

OCCUPATIONAL RESPIRATORY DISEASES

Editor

James A. Merchant, M.D., Dr. P.H.

Associate Editors

Brian A. Boehlecke, M.D.

Geoffrey Taylor, M.D.

Technical Editor

Molly Pickett-Harner, M.F.A.

**Division of Respiratory Disease Studies
Appalachian Laboratory for Occupational Safety and Health**

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Public Health Service

Centers for Disease Control

National Institute for Occupational Safety and Health

September 1986

Disclaimer

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

DHHS (NIOSH) Publication No. 86-102

*For sale by the Superintendent of Documents, U.S. Government
Printing Office, Washington, D.C. 20402*

TUBERCULOSIS AS AN OCCUPATIONAL DISEASE

Laurence S. Farer

Kenneth E. Powell

DEFINITION

Tuberculosis is a communicable disease of man and animals caused by the bacterium *Mycobacterium tuberculosis* and, less frequently, *M. bovis*. Lesions most often occur in the lungs but may be found in any part of the body.

CAUSATIVE AGENTS

Species of *Mycobacterium* are characterized by unusual "acid fast" staining properties, slow growth, relative resistance to chemical disinfectants, and the ability to survive for decades within cells in the infected animal. Several species are known to cause human illness, but the virulence and communicability of *M. tuberculosis* make it by far the most significant human pathogen. With the exception of a comment in Research Needs, the information in this section pertains to *M. tuberculosis*.

LIST OF OCCUPATIONS AND INDUSTRIES INVOLVED

Tuberculosis is a contagious disease and can spread among individuals of any occupation. The few published studies of tuberculosis as an occupational hazard suggest that physicians, nurses, medical laboratory workers, and miners are at increased risk of tuberculosis (1)(3)(4). Other occupations presumably at increased risk are migrant workers, overseas personnel in any occupation, zoo employees, prison guards, and social workers and others who work with the impoverished and the derelict.

EPIDEMIOLOGY

Infection is almost always acquired via inhalation of contaminated microscopic particles generated by coughing, sneezing, speaking, or singing. Therefore, persons most likely to become infected are those with a prolonged exposure in a confined area to an infectious person.

In 1981, 27,373 cases of tuberculosis were reported in the United States for an annual incidence of 11.9 per 100,000 persons. The incidence is higher in older age groups, in nonwhite persons, and in males. The incidence is also high among immigrants, alcoholics, and prisoners, but sufficient data are not available to calculate specific rates.

Unfortunately, few studies of tuberculosis incidence among various occupations have been reported. Therefore, only general and somewhat unsatisfactory comments can be made about tuberculosis as an occupational hazard.

Doctors, nurses, and medical laboratory workers are at greater risk than the population as a whole because they care for persons with tuberculosis. Barrett-Connor recently estimated the infection rate of physicians was about twice that of the general population (1). Harrington calculated the disease rate of medical laboratory workers in England was above five times that of the general population (see Table IX-3) (3). Individuals who work with elderly persons, nonwhite persons, immigrants, alcoholics, or prisoners presumably are at increased (but unquantitated) risk of infection.

Miners and others who work in poorly ventilated areas are more likely to be infected by a fellow worker who has tuberculosis than are persons who work in well ventilated areas. Studies among different groups of miners show that the tuberculosis mortality rate ranges from approximately 1.5 times expected for coal miners to approximately 10 times expected for cumingtonite-grunerite miners (see Table IX-3)(4)(5).

ESTIMATE OF POPULATION AT RISK AND PREVALENCE OF DISEASE

Table IX-3 shows that between 1,099 and 4,784 persons have tuberculosis disease because they work in a medical or mining occupation. Un-

Table IX-3
ESTIMATED NUMBER OF PERSONS WITH TUBERCULOSIS
ATTRIBUTABLE TO OCCUPATIONAL EXPOSURE, 1977

Occupation	Number of Persons (1)	Relative Risk of Tuberculosis	Estimated Incidence (4)	Estimated Attributable Incidence	Estimated Prevalence (5)	Estimated Attributable Prevalence
Medicine	3,853,000	2-5(2)	1,071-2,678	536-2,142	2,142-5,356	1,071-4,284
Mining	200,000	1½-10(3)	42-278	14-250	84-556	28-500
Social Workers	444,000					
Prison Guards						
Zoo employees						
Migrant Workers	130,000					
Overseas employees						

(1) U.S. Bureau of Census, 1978.

(2) Barrett-Conner, 1979; Harrington 1976.

(3) McDonald, 1978; Rockette, 1977.

(4) Based on United States incidence in 1977 of 13.9 per 100,000.

(5) Assumes average duration of illness is 2 years.

fortunately, sufficient data are not available to estimate the risk to persons in other occupations.

PATHOLOGY

Most tuberculous infections follow inhalation of the bacteria. Less frequently, infections occur after ingestion of direct inoculation through the skin. The bacilli multiply at the site of initial implantation and, if not contained by host defenses, are carried through the lymphatics to local and then more distant lymph nodes.

Usually the bacilli are contained by the host defenses. In some cases, however, either shortly after infection or after a prolonged dormancy, the organisms continue to multiply causing the systemic signs and symptoms of chronic infection with progressive destruction of the organ primarily involved (most often the lungs).

Workers exposed to silica are more likely to have tuberculosis because silica interferes with the function of the pulmonary macrophages (6). We do not know if other chemicals or minerals predispose to tuberculosis for similar reasons.

CLINICAL DESCRIPTION

Symptoms

Pulmonary tuberculosis is manifested by constitutional symptoms of loss of appetite, weight loss, fatigue, fever, night sweats, malaise, and organ-specific symptoms of cough (often productive of sputum and/or blood) and chest pain. Tuberculosis of other organs (such as kidneys or bones) causes the constitutional symp-

toms listed above plus symptoms specific to the organ involved.

Signs

Pulmonary tuberculosis, depending on its severity and duration, may be associated with nonspecific signs of chronic infection such as anemia. Pulmonary tuberculosis also may produce a variety of signs related to the respiratory tract such as rapid breathing and abnormal physical signs on percussion and auscultation of the chest. The chest x-ray is usually abnormal and often characteristic, but never diagnostic of tuberculosis. The lesions are usually patchy, in the apices of the lungs, and often cavitory.

The signs of tuberculosis of other organs include the (already mentioned) nonspecific signs plus signs specific to the organ involved. For example, tuberculous meningitis may cause cranial nerve damage, blindness, deafness, and disorders of consciousness from confusion to coma. Examination of the cerebral spinal fluid usually shows an increased cell count, increased protein concentration, and decreased glucose concentration. *Mycobacterium tuberculosis* may be demonstrated by appropriate stain or culture.

The Natural History of Disease

Most infections with *M. tuberculosis* are subclinical or unrecognized; the only evidence of infection is a positive tuberculin skin test. Progressive disease occurs in about 5% of persons within the first year after infection and in another 5% later in life. Therefore, once in-

fects, the risk of progressive disease exists for life. Unless treated with antituberculous chemotherapy, about 50% of persons who develop clinical illness die, frequently after months to years of progressive debilitation. Modern chemotherapy, however, if administered promptly and properly (see Treatment), will cure most patients.

Appropriate Laboratory Studies

The single most important laboratory study is the examination of secretions—usually sputum—or tissue for the infecting organism. Special media and procedures are required to culture *M. tuberculosis*. For persons with pulmonary tuberculosis, chest x-ray also is important.

Treatment

Antimicrobial drugs can cure tuberculosis. However the capability of the slowly growing *M. tuberculosis* to lie dormant within the host's cells necessitates prolonged drug treatment. Currently recommended therapy is 9-18 months of daily treatment with 2 or more drugs. Prolonged bed-rest and surgery, formerly the mainstays of therapy, now have a very limited role in the treatment of tuberculosis.

Prognosis

Promptly and properly administered chemotherapy confers an excellent prognosis. Although treatment is prolonged, most patients recover with minimal residua. Unfortunately, the long duration of treatment often results in erratic or incomplete ingestion of medicines. Inadequate chemotherapy may result in recurrent episodes of disease, progressive disability, and death.

DIAGNOSTIC CRITERIA

The diagnosis of tuberculosis is confirmed by the growth of *M. tuberculosis* from culture of sputum, CSF, urine, lymph nodes, or other infected tissue. If the organism cannot be grown, the diagnosis of tuberculosis should be made if the patient has a positive tuberculin skin test, the signs and symptoms are compatible with tuberculosis, a thorough evaluation uncovers no other cause for the illness, and the response to therapy is appropriate.

The most common diseases mimicking tuberculosis are systemic fungal infections, other mycobacterial infections, sarcoidosis, cancer, and the pneumoconioses.

METHODS OF PREVENTION

Transmission of tuberculosis can be prevented by the rapid identification and treatment of persons with disease and by the identification and treatment of those persons infected but not yet diseased (i.e., persons with only a positive skin test).

RESEARCH NEEDS

1. More information is needed about the incidence of tuberculosis in occupational groups, particularly those presumed to be at risk of infection.
2. Although a synergism between silicosis and tuberculosis is established, little information exists about possible synergism between tuberculosis and other mineral and chemical exposures.
3. The probable salubrious effect of more active participation of employers in the maintenance of chemotherapy among infected employees should be explored. Patients with tuberculosis can work and the workplace may be a good place to encourage regular drug usage to prevent relapse, progressive disease, and possible transmission. (Denial of employment to a noninfectious person who is on medication, because of fear of spread to fellow employees, is counter-productive and should not be tolerated.)
4. Cost-effective methods to identify contagious persons earlier in the course of illness need imaginative research.
5. Information is needed about the incidence of other Mycobacterial infections among various occupational groups.

REFERENCES

1. Barrett-Connor, E.: The epidemiology of tuberculosis in physicians. *JAMA* 241: 33-38, 1979.
2. Farer, L. S.: Mycobacterial infections in zoo animals: Health implications for humans. In: *Mycobacteria infections of zoo animals*, edited by Montali, R. J., Smithsonian Institution Press, Washington, D. C., pp. 223-226, 1978.
3. Harrington, J. M. and Shannon, H. S.: Incidence of tuberculosis, hepatitis, brucel-

- losis, and shingellosis in British medical laboratory workers. *Br Med J* 1:759-762, 1976.
4. McDonald, J. C., Gibbs, G. W., Liddell, F. D. K., and McDonald, A. D.: Mortality after long exposure to Cummingtinite-Grunerite. *Am Rev Respir Dis* 118:271-277, 1978.
 5. Rockette, H. E.: Cause specific mortality of coal miners. *JOM* 19:795-801, 1977.
 6. Snider, D. E.: The relationship between tuberculosis and silicosis. *Am Rev Respir Dis* 118:455-460, 1978.
 7. U.S. Bureau of Census, Statistical Abstract of the United States: 1978. (99th edition) Washington, DC, 1978.