americas contracting tuberculosis with the domestication of turkey, however, is not as great. Although *M. avium* has been isolated from lesions of people diagnosed with tuberculosis, the cases are rare and the lesions are generally limited (Rich 1944). Likewise, adult fowl are highly resistant to both forms of human and bovine tubercle bacilli (Francis 1958).

## Summary

Tuberculosis is probably one of the most serious infectious diseases in the history of humankind. It may be impossible to know just how long humans and tubercle bacilli have been evolving together. Certainly, humans have served as hosts for several millennium and probably longer. As with almost any infectious disease, individual differences in susceptibility to tuberculosis are going to occur, and at least some racial differences in susceptibility can not be ruled out. But, most of the racial differences seen in morbidity and mortality statistics are probably due to environmental factors. I agree with Cockburn (1963) that both humans and their domesticated animals became hosts around the same time period. The major difference between the bovine bacillus and the human bacillus seems to be the mode of entry. However, the human lung appears to be more susceptible to *M. tuberculosis*. If Hare (1967) is correct in his hypothesis that pulmonary tuberculosis did not develop until the second millennium, then *M. tuberculosis* probably represents a mutant strain that became more specific for humans and *M. bovis* the primitive strain.

Evidence of tuberculosis in the Americas is overwhelming, especially with the recent discovery of tubercle bacillus DNA. Lower resistance of American Indians to tuberculosis in early historic times suggested by some authors as testimony of the absence of tuberculosis before European contact in the New World does not clearly demonstrate that they were not exposed to tubercle bacilli. The act of European contact and the crowding of American Indians onto reservations probably placed great strains on the immune system of many Native Americans. Furthermore, slightly different strains could have resulted in higher susceptibility. It is possible that tuberculosis was carried into the Americans by people crossing the Bering Strait. But, was this tuberculosis pulmonary? If Hare (1967) is correct, then pulmonary tuberculosis would not have developed until well after the land bridge closed. In this case tuberculosis would have probably developed in the Old World and not have been introduced into the Americas until after European contact unless it was carried across via water migration. This does not mean that tuberculosis of the lungs was not possible. Bovine bacilli have been isolated from forms of tuberculosis, including pulmonary tuberculosis. However, the incidence of tuberculosis probably would not be as great since bovine tuberculosis is not as specialized for the lungs as *M. tuberculosis*. This idea tends to correlate with the evidence from the New World. Hopefully future DNA studies will allow researchers to positively identify the strain of tubercle bacillus from prehistoric sites. Until that time, however, one can really only speculate.

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