

Epidemiology of tuberculosis

Author:

C Robert Horsburgh Jr, MD, MUS

Section Editor:

Neil W Schluger, MD

Deputy Editor:

Elinor L Baron, MD, DTMH

INTRODUCTION

An understanding of the epidemiology of *Mycobacterium tuberculosis* is critical for effective control. The global burden of tuberculosis (TB), risk factors for transmission, and the epidemiology of TB in the United States will be reviewed here. The epidemiology of drug-resistant TB is discussed separately. (See "[Epidemiology and molecular mechanisms of drug-resistant tuberculosis](#)".)

TERMINOLOGY

Tuberculosis terminology is inconsistent in the literature and in practice [1]. Relevant terms are defined in the table ([table 1](#)).

M. TUBERCULOSIS COMPLEX

M. tuberculosis is a member of the *M. tuberculosis* complex; other members include *Mycobacterium africanum* and *Mycobacterium bovis*.

M. africanum is most commonly found in West African countries; it causes up to a quarter of cases of TB in the Gambia [2]. The symptoms of infection resemble those of *M. tuberculosis*. The infectivity is similar to *M. tuberculosis*, and it is an important opportunistic pathogen in the setting of advanced immunosuppression due to human immunodeficiency virus (HIV) or other causes. Management is identical to management for disease due to *M. tuberculosis*.

M. bovis is discussed in detail separately. (See "[Mycobacterium bovis](#)".)

GLOBAL BURDEN

Epidemiology and socioeconomic factors

— The World Health Organization (WHO) estimated that 10.7 million new tuberculosis (TB) cases occurred in 2024 ([figure 1](#)) [3]. Of these, an estimated 390,000 had TB resistant to [rifampin](#) (multidrug-resistant TB).

The global incidence of TB peaked around 2003 and was declining until 2020 [4]. The COVID pandemic led to an increase in overall TB incidence ([figure 2](#)). The number of people diagnosed with TB in 2024 reached a new high of 8.3 million, but this means that 2.4 million persons with TB were still undiagnosed (the "missing millions"). Moreover, in 2024, TB caused an estimated 1.23 million deaths. More than 1.7 billion people (approximately 22 percent of the world population) are estimated to be infected with *M. tuberculosis* [4,5].

The epidemiology of TB varies substantially around the world ([figure 1](#) and [table 2](#)). The highest rates (300 per 100,000 or higher) are observed in sub-Saharan Africa, India, and the islands of Southeast Asia and Micronesia. Intermediate rates of TB (26 to 300 cases per 100,000) occur in China, Central and South America, Eastern Europe, and northern Africa. Low rates (less than 25 cases per 100,000 inhabitants) occur in the United States, Western Europe, Canada, Japan, and Australia.

Poverty, HIV, and drug resistance are major contributors to the resurging global TB epidemic [6,7]. Approximately 95 percent of TB cases occur in resource-limited countries. Approximately 1 in 12 new TB cases occur in individuals who are infected with HIV; 74 percent of these HIV-coinfected persons resided in Africa [4]. An estimated 390,000 cases of multidrug-resistant (MDR)-TB or rifampin-resistant TB also occurred in 2024.

Socioeconomic development and access to quality of health services appear to be at least as important as any specific TB control measure. The likelihood of success of TB control efforts is likely related to socioeconomic indicators, including gross domestic product per capita, mortality of children <5, access to clean water, and adequate sanitation and health expenditure per capita [8].

The importance of addressing these socioeconomic factors to achieve TB control is reinforced by the fact that many countries have experienced a rapid decline in TB burden without good access to high-quality TB treatment. In Europe, for example, TB morbidity and mortality declined long before effective chemotherapy was available, largely because of socioeconomic development, improved living conditions, better nutrition, and isolation of infectious cases in sanatoria [9].

Approach to global elimination

WHO "End TB" strategy

— The WHO's "End TB" strategy for 2016 to 2035 focuses on reducing deaths, reducing disease incidence, and reducing catastrophic costs due to TB ([table 3](#)). These TB goals were established as part of the Sustainable Development Goals (SDG) for 2016 to 2030 [[10](#)].

The new goals include a 90 percent reduction in TB deaths and an 80 percent reduction in TB incidence rate by 2030. Accomplishing these goals will require accelerating the decline in TB incidence from the current rate (1.5 to 2 percent) to 15 percent annually; such a decline could reduce the incidence of TB to <1 case per million by 2050. Achievement of this goal requires improvements in diagnosis, completion of TB disease treatment, and diagnosis and treatment of TB infection (TBI; formerly latent tuberculosis infection).

The 2016 to 2035 SDGs for TB also recognize that TB treatment forces many patients and their families into poverty; thus, an additional important goal is that "no affected families face catastrophic costs related to TB."

Reasonable progress was made towards these goals in 2018 and 2019, but the coronavirus disease pandemic in 2019 and 2020 disrupted progress and will likely have a long-term negative impact [[11](#)].

Active case finding

— The greatest barrier to reducing TB incidence and TB deaths is the substantial proportion of persons with TB disease that are never diagnosed and thus never treated. The WHO estimates that roughly 30 percent of such persons are never diagnosed [[12](#)]. Thus, efficient strategies for screening and treating these individuals, known as "active case finding," are needed.

Such programs need to screen for TB disease among patients not seeking health care for symptoms; approaches have included radiographic screening, sputum screening, and symptom surveys [[13,14](#)]. Interventions have targeted populations at increased risk, including known contacts of individuals with TB disease as well as individuals who are homeless, incarcerated, and/or HIV-infected.

The optimal approach to finding individuals with undiagnosed TB disease in high-incidence regions is uncertain; most cases occur in the absence of recognized risk factors [[15](#)]. In a cluster-randomized trial in Vietnam, active community-wide screening was compared with standard passive case detection (control) for reducing the prevalence of pulmonary TB among individuals ≥ 15 years, regardless of symptoms [[16](#)]. In 60 intervention clusters, active screening via sputum nucleic acid amplification testing (NAAT) was performed annually for three years, and antituberculous therapy was administered to patients diagnosed with TB; in 60 control clusters, no active screening was performed. In the fourth year, the prevalence of pulmonary TB was assessed via sputum NAAT (42,150 participants in the intervention group and 41,680 participants in the control group); active screening was associated with a lower prevalence of pulmonary TB than standard passive case detection alone (126 versus 225 per 100,000; prevalence ratio 0.56, 95% CI 0.40-0.78).

Further investigation is needed prior to broad implementation of active screening to assess the generalizability of these findings, the durability of the effect in endemic settings, and the feasibility of scaling up screening with NAATs.

TBI screening and treatment

— Controlling the TB epidemic also requires programs addressing the large burden of TB infection (TBI). (See "[Tuberculosis infection \(latent tuberculosis\) in adults: Approach to diagnosis \(screening\)](#)".)

Progress toward targets

— By 2015, the millennium development goal target of reversing TB incidence was achieved, with TB incidence falling globally for several years beginning around 2006, at a rate of 1.5 to 2 percent per year [[4](#)]. Moreover, the global TB mortality rate has fallen by 45 percent since 1990. However, if the slow rate of decline in TB incidence and mortality is not accelerated, TB will continue to be a substantial source of global morbidity and mortality for decades.

An estimated 30 percent of patients with TB disease remain undiagnosed. Nearly half of the "missing cases" occur in Indonesia, India, and China [[12](#)]. Data on treatment success rates follow similar geographic patterns, although these rates reflect only the proportion of patients who received treatment. It is sometimes uncertain whether these patients completed an effective treatment course. The western Pacific and eastern Mediterranean have the greatest success (91 to 92 percent); other regions have fared less well (75 to 82 percent). Success is limited in part by the high prevalence of HIV infection in Africa and drug resistance in Eastern Europe.

Most patients who are coinfectd with TB and HIV are not recognized to be coinfectd. For example, the worldwide percentage of patients with TB disease who underwent subsequent HIV testing improved only marginally (from 1 to 55 percent) during the time period 2002 to 2015.

Detection and treatment of MDR-TB are also inadequate. The WHO estimates that only forty percent of patients with MDR-TB were identified and started on treatment in 2022 [[12](#)].

IN THE UNITED STATES

— Following a marked decline in the incidence of tuberculosis (TB) in the United States over several decades, the incidence rose in the period 1985 to 1992 [[17,18](#)]. Since 1992, there had been a substantial decline in the rate of TB; in 2020, it fell to a historic low of 2.2 per 100,000 ([figure 3](#)). In 2023, it had risen to 2.9 per 100,000, an increase that may partially be attributable to the coronavirus disease (COVID-19) pandemic [[19,20](#)].

A majority of TB cases in the United States now occurs in foreign-born individuals emigrating from countries with high rates of endemic TB (77 percent of United States TB cases in 2025; the case rate in this group is 15.4 per 100,000) [20]. Of such cases, 16.5 percent occurred in individuals residing in the United States for <1 year, while 24 percent occurred in persons who had been in the US for >5 years [21]. Restriction fragment length polymorphism (RFLP) analysis has demonstrated that most of these cases are due to reactivation of TB infection rather than transmission within communities [22-27]. Therefore, screening for and treatment of TB infection is an important prevention tool among foreign-born individuals.

Among United States-born individuals, a study including national TB registry data for 2011 to 2021 found that, compared with individuals who identify as White, TB incidence rate ratios were 4.4 to 14.2 times higher for individuals who self-identified as American Indian/Alaska Native, Asian, Black, or Hispanic [28]. Genotyping data suggested an important role of recent transmission in the observed disparities, supporting the need for timely identification and prevention of ongoing chains of transmission.

Increased travel and immigration of individuals from countries with endemic TB are certain to bring new challenges to TB control. In 2000, it was estimated that 11 million individuals with TB infection (with capacity for future reactivation) reside in the United States [29]. The countries accounting for the majority of cases of TB in foreign-born persons in the United States are Mexico, the Philippines, Vietnam, India, China, and the Dominican Republic and Haiti. The states accounting for the greatest number of United States cases are California, New York, New Jersey, Texas, and Florida (figure 4) [30,31].

According to data from the United States National TB Surveillance System, greater than 84.7 percent of TB patients in the United States received HIV testing in 2022; of these, 4.7 percent were HIV infected [32]. HIV infection rates in TB patients are highest among people who inject drugs, people who are homeless, correctional facility inmates, and people with an alcohol use disorder (35, 22, 16, and 15 percent, respectively) [33].

TB in the United States has been shown to be seasonal, with a peak in the spring and a trough in the fall [34]. Latitude-dependent factors, including reduced winter sunlight and its potential effect on vitamin D levels, do not appear to contribute significantly to seasonality in the United States. Instead, seasonality appears to be greater among clustered cases and children, groups in whom disease likely reflects recent transmission of TB. Therefore, TB disease resulting from recent infection with early progression to disease appears to be more influenced by season than disease that results from activation of TB infection.

RISK FACTORS

Risk factors for tuberculosis (TB) may be divided as follows:

- Impaired immunity (host factors)
- Increased exposure to infectious persons (environmental factors)

Host factors

— Categories of host factors are outlined in the following sections. The relative risk for selected risk factors is outlined below. The relative importance of different risk factors varies with prevalence of exposure across regions.

Immunosuppression

HIV infection

— Among HIV-infected individuals, the risk of acquiring TB is 9 to 16 times that of HIV-uninfected individuals [35,36]. The magnitude of risk is likely variable depending on the degree of HIV-induced immunosuppression [30,37-39].

Of the 10.8 million estimated new TB cases in 2023, 6.1 percent occurred among people with HIV infection [40]. The 2020 estimated prevalence of HIV among patients with TB (by country) is shown in the figure (figure 5).

The global incidence of new TB cases among HIV-infected patients has declined since 2000, largely due to increasing access to antiretroviral therapy (ART) (figure 2).

A large proportion of the global burden of HIV-associated TB occurs in sub-Saharan Africa, with 74 percent of new cases of TB and 79 percent of deaths. The burden is particularly high in southern Africa, where some countries have annual TB incidence rates exceeding 1000 per 100,000 population.

Dual infection with HIV and TB leads to reciprocal interactions that have significant clinical impact, as discussed in the following sections.

Effect of HIV on TB — The risk of TB doubles within the first year of HIV seroconversion due to rapid depletion of TB-specific T helper cells [41-43]. Thereafter, the risk of TB progressively increases with declining immunity [36,44-46].

HIV-infected individuals appear to be more likely to acquire *M. tuberculosis* if exposed [47,48], and they are also at increased risk of developing TB disease from reactivated TB infection [46]. In addition, HIV infection is a risk factor for accelerated progression of TB following exposure; in one study, the duration of TB disease prior to diagnosis was estimated to be three times shorter in HIV-infected patients than in HIV-uninfected patients [49]. HIV-infected persons starting ART are at particularly high risk for being diagnosed with TB disease; this is likely "unmasking" of clinically unrecognized disease [50-52].

The risk of disease and death from TB is substantially increased among HIV-infected individuals due to reactivation from pre-existing TB infection and in the setting of new TB disease with rapid progression of disease [53-57]:

- In low-incidence settings with low transmission risk, the rate of progression to TB disease is 3 to 13 percent per year [36,46]; the higher risk occurs in patients with CD4 <200 [46]. In two nosocomial TB outbreaks described in the United States and Italy, the risk of TB disease following exposure was 37 to 50 percent in

individuals with HIV infection and no known prior exposure [58,59].

- In high-incidence settings with high transmission risk, the likelihood of progression to TB disease is generally higher. In tuberculin skin test-positive HIV-infected individuals, the incidence of TB disease ranges from 3 to 21 per 100 person-years [60]. In a cohort of individuals with advanced HIV infection in South Africa, the incidence of TB disease was 68 per 100 person-years, likely the result of new TB disease and prevalent TB infection [44].

The risk of TB transmission from HIV-infected patients with TB is lower than the risk of TB transmission from HIV-uninfected patients with TB; this is because of the shorter disease duration as well as the higher proportion of sputum smear-negative TB seen with HIV infection [61]. In addition, advanced HIV-related immunosuppression ($CD4 \leq 250$ cells/mm³) has also been associated with reduced likelihood of TB transmission [62].

Effect of TB on HIV – — TB appears to increase the risk of progression to acquired immunodeficiency syndrome (AIDS) or death [63,64]. The acceleration of HIV diseases by TB may occur via one or more of the following mechanisms:

- TB infection is associated with significant increases in HIV viremia [65]. HIV viremia usually declines after initiation of successful TB treatment [66]; however, persistently high levels of viremia have been observed in some cases despite initiation of effective antituberculous therapy [67,68].
- Generalized immune activation, due to TB infection, may increase the proportion of CD4 cells that are preferential targets for HIV [69].
- Increased expression of the HIV co-receptors CCR5 and CXCR4 occurs in HIV-infected patients with TB coinfection [67].

Impact of ART on TB incidence – — Despite unmasking of TB seen soon after initiation of antiretroviral therapy (ART), the long-term risk of TB decreases with initiation of ART [70-80]:

- In a randomized trial including more than 800 HIV-infected patients with baseline CD4 count between 200 and 350 cells/mm³ randomized to "early ART" (within two weeks of enrollment) or "deferred ART" (ART initiated based on time to onset of clinical AIDS or attainment of a CD4 cell count <200 cells/mm³), patients who received early ART had lower rate of incident TB than those who received deferred ART (18 versus 36 cases; hazard ratio [HR] 2, 95% CI 1.2-3.6) [80].
- In an observational study in South Africa including more than 1500 patients on ART, TB incidence in patients with CD4 count <100 and CD4 count >700 cells/mm³ was 26 and 3 per 100 person-years, respectively [77]. The TB incidence among patients with CD4 >700 cells/mm³ was 4.4-fold higher than the incidence in HIV-uninfected individuals from the same community; however, this is likely an overestimate as TB case ascertainment was high in this cohort.

Risk for relapse and recurrence – — Patients with HIV infection have an increased risk of recurrent TB after successful therapy, usually due to exogenous reinfection [81-83]. In one study including more than 580 culture-positive episodes of TB, recurrent TB was more common among patients with HIV infection than in patients without HIV infection; reinfection accounted for 12 of 23 in individuals with HIV infection and 1 of 16 recurrences among patients without HIV infection [84].

Patients with an initial episode of TB represent a susceptible population at high risk for a second infection with a new strain. In one study including 342 HIV-infected and 321 HIV-uninfected miners in South Africa, rates of recurrent TB (more than two years after the initial episode) were 24 and 4 percent, respectively [82]. Rates of recurrent TB were higher than rates of initial infection.

Low CD4 lymphocyte count is a key risk factor for relapse [85,86]. Among HIV-uninfected patients, cavitation and positive culture after two months of treatment are predictors of subsequent relapse among HIV-uninfected patients [87]; these may also be predictors among HIV-infected patients but have been less well studied.

Other forms of immunosuppression

- Glucocorticoids – Patients receiving a daily dose of ≥ 15 mg of prednisone (or its equivalent) for ≥ 1 month are at increased risk for TB [88]. A case-control study in the United Kingdom, including more than 16 million person-years of TB risk demonstrated that patients with TB were 4.9 times more likely to have been using glucocorticoids than those without TB [89]. A meta-analysis also suggests that inhaled corticosteroids increase the risk of TB [90].
- Tumor necrosis factor (TNF) inhibitors and other biologics – TNF-alpha inhibitors (used in the treatment of rheumatic diseases and inflammatory bowel disease) impair host resistance to TB. This issue is discussed in detail separately. (See "Risk of mycobacterial infection associated with biologic agents and JAK inhibitors".)
- Transplant – Renal, cardiac, liver, and allogeneic stem cell transplants are all associated with increased risk for TB [91-94]. The risk in allogeneic stem cell transplants is less than in solid organ transplant patients; there does not appear to be an increased risk of TB in autologous stem cell transplant patients.

Substance use

— Substance use is the most common behavioral risk factor among patients with TB in the United States. Tobacco use is the most common (31 percent), followed by drug use and alcohol use (9 and 7 percent, respectively) [95].

- Tobacco – Cigarette smoking confers a relative risk of about 1.5 to 2 for the development of TB [96-100]. Smoking has been found to be associated with both risk of relapse of TB and TB mortality.
- Drug use – The epidemiologic factors associated with injection and noninjection drug use (eg, homelessness, incarceration) contribute to the high prevalence of TB among drug users [46,101-105].
- Alcohol – The risk of TB disease is substantially elevated in individuals who consume more than 40 g alcohol per day [106]. This may be due to the effect of alcohol and alcohol-related conditions on the immune system.

Nutritional status

— Malnutrition is an important risk factor for TB [107]; however, the relationship between impaired immunity due to malnutrition and risk of acquiring TB has not been well characterized [108-112].

- Underweight – Persons who are underweight (body mass index of <18.5) have increased risk for TB by a factor of 2.6 (1.2 to 4.8) [108,109].
- Vitamin D – Vitamin D plays an important role in macrophage activation and restriction of mycobacterial growth, and diminished serum vitamin D levels appear to increase risk for TB infection [113-120]. Among African immigrants in Australia, for example, individuals with TB infection or TB disease were observed to have substantially lower serum vitamin D levels than those without TB [117].
- Iron status – Iron is an important growth factor for *M. tuberculosis* in macrophages and appears to play an important role in host susceptibility to TB infection [121].

Issues related to nutrition supplementation for prevention of incident TB are discussed separately. (See "Prevention of tuberculosis: BCG immunization and nutritional supplementation", section on 'Nutritional supplementation'.)

Systemic diseases

— The diseases discussed in this section have been noted to confer some degree of increased risk for TB reactivation. However, in some cases, it can be difficult to discern the relative risk of systemic diseases for development of TB disease, since many studies were performed in areas where the prevalence of TB is relatively low.

- Silicosis – The risk of TB is increased among individuals with occupational exposure to silica; these include miners and construction workers working with stone. The mechanism is not fully understood but may be related to impairment of pulmonary macrophage function by silica crystals. The relative risk depends on the severity of the silicosis and has been estimated at 1.4 to 2.9 [122-124].
- Malignancy – The risk of TB is increased in patients with hematologic malignancies and head and neck cancer [125]. In a 25-year review of cancer patients in the United States, the rate of TB among patients with hematologic neoplasms was >200 cases per 100,000 persons, or about 40 times the rate among the general population. Among patients with head and neck cancer, the rate was >100 cases per 100,000 persons. It is important to note that these studies were performed in areas where the prevalence of TB is relatively low.

Patients with solid tumors other than head and neck cancer do not have an increased risk for TB; among these individuals, the rate paralleled that of the general population.

- Diabetes – The risk of developing TB increases with increasing diabetes severity [126-132]. A case-control study of 5290 patients demonstrated that poorly controlled diabetes confers a 2.9-fold increase in the risk of developing pulmonary TB; the risk associated with well-controlled diabetes was minimal [38]. The mechanism by which diabetes confers an altered immune response to *M. tuberculosis* is not fully understood but may be related in part to altered cytokine expression [133].
- Renal disease – The risk of TB among patients with chronic renal disease risk is 6.9 to 52.5 times that of individuals without renal disease [134,135]. Uremia causes reduced cellular immunity. Other factors that may diminish immunity in the setting of renal failure include malnutrition, vitamin D deficiency, and hyperparathyroidism.
- Gastric surgery – Gastric resection for peptic ulcer disease has been described as a risk factor for TB (relative risk 1.7 to 2) [136,137]. Although this procedure is no longer performed routinely, gastric bypass is a similar procedure that may confer similar risk [138]. The mechanism is not understood but may be related to loss of gastric acidity; however, the risk of TB among persons with gastric achlorhydria has not been studied.
- Celiac disease – Celiac disease (autoimmune inflammation of the small intestine) is a risk factor for TB; the mechanism is not fully understood but may be related to malabsorption [139]. In a study of two national registries, the risk of TB was significantly higher among 14,335 individuals with a prior diagnosis of celiac disease than among the 69,888 matched controls (hazard ratio 3.74).
- Cirrhosis – Two studies have demonstrated that cirrhosis is a risk factor for TB; one study evaluated persons largely affected by alcohol-associated cirrhosis [140] and a second in persons with cirrhosis largely resulting from chronic infectious hepatitis [141].
- Chronic obstructive pulmonary disease (COPD) – COPD is also a predisposing factor for TB. In two studies, COPD was associated with increased risk for TB of 2.2 (1.2 to 4.1) and 2.5 (2.2 to 2.8) [142,143]. However, some of this increased risk may be attributable to smoking or use of corticosteroids among persons with COPD.

Age and sex

- Age – In resource-limited settings, TB rates are highest among young adults, reflecting primary transmission in this age group. In the United States and other developed countries, the rate of TB among older adults is higher than among younger adults and children, reflecting reactivation disease, possibly attributable to impaired immunity with aging [144].
- Sex – Among HIV-uninfected individuals, the rate of TB is higher among males than females, beginning in the young adult years and persisting throughout life. This observation is thought to reflect more frequent TB exposure in the community among men than women [145].

Social and environmental factors

Household contacts

— Close household contact with an individual with smear-positive pulmonary TB is the most important risk factor for TB [146,147]. In a study of TB contact investigation, including 1080 smear-positive patients and their 6225 close contacts, 36 percent of contacts had positive tuberculin skin tests [148]; this compares with an

expected skin test–positive rate of only 2.9 percent in the general population [29].

Birth in a TB-endemic area

— The fraction of TB cases in the United States ascribed to foreign-born individuals increased from 22 to 58 percent between 1986 and 2007 and has continued to climb since then; by 2023, it was 76 percent (figure 3) [19,31,149]. The risk of TB is highest in the first year after immigration but remains higher than the United States–born population for up to 20 years after arrival [21,150]. Seven countries account for the majority of cases of TB in foreign-born persons in the United States: Mexico, the Philippines, Vietnam, India, China, the Dominican Republic, and Haiti [21,149,151].

Overseas screening for TB among United States–bound immigrants and refugees is a high-yield intervention for identifying TB and could reduce the number of TB cases among foreign-born persons in the United States [152,153]. Between 1999 and 2005, the prevalence of smear-negative and TB infection cases among immigrants was 961 and 837 cases per 100,000, respectively; the prevalence of these entities among refugees was 1036 and 2838 per 100,000, respectively. TB disease and TB infection were diagnosed in the United States in 7 and 1.6 percent of those with overseas diagnoses, respectively. The risk for reactivation of TB infection is highest just after entry into the United States but remains elevated for many years after arrival [154,155].

Community settings

— In places where contact with infectious individuals may occur, risk for acquiring TB infection is increased [156]. Crowding and poor ventilation can increase the risk of transmission in such settings [157-164]. Those at risk for increased exposure include residents and employees of congregate settings such as hospitals, correctional facilities, nursing homes, and homeless shelters. Among 1289 inmates incarcerated in 16 Maryland prisons in 1997, the incidence of tuberculin skin conversion was 6.3 infections per 100 person-years [157]. (See "Tuberculosis transmission and control in health care settings".)

Socioeconomic status

— TB has traditionally been associated with low socioeconomic status, which also may be associated with crowding, poor nutrition, poor access to medical care, public assistance, unemployment, and low education [165].

Minority groups

— TB disease rates in the United States vary among demographic groups. In 2020, the disease incidence among non-United States-born White, Hispanic, Black, and Asian individuals was 2.8, 8, 15.3, and 21.7 per 100,000, respectively [166].

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Tuberculosis (The Basics)")
- Beyond the Basics topic (see "Patient education: Tuberculosis (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Global burden** (see 'Global burden' above)
 - More than 1.7 billion people are estimated to be infected with tuberculosis (TB). The global incidence of TB disease peaked around 2003 and appears to be declining slowly. The World Health Organization (WHO) estimated that the incidence of TB disease in 2023 was 10.8 million new cases, an incidence rate of 134 per 100,000 persons, but 2.6 million people with TB remained undiagnosed.
 - Poverty, HIV, and drug resistance fan the flames of the TB epidemic. Worldwide, about 95 percent of TB cases occur in resource-limited countries. Among all new TB cases in 2023, 6.1 percent were estimated to be HIV infected; 74 percent of these cases occur in Africa. In 2023, there were an estimated 400,000 cases of multidrug-resistant (MDR)-TB.
- **Risk factors**
 - Socioeconomic development and access and quality of health services appear to be at least as important as any specific TB control measure. Risk factors for TB may be divided into issues related to host immunity (eg, immunologic defects that lead to increased susceptibility to infection) and issues related to environmental exposure to infection (eg, risk of exposure to a case of infectious TB). (See 'Epidemiology and socioeconomic factors' above and 'Risk factors' above.)
 - HIV is a major driver of the global TB epidemic; patients with HIV infection are at increased risk of acquiring TB infection, at increased risk of progressing from TB infection to TB disease, and at increased risk of death, compared with patients without HIV infection. These risks can be greatly reduced by administration of antiretroviral therapy. (See 'HIV infection' above.)

- **Approach to elimination** – In 2015, the WHO announced the "End TB" strategy, with new milestones and targets for 2025 and 2035 that could lead to substantial reductions in the global TB burden (table 3). (See 'WHO "End TB" strategy' above.)
- **In the United States** – A majority of TB cases in the United States occurs in foreign-born individuals emigrating from countries with high rates of endemic TB. (See 'In the United States' above.)

REFERENCES

- Behr MA, Kaufmann E, Duffin J, et al. Latent Tuberculosis: Two Centuries of Confusion. *Am J Respir Crit Care Med* 2021; 204:142.
- de Jong BC, Antonio M, Gagneux S. *Mycobacterium africanum*--review of an important cause of human tuberculosis in West Africa. *PLoS Negl Trop Dis* 2010; 4:e744.
- World Health Organization. Global tuberculosis report 2025. <https://iris.who.int/server/api/core/bitstreams/e97dd6f4-b567-4396-8680-717bac6869a9/content> (Accessed on April 03, 2026).
- World Health Organization. Global tuberculosis report 2020. <https://www.who.int/publications/i/item/9789240013131> (Accessed on April 05, 2021).
- Houben RM, Dodd PJ. The Global Burden of Latent Tuberculosis Infection: A Re-estimation Using Mathematical Modelling. *PLoS Med* 2016; 13:e1002152.
- Corbett EL, Marston B, Churchyard GJ, De Cock KM. Tuberculosis in sub-Saharan Africa: opportunities, challenges, and change in the era of antiretroviral treatment. *Lancet* 2006; 367:926.
- Wright A, Zignol M, Van Deun A, et al. Epidemiology of antituberculosis drug resistance 2002-07: an updated analysis of the Global Project on Anti-Tuberculosis Drug Resistance Surveillance. *Lancet* 2009; 373:1861.
- Dye C, Lönnroth K, Jaramillo E, et al. Trends in tuberculosis incidence and their determinants in 134 countries. *Bull World Health Organ* 2009; 87:683.
- Lienhardt C. From exposure to disease: the role of environmental factors in susceptibility to and development of tuberculosis. *Epidemiol Rev* 2001; 23:288.
- Lönnroth K, Raviglione M. The WHO's new End TB Strategy in the post-2015 era of the Sustainable Development Goals. *Trans R Soc Trop Med Hyg* 2016; 110:148.
- Fukunaga R, Glaziou P, Harris JB, et al. Epidemiology of Tuberculosis and Progress Toward Meeting Global Targets - Worldwide, 2019. *MMWR Morb Mortal Wkly Rep* 2021; 70:427.
- Global tuberculosis report 2023. World Health Organization. <https://iris.who.int/bitstream/handle/10665/373828/9789240083851-eng.pdf?sequence=1> (Accessed on April 08, 2024).
- Golub JE, Mohan CI, Comstock GW, Chaisson RE. Active case finding of tuberculosis: historical perspective and future prospects. *Int J Tuberc Lung Dis* 2005; 9:1183.
- Corbett EL, Bandason T, Duong T, et al. Comparison of two active case-finding strategies for community-based diagnosis of symptomatic smear-positive tuberculosis and control of infectious tuberculosis in Harare, Zimbabwe (DETECTB): a cluster-randomised trial. *Lancet* 2010; 376:1244.
- Verver S, Warren RM, Munch Z, et al. Proportion of tuberculosis transmission that takes place in households in a high-incidence area. *Lancet* 2004; 363:212.
- Marks GB, Nguyen NV, Nguyen PTB, et al. Community-wide Screening for Tuberculosis in a High-Prevalence Setting. *N Engl J Med* 2019; 381:1347.
- Burzynski J, Schluger NW. The epidemiology of tuberculosis in the United States. *Semin Respir Crit Care Med* 2008; 29:492.
- Khan K, Wang J, Hu W, et al. Tuberculosis infection in the United States: national trends over three decades. *Am J Respir Crit Care Med* 2008; 177:455.
- Williams PM, Pratt RH, Walker WL, et al. Tuberculosis - United States, 2023. *MMWR Morb Mortal Wkly Rep* 2024; 73:265.
- Provisional 2025 Tuberculosis Data, United States. Centers for Disease Control and Prevention. <https://www.cdc.gov/tb-data/aboutprovisionaldata/2025-provisional-data.html> (Accessed on April 03, 2026).
- Cain KP, Haley CA, Armstrong LR, et al. Tuberculosis among foreign-born persons in the United States: achieving tuberculosis elimination. *Am J Respir Crit Care Med* 2007; 175:75.
- Geng E, Kreiswirth B, Driver C, et al. Changes in the transmission of tuberculosis in New York City from 1990 to 1999. *N Engl J Med* 2002; 346:1453.
- Chin DP, DeRiemer K, Small PM, et al. Differences in contributing factors to tuberculosis incidence in U.S. -born and foreign-born persons. *Am J Respir Crit Care Med* 1998; 158:1797.
- Tornieporth NG, Ptachewich Y, Poltoratskaia N, et al. Tuberculosis among foreign-born persons in New York City, 1992-1994: implications for tuberculosis control. *Int J Tuberc Lung Dis* 1997; 1:528.
- Borgdorff MW, Behr MA, Nagelkerke NJ, et al. Transmission of tuberculosis in San Francisco and its association with immigration and ethnicity. *Int J Tuberc Lung Dis* 2000; 4:287.
- Jasmer RM, Ponce de Leon A, Hopewell PC, et al. Tuberculosis in Mexican-born persons in San Francisco: reactivation, acquired infection and transmission. *Int J Tuberc Lung Dis* 1997; 1:536.
- Walter ND, Jasmer RM, Grinsdale J, et al. Reaching the limits of tuberculosis prevention among foreign-born individuals: a tuberculosis-control program perspective. *Clin Infect Dis* 2008; 46:103.
- Li Y, Regan M, Swartwood NA, et al. Disparities in Tuberculosis Incidence by Race and Ethnicity Among the U.S.-Born Population in the United States, 2011 to 2021 : An Analysis of National Disease Registry Data. *Ann Intern Med* 2024; 177:418.
- Bennett DE, Courval JM, Onorato I, et al. Prevalence of tuberculosis infection in the United States population: the national health and nutrition examination survey, 1999-2000. *Am J Respir Crit Care Med* 2008; 177:348.
- Horsburgh CR, Moore M, Castro KG. Epidemiology of tuberculosis in the United States. In: *Tuberculosis*, 2nd ed, Rom WN, Garay SM (Eds), Lippincott Williams and Wilkins, Philadelphia 2004. p.31.
- Centers for Disease Control and Prevention (CDC). Trends in tuberculosis--United States, 2008. *MMWR Morb Mortal Wkly Rep* 2009; 58:249.
- Schildknecht KR, Pratt RH, Feng PI, et al. Tuberculosis - United States, 2022. *MMWR Morb Mortal Wkly Rep* 2023; 72:297.
- Centers for Disease Control and Prevention (CDC). Reported HIV status of tuberculosis patients--United States, 1993-2005. *MMWR Morb Mortal Wkly Rep* 2007; 56:1103.
- Willis MD, Winston CA, Heilig CM, et al. Seasonality of tuberculosis in the United States, 1993-2008. *Clin Infect Dis* 2012; 54:1553.
- Guelar A, Gatell JM, Verdejo J, et al. A prospective study of the risk of tuberculosis among HIV-infected patients. *AIDS* 1993; 7:1345.

36. Antonucci G, Girardi E, Raviglione MC, Ippolito G. Risk factors for tuberculosis in HIV-infected persons. A prospective cohort study. The Gruppo Italiano di Studio Tubercolosi e AIDS (GISTA). *JAMA* 1995; 274:143.
37. Corbett EL, Watt CJ, Walker N, et al. The growing burden of tuberculosis: global trends and interactions with the HIV epidemic. *Arch Intern Med* 2003; 163:1009.
38. Pablos-Méndez A, Blustein J, Knirsch CA. The role of diabetes mellitus in the higher prevalence of tuberculosis among Hispanics. *Am J Public Health* 1997; 87:574.
39. Havlir DV, Getahun H, Sanne I, Nunn P. Opportunities and challenges for HIV care in overlapping HIV and TB epidemics. *JAMA* 2008; 300:423.
40. World Health Organization. 2.4 Drug-resistant TB: treatment enrolment, coverage and outcomes. 2024. <https://www.who.int/teams/global-tuberculosis-programme/tb-reports/global-tuberculosis-report-2024/tb-diagnosis-and-treatment/2-4-drug-resistant-tb-treatment>.
41. Sonnenberg P, Glynn JR, Fielding K, et al. How soon after infection with HIV does the risk of tuberculosis start to increase? A retrospective cohort study in South African gold miners. *J Infect Dis* 2005; 191:150.
42. Geldmacher C, Schuetz A, Ngwenyama N, et al. Early depletion of Mycobacterium tuberculosis-specific T helper 1 cell responses after HIV-1 infection. *J Infect Dis* 2008; 198:1590.
43. Brenchley JM, Price DA, Schacker TW, et al. Microbial translocation is a cause of systemic immune activation in chronic HIV infection. *Nat Med* 2006; 12:1365.
44. Wood R, Maartens G, Lombard CJ. Risk factors for developing tuberculosis in HIV-1-infected adults from communities with a low or very high incidence of tuberculosis. *J Acquir Immune Defic Syndr* 2000; 23:75.
45. Holmes CB, Wood R, Badri M, et al. CD4 decline and incidence of opportunistic infections in Cape Town, South Africa: implications for prophylaxis and treatment. *J Acquir Immune Defic Syndr* 2006; 42:464.
46. Selwyn PA, Hartel D, Lewis VA, et al. A prospective study of the risk of tuberculosis among intravenous drug users with human immunodeficiency virus infection. *N Engl J Med* 1989; 320:545.
47. Jones-López EC, Acuña-Villaorduña C, Ssebidi M, et al. Cough Aerosols of Mycobacterium tuberculosis in the Prediction of Incident Tuberculosis Disease in Household Contacts. *Clin Infect Dis* 2016; 63:10.
48. Whalen CC, Chiunda A, Zalwango S, et al. Immune correlates of acute Mycobacterium tuberculosis infection in household contacts in Kampala, Uganda. *Am J Trop Med Hyg* 2006; 75:55.
49. Corbett EL, Charalambous S, Moloi VM, et al. Human immunodeficiency virus and the prevalence of undiagnosed tuberculosis in African gold miners. *Am J Respir Crit Care Med* 2004; 170:673.
50. Lawn SD, Wilkinson RJ, Lipman MC, Wood R. Immune reconstitution and "unmasking" of tuberculosis during antiretroviral therapy. *Am J Respir Crit Care Med* 2008; 177:680.
51. Wood R, Middelkoop K, Myer L, et al. Undiagnosed tuberculosis in a community with high HIV prevalence: implications for tuberculosis control. *Am J Respir Crit Care Med* 2007; 175:87.
52. Lawn SD, Myer L, Bekker LG, Wood R. Burden of tuberculosis in an antiretroviral treatment programme in sub-Saharan Africa: impact on treatment outcomes and implications for tuberculosis control. *AIDS* 2006; 20:1605.
53. World Health Organization. Global Tuberculosis Control. Surveillance, Planning, Financing: 2006. World Health Organization, Geneva 2010 (WHO/HTM/TB/2010.362).
54. Harries AD, Zachariah R, Corbett EL, et al. The HIV-associated tuberculosis epidemic--when will we act? *Lancet* 2010; 375:1906.
55. Horsburgh CR Jr. Priorities for the treatment of latent tuberculosis infection in the United States. *N Engl J Med* 2004; 350:2060.
56. van der Sande MA, Schim van der Loeff MF, Bennett RC, et al. Incidence of tuberculosis and survival after its diagnosis in patients infected with HIV-1 and HIV-2. *AIDS* 2004; 18:1933.
57. Whalen CC, Zalwango S, Chiunda A, et al. Secondary attack rate of tuberculosis in urban households in Kampala, Uganda. *PLoS One* 2011; 6:e16137.
58. Daley CL, Small PM, Schechter GF, et al. An outbreak of tuberculosis with accelerated progression among persons infected with the human immunodeficiency virus. An analysis using restriction-fragment-length polymorphisms. *N Engl J Med* 1992; 326:231.
59. Di Perri G, Cruciani M, Danzi MC, et al. Nosocomial epidemic of active tuberculosis among HIV-infected patients. *Lancet* 1989; 2:1502.
60. Akolo C, Adetifa I, Shepperd S, Volmink J. Treatment of latent tuberculosis infection in HIV infected persons. *Cochrane Database Syst Rev* 2010; :CD000171.
61. Behr MA, Warren SA, Salamon H, et al. Transmission of Mycobacterium tuberculosis from patients smear-negative for acid-fast bacilli. *Lancet* 1999; 353:444.
62. Huang CC, Tchetgen ET, Becerra MC, et al. The effect of HIV-related immunosuppression on the risk of tuberculosis transmission to household contacts. *Clin Infect Dis* 2014; 58:765.
63. López-Gatell H, Cole SR, Hessol NA, et al. Effect of tuberculosis on the survival of women infected with human immunodeficiency virus. *Am J Epidemiol* 2007; 165:1134.
64. Badri M, Ehrlich R, Wood R, et al. Association between tuberculosis and HIV disease progression in a high tuberculosis prevalence area. *Int J Tuberc Lung Dis* 2001; 5:225.
65. Toossi Z, Mayanja-Kizza H, Hirsch CS, et al. Impact of tuberculosis (TB) on HIV-1 activity in dually infected patients. *Clin Exp Immunol* 2001; 123:233.
66. Goletti D, Weissman D, Jackson RW, et al. Effect of Mycobacterium tuberculosis on HIV replication. Role of immune activation. *J Immunol* 1996; 157:1271.
67. Wolday D, Tegbaru B, Kassu A, et al. Expression of chemokine receptors CCR5 and CXCR4 on CD4+ T cells and plasma chemokine levels during treatment of active tuberculosis in HIV-1-coinfected patients. *J Acquir Immune Defic Syndr* 2005; 39:265.
68. Wolday D, Hailu B, Girma M, et al. Low CD4+ T-cell count and high HIV viral load precede the development of tuberculosis disease in a cohort of HIV-positive ethiopians. *Int J Tuberc Lung Dis* 2003; 7:110.
69. Vanham G, Edmonds K, Qing L, et al. Generalized immune activation in pulmonary tuberculosis: co-activation with HIV infection. *Clin Exp Immunol* 1996; 103:30.
70. Badri M, Wilson D, Wood R. Effect of highly active antiretroviral therapy on incidence of tuberculosis in South Africa: a cohort study. *Lancet* 2002; 359:2059.
71. Golub JE, Saraceni V, Cavalcante SC, et al. The impact of antiretroviral therapy and isoniazid preventive therapy on tuberculosis incidence in HIV-infected patients in Rio de Janeiro, Brazil. *AIDS* 2007; 21:1441.
72. Lawn SD, Bekker LG, Wood R. How effectively does HAART restore immune responses to Mycobacterium tuberculosis? Implications for tuberculosis control. *AIDS* 2005; 19:1113.

73. Girardi E, Antonucci G, Vanacore P, et al. Impact of combination antiretroviral therapy on the risk of tuberculosis among persons with HIV infection. *AIDS* 2000; 14:1985.
74. Girardi E, Sabin CA, d'Arminio Monforte A, et al. Incidence of Tuberculosis among HIV-infected patients receiving highly active antiretroviral therapy in Europe and North America. *Clin Infect Dis* 2005; 41:1772.
75. Lawn SD, Badri M, Wood R. Tuberculosis among HIV-infected patients receiving HAART: long term incidence and risk factors in a South African cohort. *AIDS* 2005; 19:2109.
76. Keiper MD, Beumont M, Elshami A, et al. CD4 T lymphocyte count and the radiographic presentation of pulmonary tuberculosis. A study of the relationship between these factors in patients with human immunodeficiency virus infection. *Chest* 1995; 107:74.
77. Gupta A, Wood R, Kaplan R, et al. Tuberculosis incidence rates during 8 years of follow-up of an antiretroviral treatment cohort in South Africa: comparison with rates in the community. *PLoS One* 2012; 7:e34156.
78. del Amo J, Moreno S, Bucher HC, et al. Impact of antiretroviral therapy on tuberculosis incidence among HIV-positive patients in high-income countries. *Clin Infect Dis* 2012; 54:1364.
79. Lawn SD, Wood R, De Cock KM, et al. Antiretrovirals and isoniazid preventive therapy in the prevention of HIV-associated tuberculosis in settings with limited health-care resources. *Lancet Infect Dis* 2010; 10:489.
80. Severe P, Juste MA, Ambroise A, et al. Early versus standard antiretroviral therapy for HIV-infected adults in Haiti. *N Engl J Med* 2010; 363:257.
81. Lahey T, Mackenzie T, Arbeit RD, et al. Recurrent tuberculosis risk among HIV-infected adults in Tanzania with prior active tuberculosis. *Clin Infect Dis* 2013; 56:151.
82. Glynn JR, Murray J, Bester A, et al. High rates of recurrence in HIV-infected and HIV-uninfected patients with tuberculosis. *J Infect Dis* 2010; 201:704.
83. Narayanan S, Swaminathan S, Supply P, et al. Impact of HIV infection on the recurrence of tuberculosis in South India. *J Infect Dis* 2010; 201:691.
84. Crampin AC, Mwaungulu JN, Mwaungulu FD, et al. Recurrent TB: relapse or reinfection? The effect of HIV in a general population cohort in Malawi. *AIDS* 2010; 24:417.
85. Nettles RE, Mazo D, Alwood K, et al. Risk factors for relapse and acquired rifamycin resistance after directly observed tuberculosis treatment: a comparison by HIV serostatus and rifamycin use. *Clin Infect Dis* 2004; 38:731.
86. Nahid P, Gonzalez LC, Rudoy I, et al. Treatment outcomes of patients with HIV and tuberculosis. *Am J Respir Crit Care Med* 2007; 175:1199.
87. Nahid P, Dorman SE, Alipanah N, et al. Official American Thoracic Society/Centers for Disease Control and Prevention/Infectious Diseases Society of America Clinical Practice Guidelines: Treatment of Drug-Susceptible Tuberculosis. *Clin Infect Dis* 2016; 63:e147.
88. Targeted tuberculin testing and treatment of latent tuberculosis infection. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, July 1999. This is a Joint Statement of the American Thoracic Society (ATS) and the Centers for Disease Control and Prevention (CDC). This statement was endorsed by the Council of the Infectious Diseases Society of America (IDSA), September 1999, and the sections of this statement. *Am J Respir Crit Care Med* 2000; 161:S221.
89. Jick SS, Lieberman ES, Rahman MU, Choi HK. Glucocorticoid use, other associated factors, and the risk of tuberculosis. *Arthritis Rheum* 2006; 55:19.
90. Dong YH, Chang CH, Lin Wu FL, et al. Use of inhaled corticosteroids in patients with COPD and the risk of TB and influenza: A systematic review and meta-analysis of randomized controlled trials. a systematic review and meta-analysis of randomized controlled trials. *Chest* 2014; 145:1286.
91. Lichtenstein IH, MacGregor RR. Mycobacterial infections in renal transplant recipients: report of five cases and review of the literature. *Rev Infect Dis* 1983; 5:216.
92. Muñoz P, Palomo J, Muñoz R, et al. Tuberculosis in heart transplant recipients. *Clin Infect Dis* 1995; 21:398.
93. Meyers BR, Halpern M, Sheiner P, et al. Tuberculosis in liver transplant patients. *Transplantation* 1994; 58:301.
94. Akan H, Arslan O, Akan OA. Tuberculosis in stem cell transplant patients. *J Hosp Infect* 2006; 62:421.
95. TB by Social Risk Factors. Centers for Disease Control and Prevention. <https://www.cdc.gov/tb-surveillance-report-2024/data/social-risk-factors.html> (Accessed on April 03, 2026).
96. Bates MN, Khalakdina A, Pai M, et al. Risk of tuberculosis from exposure to tobacco smoke: a systematic review and meta-analysis. *Arch Intern Med* 2007; 167:335.
97. Lin HH, Ezzati M, Chang HY, Murray M. Association between tobacco smoking and active tuberculosis in Taiwan: prospective cohort study. *Am J Respir Crit Care Med* 2009; 180:475.
98. Rao VG, Bhat J, Yadav R, et al. Tobacco smoking: a major risk factor for pulmonary tuberculosis--evidence from a cross-sectional study in central India. *Trans R Soc Trop Med Hyg* 2014; 108:474.
99. Slama K, Chiang CY, Enarson DA, et al. Tobacco and tuberculosis: a qualitative systematic review and meta-analysis. *Int J Tuberc Lung Dis* 2007; 11:1049.
100. Leung CC, Yew WW, Chan CK, et al. Smoking adversely affects treatment response, outcome and relapse in tuberculosis. *Eur Respir J* 2015; 45:738.
101. Deiss RG, Rodwell TC, Garfein RS. Tuberculosis and illicit drug use: review and update. *Clin Infect Dis* 2009; 48:72.
102. Durante AJ, Selwyn PA, O'Connor PG. Risk factors for and knowledge of Mycobacterium tuberculosis infection among drug users in substance abuse treatment. *Addiction* 1998; 93:1393.
103. Daley CL, Hahn JA, Moss AR, et al. Incidence of tuberculosis in injection drug users in San Francisco: impact of anergy. *Am J Respir Crit Care Med* 1998; 157:19.
104. Pevzner ES, Robison S, Donovan J, et al. Tuberculosis transmission and use of methamphetamines in Snohomish County, WA, 1991-2006. *Am J Public Health* 2010; 100:2481.
105. Gardy JL, Johnston JC, Ho Sui SJ, et al. Whole-genome sequencing and social-network analysis of a tuberculosis outbreak. *N Engl J Med* 2011; 364:730.
106. Lönnroth K, Williams BG, Stadlin S, et al. Alcohol use as a risk factor for tuberculosis - a systematic review. *BMC Public Health* 2008; 8:289.
107. Franco JV, Bongaerts B, Metzendorf MI, et al. Undernutrition as a risk factor for tuberculosis disease. *Cochrane Database Syst Rev* 2024; 6:CD015890.
108. PALMER CE, JABLON S, EDWARDS PQ. Tuberculosis morbidity of young men in relation to tuberculin sensitivity and body build. *Am Rev Tuberc* 1957; 76:517.
109. Edwards LB, Livesay VT, Acquaviva FA, Palmer CE. Height, weight, tuberculous infection, and tuberculous disease. *Arch Environ Health* 1971; 22:106.
110. Tverdal A. Body mass index and incidence of tuberculosis. *Eur J Respir Dis* 1986; 69:355.
111. Leung CC, Lam TH, Chan WM, et al. Lower risk of tuberculosis in obesity. *Arch Intern Med* 2007; 167:1297.

112. Gegielski JP, McMurray DN. The relationship between malnutrition and tuberculosis: evidence from studies in humans and experimental animals. *Int J Tuberc Lung Dis* 2004; 8:286.
113. Chandra G, Selvaraj P, Jawahar MS, et al. Effect of vitamin D3 on phagocytic potential of macrophages with live *Mycobacterium tuberculosis* and lymphoproliferative response in pulmonary tuberculosis. *J Clin Immunol* 2004; 24:249.
114. Sita-Lumsden A, Lapthorn G, Swaminathan R, Milburn HJ. Reactivation of tuberculosis and vitamin D deficiency: the contribution of diet and exposure to sunlight. *Thorax* 2007; 62:1003.
115. Wilkinson RJ, Llewelyn M, Toossi Z, et al. Influence of vitamin D deficiency and vitamin D receptor polymorphisms on tuberculosis among Gujarati Asians in west London: a case-control study. *Lancet* 2000; 355:618.
116. Ustianowski A, Shaffer R, Collin S, et al. Prevalence and associations of vitamin D deficiency in foreign-born persons with tuberculosis in London. *J Infect* 2005; 50:432.
117. Gibney KB, MacGregor L, Leder K, et al. Vitamin D deficiency is associated with tuberculosis and latent tuberculosis infection in immigrants from sub-Saharan Africa. *Clin Infect Dis* 2008; 46:443.
118. Martineau AR, Nhamoyebonde S, Oni T, et al. Reciprocal seasonal variation in vitamin D status and tuberculosis notifications in Cape Town, South Africa. *Proc Natl Acad Sci U S A* 2011; 108:19013.
119. Wallis RS, Zumla A. Vitamin D as Adjunctive Host-Directed Therapy in Tuberculosis: A Systematic Review. *Open Forum Infect Dis* 2016.
120. Aibana O, Huang CC, Aboud S, et al. Vitamin D status and risk of incident tuberculosis disease: A nested case-control study, systematic review, and individual-participant data meta-analysis. *PLoS Med* 2019; 16:e1002907.
121. Boelaert JR, Vandecasteele SJ, Appelberg R, Gordeuk VR. The effect of the host's iron status on tuberculosis. *J Infect Dis* 2007; 195:1745.
122. Cowie RL. The epidemiology of tuberculosis in gold miners with silicosis. *Am J Respir Crit Care Med* 1994; 150:1460.
123. Corbett EL, Churchyard GJ, Clayton TC, et al. HIV infection and silicosis: the impact of two potent risk factors on the incidence of mycobacterial disease in South African miners. *AIDS* 2000; 14:2759.
124. Kleinschmidt I, Churchyard G. Variation in incidences of tuberculosis in subgroups of South African gold miners. *Occup Environ Med* 1997; 54:636.
125. Kamboj M, Sepkowitz KA. The risk of tuberculosis in patients with cancer. *Clin Infect Dis* 2006; 42:1592.
126. Jeon CY, Murray MB. Diabetes mellitus increases the risk of active tuberculosis: a systematic review of 13 observational studies. *PLoS Med* 2008; 5:e152.
127. Baker MA, Harries AD, Jeon CY, et al. The impact of diabetes on tuberculosis treatment outcomes: a systematic review. *BMC Med* 2011; 9:81.
128. Ugarte-Gil C, Alisjahbana B, Ronacher K, et al. Diabetes Mellitus Among Pulmonary Tuberculosis Patients From 4 Tuberculosis-endemic Countries: The TANDEM Study. *Clin Infect Dis* 2020; 70:780.
129. Eckold C, Kumar V, Weiner J, et al. Impact of Intermediate Hyperglycemia and Diabetes on Immune Dysfunction in Tuberculosis. *Clin Infect Dis* 2021; 72:69.
130. Baker MA, Lin HH, Chang HY, Murray MB. The risk of tuberculosis disease among persons with diabetes mellitus: a prospective cohort study. *Clin Infect Dis* 2012; 54:818.
131. Yoon YS, Jung JW, Jeon EJ, et al. The effect of diabetes control status on treatment response in pulmonary tuberculosis: a prospective study. *Thorax* 2017; 72:263.
132. Franco JV, Bongaerts B, Metzendorf MI, et al. Diabetes as a risk factor for tuberculosis disease. *Cochrane Database Syst Rev* 2024; 8:CD016013.
133. Restrepo BI, Fisher-Hoch SP, Pino PA, et al. Tuberculosis in poorly controlled type 2 diabetes: altered cytokine expression in peripheral white blood cells. *Clin Infect Dis* 2008; 47:634.
134. Hussein MM, Mooij JM, Roujouleh H. Tuberculosis and chronic renal disease. *Semin Dial* 2003; 16:38.
135. Pien FD, Younoszai BG, Pien BC. Mycobacterial infections in patients with chronic renal disease. *Infect Dis Clin North Am* 2001; 15:851.
136. THORN PA, BROOKES VS, WATERHOUSE JA. Peptic ulcer, partial gastrectomy, and pulmonary tuberculosis. *Br Med J* 1956; 1:603.
137. Steiger Z, Nickel WO, Shannon GJ, et al. Pulmonary tuberculosis after gastric resection. *Am J Surg* 1976; 131:668.
138. Bruce RM, Wise L. Tuberculosis after jejunioileal bypass for obesity. *Ann Intern Med* 1977; 87:574.
139. Ludvigsson JF, Wahlstrom J, Grunewald J, et al. Coeliac disease and risk of tuberculosis: a population based cohort study. *Thorax* 2007; 62:23.
140. Thulstrup AM, Mølle I, Svendsen N, Sørensen HT. Incidence and prognosis of tuberculosis in patients with cirrhosis of the liver. A Danish nationwide population based study. *Epidemiol Infect* 2000; 124:221.
141. Lin YT, Wu PH, Lin CY, et al. Cirrhosis as a risk factor for tuberculosis infection--a nationwide longitudinal study in Taiwan. *Am J Epidemiol* 2014; 180:103.
142. Inghammar M, Ekblom A, Engström G, et al. COPD and the risk of tuberculosis--a population-based cohort study. *PLoS One* 2010; 5:e10138.
143. Lee CH, Lee MC, Shu CC, et al. Risk factors for pulmonary tuberculosis in patients with chronic obstructive airway disease in Taiwan: a nationwide cohort study. *BMC Infect Dis* 2013; 13:194.
144. Hochberg NS, Horsburgh CR Jr. Prevention of tuberculosis in older adults in the United States: obstacles and opportunities. *Clin Infect Dis* 2013; 56:1240.
145. Comstock GW. Epidemiology of tuberculosis. *Am Rev Respir Dis* 1982; 125:8.
146. WHO. The Stop TB Strategy: Building on and Enhancing DOTS to meet the TB-Related Millennium Development Goals <https://apps.who.int/iris/handle/10665/69241> (Accessed on December 08, 2011).
147. Becerra MC, Appleton SC, Franke MF, et al. Tuberculosis burden in households of patients with multidrug-resistant and extensively drug-resistant tuberculosis: a retrospective cohort study. *Lancet* 2011; 377:147.
148. Marks SM, Taylor Z, Qualls NL, et al. Outcomes of contact investigations of infectious tuberculosis patients. *Am J Respir Crit Care Med* 2000; 162:2033.
149. Zuber PL, McKenna MT, Binkin NJ, et al. Long-term risk of tuberculosis among foreign-born persons in the United States. *JAMA* 1997; 278:304.
150. Tsang CA, Langer AJ, Navin TR, Armstrong LR. Tuberculosis Among Foreign-Born Persons Diagnosed ≥ 10 Years After Arrival in the United States, 2010-2015. *MMWR Morb Mortal Wkly Rep* 2017; 66:295.
151. McKenna MT, McCray E, Jones JL, et al. The fall after the rise: Tuberculosis in the United States, 1991 through 1994. *Am J Public Health* 1998; 88:1059.
152. Liu Y, Weinberg MS, Ortega LS, et al. Overseas screening for tuberculosis in U.S.-bound immigrants and refugees. *N Engl J Med* 2009; 360:2406.
153. Barnett ED, Weld LH, McCarthy AE, et al. Spectrum of illness in international migrants seen at GeoSentinel clinics in 1997-2009, part 1: US-bound migrants evaluated by comprehensive protocol-based health assessment. *Clin Infect Dis* 2013; 56:913.
154. Walter ND, Painter J, Parker M, et al. Persistent latent tuberculosis reactivation risk in United States immigrants. *Am J Respir Crit Care Med* 2014; 189:88.

155. Aldridge RW, Zenner D, White PJ, et al. Tuberculosis in migrants moving from high-incidence to low-incidence countries: a population-based cohort study of 519 955 migrants screened before entry to England, Wales, and Northern Ireland. *Lancet* 2016; 388:2510.
156. Ribeiro FK, Pan W, Bertolde A, et al. Genotypic and Spatial Analysis of Mycobacterium tuberculosis Transmission in a High-Incidence Urban Setting. *Clin Infect Dis* 2015; 61:758.
157. MacIntyre CR, Kendig N, Kummer L, et al. Impact of tuberculosis control measures and crowding on the incidence of tuberculous infection in Maryland prisons. *Clin Infect Dis* 1997; 24:1060.
158. Stead WW, Lofgren JP, Warren E, Thomas C. Tuberculosis as an endemic and nosocomial infection among the elderly in nursing homes. *N Engl J Med* 1985; 312:1483.
159. Schieffelin CW Jr, Snider DE Jr. Tuberculosis control among homeless populations. *Arch Intern Med* 1988; 148:1843.
160. Pablos-Mendez A, Raviglione MC, Battan R, Ramos-Zuniga R. Drug resistant tuberculosis among the homeless in New York City. *N Y State J Med* 1990; 90:351.
161. Centers for Disease Control (CDC). Drug-resistant tuberculosis among the homeless--Boston. *MMWR Morb Mortal Wkly Rep* 1985; 34:429.
162. Nolan CM, Elarth AM, Barr H, et al. An outbreak of tuberculosis in a shelter for homeless men. A description of its evolution and control. *Am Rev Respir Dis* 1991; 143:257.
163. Nardell E, McInnis B, Thomas B, Weidhaas S. Exogenous reinfection with tuberculosis in a shelter for the homeless. *N Engl J Med* 1986; 315:1570.
164. Centers for Disease Control and Prevention (CDC). Tuberculosis outbreak associated with a homeless shelter - Kane County, Illinois, 2007-2011. *MMWR Morb Mortal Wkly Rep* 2012; 61:186.
165. Cantwell MF, McKenna MT, McCray E, Onorato IM. Tuberculosis and race/ethnicity in the United States: impact of socioeconomic status. *Am J Respir Crit Care Med* 1998; 157:1016.
166. Deutsch-Feldman M, Pratt RH, Price SF, et al. Tuberculosis - United States, 2020. *MMWR Morb Mortal Wkly Rep* 2021; 70:409.