

## REVIEW

# Tuberculosis as an infectious disease and its prevalence in society current status

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## ABSTRACT

Since the beginning of recorded history, tuberculosis has been and continues to be a major health concern all over the world. Tuberculosis (TB) is a disease that is caused by bacteria that are passed from person to person through the air. In the past 15 years, the number of new cases of TB as well as the number of deaths has been declining. However, TB is still a very serious condition. The lungs are the most common target of tuberculosis infection, nevertheless, the disease can attack and harm any region of the body, including the brain, kidneys, or spine. Some of the general signs of tuberculosis disease include feelings of sickness or weakness, loss of weight, fever, and sweating at night. In addition to coughing, chest pain, and blood in the sputum, other symptoms of tuberculosis lung illness may include: Signs and symptoms of tuberculosis. The treatment of tuberculosis disease includes the use of various medications, each of which must be taken for a period ranging from six months to nine months (or even longer in the case of drug-resistant TB), depending on the chosen regimen. The treatment for drug-resistant tuberculosis is laborious, time consuming, difficult, and costly. It has the ability to inflict damage on people's lives and cause catastrophic, even fatal, adverse effects. What happens in other regions of the body is dependent on the location that is affected. In this article we focus on infection of TB and its prevalence.

**Key words:** tuberculosis, *Mycobacterium bovis*, lungs, drug-resistant

## INTRODUCTION

Since the beginning of the 1990s, the world has recognised tuberculosis as a major threat to public health, as it is the infectious disease that is responsible for the most deaths among adults all over the world.

*Mycobacterium tuberculosis* and *Mycobacterium bovis* are the two species of mycobacteria that are responsible for the infection that is known as tuberculosis (TB).<sup>[1,2]</sup> In industrialised nations, tuberculosis was thought to be under control not too long ago; however, the disease has recently returned in epidemic proportions, demonstrating that considerable effort remains to be done to protect patients and health care professionals from the disease's potentially fatal effects.<sup>[3,4]</sup> Molecular evidence suggests that tuberculosis has been affecting humans for more than 17,000 years, making it one of the world's oldest infectious diseases.<sup>[5]</sup> Despite the development of more recent methods for the detection and treatment of tuberculosis, unfortunately, individuals are still being affected by it, and on a global scale, it is among the top 10 infectious diseases that cause death, coming in second only to human immunodeficiency virus (HIV). The World Health Organization (WHO)

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Received: 20 September 2022; Revised: 11 July 2023; Accepted: 26 July 2023;

Published: 14 August 2023

<https://doi.org/10.54844/cai.2022.0152>

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describes tuberculosis as a pandemic that has spread throughout the entire world. It is the most common cause of mortality for those who are living with HIV.<sup>[6-8]</sup> Historically speaking, the fight against tuberculosis in India can be broken down into three distinct periods: The early period, which occurred before the invention of X-rays and chemotherapy; the post-independence period, which was the time period in which nationwide TB control programmes were initiated and implemented; and the current period, which is the time period in which an ongoing TB control programme that is assisted by the WHO is in place.<sup>[9]</sup> India's DOTS (directly observed therapy-short course) programme is currently the programme that is increasing at the fastest rate and is the largest programme in the world in terms of the number of patients who have begun treatment. In terms of population coverage, it is the second largest programme. Poor primary health-care infrastructure in rural parts of many states is one of the most significant obstacles in India's fight against tuberculosis.<sup>[10]</sup>

## HISTORY OF TUBERCULOSIS

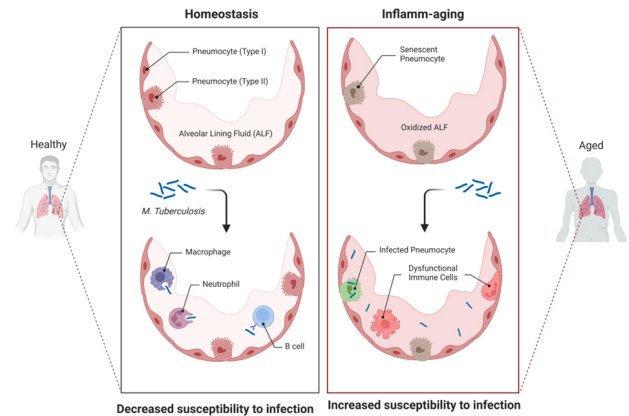
During the Paleolithic period, people lived as nomads; they did not establish permanent communities such as villages or locations; and they did not get together in large groups. Although cases of tuberculosis may have occurred on occasion, the disease, along with other infectious diseases, most likely did not spread rapidly enough to cause epidemics.<sup>[11,12]</sup> Around 8000 BC, humans developed the first agricultural techniques, which enabled them to settle in permanent locations. Along with this advancement came the domestication of cattle, pigs, and sheep. In spite of the fact that tuberculosis was most likely diagnosed more frequently in this environment, the disease was still quite uncommon. According to McGrath's calculations, a stable host-pathogen relationship, which is necessary for tuberculosis infection to become endemic in a community, requires a social network of between 180 and 440 people in order to be established.<sup>[12,13]</sup> It is likely that tuberculosis was present as an endemic disease among animals a very long time before it was found in humans. *M. bovis* was the most likely organism to have caused infection, and it's possible that *M. bovis* was responsible for the first human infections. Since *M. tuberculosis* can infect any primate species, it is possible that this species was present in non-human primates before it was found in humans.<sup>[14]</sup> This hypothesis is supported by the fact that *M. tuberculosis* can spread between primates. Because of this shift, the environment began to change, which was associated with a shift in the delicate balance that existed between humans and the tubercle bacillus. As the centuries and millennia passed, human beings began to live in larger and larger communities. Two competing hypotheses have been put

forward to explain the rapid spread of the tuberculosis epidemic and the subsequent decline in its incidence that followed.<sup>[15]</sup> The first explanation, which is the one that is most commonly accepted, involves the evolution of genetically determined herd immunity. In particular, in comparison to their hosts, parasites tend to have shorter lifespans. This characteristic confers a significant benefit on parasites because it allows mutations to take place in them at a higher rate than in their hosts when the latter are subjected to the same environmental stresses. It is impossible for the host to adapt at the same rate as the parasite when the generation time of the host is significantly longer than that of the parasite, as is the case with humans and the tubercle bacillus. Therefore, the parasite starts off with a significant advantage and begins to wipe out the vulnerable members of the species before those individuals can pass on their genes to their offspring. However, because not all hosts of a species are eradicated, the offspring of those hosts that do survive form a subset of the population that is distinguished by an increased resistance to that specific parasite. Therefore, as more generations pass, the once grave infection that posed a threat to the host's life becomes less devastating. This happens because the parasite's highly advantageous position gradually deteriorates over time. This is likely the reason why there has never been a case of an infectious disease completely wiping out its host population.<sup>[16]</sup>

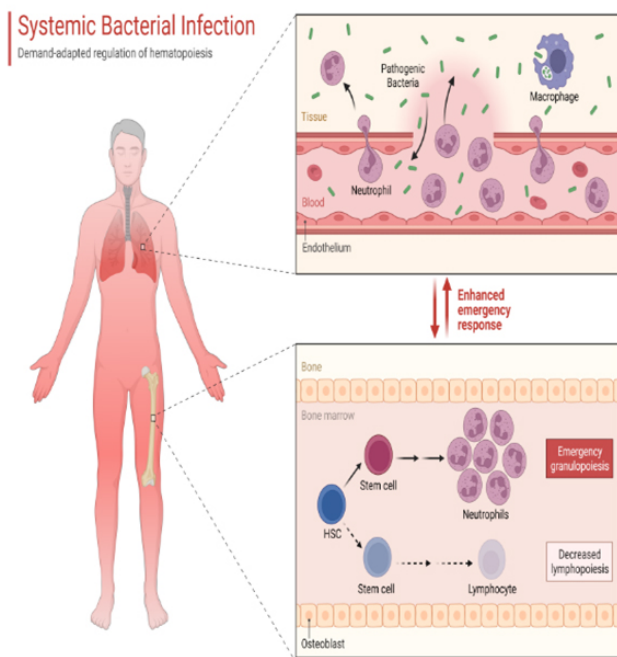
## PATHOGENESIS OF TB

*M. tuberculosis* is an obligatory aerobic intracellular pathogen. It has a predilection for lung tissue that is rich in oxygen supply. The route of infection and the site of infection are both the same. The bacilli that cause tuberculosis enter the body through the respiratory system.<sup>[4]</sup> Bacilli spread from the lung, where the infection started, to other parts of the body through the lymphatic system or the blood. The top of the lung and the lymph node in the area are especially good places for this to happen. The first event in the host-pathogen relationship that determines the outcome of an infection is called phagocytosis, and it is performed by alveolar macrophages on *M. tuberculosis*.<sup>[13,14]</sup> Within two to six weeks of the infection, cell-mediated immunity (CMI) develops, and there is an influx of lymphocytes and activated macrophages into the lesion, which ultimately results in the formation of granulomas. Interactions between macrophages and mycobacteria, as well as the role of macrophages in the host's immune response, can be summed up as follows: The attachment of *M. tuberculosis* to the surface of macrophages; the inhibition or killing of mycobacterial growth; the recruitment of accessory immune cells for a local inflammatory response; and the presentation of antigens to T cells for the development of acquired immunity.<sup>[5]</sup> The

attachment of *M. tuberculosis* to monocytes and macrophages is as follows: When it comes to the process of binding the organisms to the phagocytes, the complement receptors (CR1, CR2, CR3, and CR4), mannose receptors (MR), and other cell surface receptor molecules all play an important part. It appears that the mycobacterial surface glycoprotein lipoarabinomannan acts as a mediator for the interaction between phagocytic MR and mycobacteria (LAM). Microorganisms that have been phagocytosed are degraded by intralysosomal acidic hydrolases upon phagolysosome fusion. Figure 1 showing the elderly mucosa environment increases susceptibility and Figure 2 indicating the mechanism of systemic bacterial infection.



**Figure 2.** Mechanism of systemic bacterial infection.



**Figure 1.** The elderly lung mucosa environment increases susceptibility to *M. tuberculosis* infection.

## TUBERCULOSIS IN CHILDREN

Transmission, exposure, and infection, the likelihood, duration, and distance of an exposure to an infectious case, as well as the infectious potential of the source, all play a role in determining a child's possibility of being infected with *M. tuberculosis*.<sup>[17–19]</sup> Although older children are more likely to be the source of transmission, the cavitary pulmonary disease source is typically an adult. However, transmission can also occur between older children. The most likely place for a person to get tuberculosis depends on their age and how common tuberculosis is in their community.<sup>[20]</sup> Younger children are more likely to have their infection traced back to a household source, whereas older children are more likely to have their infection traced back to an outside source.

There is a correlation between increased rates of transmission and factors such as poverty, inadequate housing, urban environments, and overcrowding. The yearly risk of infection is the metric that is used to assess transmission within a community (ARI).<sup>[21]</sup> Increased social mobility in late adolescence and early adulthood is associated with higher infection rates. This is also true when considering the correlation between increased exposure and higher infection rates in toddlers. ARI is traditionally estimated through the use of childhood tuberculin surveys. However, this method has limitations as a result of the poor specificity of the tuberculin skin test (TST).<sup>[22]</sup> This is especially true in areas where the Bacille Calmette-Guérin (BCG) vaccine is administered at birth and non-tuberculous mycobacteria are endemic. T-cell-based interferon-release assays (IGRAs) could provide a more specific alternative. However, they have not yet found a use in this context due to the high cost of the tests, ethical concerns regarding venepuncture in healthy children, and uncertainty regarding the association between a positive result and the later development of active disease.<sup>[23]</sup>

## THE PROGRESSION FROM INFECTION TO DISEASE

It is more difficult to diagnose tuberculosis in children than in adults due to differences in the pathophysiology and clinical presentation of the disease. Also, it's not as easy to tell the difference between a latent infection and an active disease in children.<sup>[12,19]</sup> However, following infection, several factors, such as age, nutritional, vaccination, and immune status, influence the balance of risk between latent tuberculosis infection and progression to active disease. These factors include the presence of a tuberculosis latent infection. When compared to adults, the likelihood of a disease becoming active in a child is significantly higher. This risk is greatest for children younger than 2 years old, partic-

ularly infants and young children. Active surveillance data from the era before chemotherapy suggests that the majority of children developed radiological abnormalities following infection, including 60%-80% of children under the age of 2; however, less than 10% of these were notified, suggesting that the disease was controlled by the host immune response in the majority of cases.<sup>[9]</sup> The findings of these studies have repercussions for case definitions that are determined by radiological findings. The risk of disease was lowest in children aged between 5 and 10 years old, which are considered to be the "safe school years". The risk of disease was greatest in infants and individuals in their late teens overall. The risk was lowest in children aged between 5 and 10 years. The majority of cases of disease appear during the first year after infection. The paediatric disease burden provides a potentially meaningful measure of current transmission inside a community, such as multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains. This is due to the fact that disease in young children reflects recent infection rather than supplementary reactivation. A latent tuberculosis infection that is not treated can sow the seeds for a future outbreak of the disease in subsequent generations.

## METHODS OF DIAGNOSIS IN CHILDREN

Most of the time, children are checked for tuberculosis when they show signs or symptoms of the disease (this is called "passive case finding"), when a contact investigation is done, or during a routine immigration screening (active case finding).<sup>[21]</sup> The clinical manifestations of children whose infection is detected through active case finding are different from those of children whose infection is detected through passive case finding. The children in the former group frequently have infection but not disease or have disease in a very early phase. The clinical presentation of children whose infection is detected through passive case finding is similar to that of adults.<sup>[23]</sup> Young children and those who have recently been exposed to *M. tuberculosis* are at a higher risk of developing the disease than children who have tested positive for *M. tuberculosis* infection.

A brand-new test called the Xpert MTB/RIF assay is transforming the way that tuberculosis is managed by helping to quickly diagnose the disease and treatment resistance associated with TB. In less than two hours, the test can identify *M. tuberculosis* complex (MTBC) and rifampin resistance (RIF). Comparatively, MTBC can grow in standard cultures in 2 to 6 weeks, and traditional drug resistance tests can take an additional 3 weeks. The Xpert MTB/RIF assay's information helps with treatment regimen selection and hastens infection control decisions.

The GeneXpert Instrument System uses a disposable cartridge for the nucleic acid amplification (NAA) test known as the Xpert MTB/RIF assay. The patient with probable TB has a sputum sample taken. The test reagent and the sputum are combined, and a cartridge holding this mixture is put into the GeneXpert machines. From this point on, the entire processing is automated.<sup>[24]</sup>

Even though there have been many improvements in how tuberculosis is diagnosed, there is still no simple, reliable, point-of-care test that can find the disease for sure. Clinicians frequently seek a bacteriological diagnosis, although this is complemented by clinical symptoms, radiographic evidence, and tests for bacterial products that suggest the presence of *M. tuberculosis*.<sup>[25]</sup> Current WHO endorsements include a variety of diagnostic and drug susceptibility tests. There have been recent advancements in the use of radiographic screening and diagnosis for tuberculosis, and interest in this field is growing. Digital chest X-rays with computer-assisted tuberculosis detection have been increasingly used in a variety of situations, including prisons, among family connections, and for miners.<sup>[26,27]</sup> Although more study is needed to optimise the use of computer-aided detection, chest x-ray appears to be making a comeback as a triage test, and WHO currently recommends this evaluation approach for screening and diagnosis of tuberculosis in some groups. When tuberculosis bacteria proliferate in a human host, they release various proteins and by products. One of these substances, lipoarabinomannan, is the basis for the urinary LAM test, the usage of which has been related to a decrease in tuberculosis mortality.<sup>[28]</sup>

## HIV AND TUBERCULOSIS CORRELATION ASSOCIATED WITH DEATH FACTOR

Studies from several regions of the world have shown that HIV-positive people are more likely to get TB, with 5 to 10 cases per year of observation. This is in stark contrast to the 10% lifetime risk of TB for people who do not have HIV. People with HIV have a higher chance of having a new infection get worse quickly and of having a dormant infection come back to life.<sup>[29]</sup> TB is the most common opportunistic infection among HIV-positive people in India, and studies from different parts of the country have found that 60% to 70% of HIV-positive people will get TB at some point in their lives. HIV-positive TB is different from HIV-negative TB in a number of ways, including a higher number of cases with extra-pulmonary or disseminated disease; a higher number of false-negative tuberculin skin tests; atypical features on chest radiographs; fewer cavitating lung lesions; a higher number of bad drug reactions; the presence of other acquired immunodeficiency syndrome



(AIDS)-related symptoms; and a higher death rate.<sup>[30]</sup>

Both TB and HIV infections happen inside cells, and it is known that they have a big effect on each other's progress. When someone has HIV, their CD4+ T cells go down. CD4+ T cells are a big part of their immunity to TB. This is shown by how well the granuloma, which is part of the cellular immune response, works.<sup>[31]</sup> Apart from making CD4+ and CD8+ cells less numerous, HIV also changes how they work. In the same way, TB infection speeds up the progression of HIV from an infection with no symptoms to AIDS and death.

## PREVALENCE AND INCIDENCE

Tuberculosis remains the single largest infectious disease causing high mortality in humans, leading to 3 million deaths annually, about five deaths every minute. Approximately 8-10 million people are infected with this pathogen every year. Out of the total number of cases, 40 percent of cases are accommodated in South East Asia alone. In India, there are about 500,000 deaths occurring annually due to TB, with the incidence and prevalence being 1.5 and 3.5 million per year.<sup>[31]</sup>

According to estimates, India has an average prevalence of 5.05 cases of tuberculosis overall, a prevalence of 2.27 instances of smear-positive cases, and an average yearly incidence of 84 cases of smear-positive cases per 100,000 people. There is a thorough discussion of the estimates' use and trustworthiness. Reports on recent research on the disease's temporal pattern from several Indian regions, such as Chingleput in Tamil Nadu, are discussed. They confirm the gradual decrease trend that has been observed over a sizable amount of time, as in the rural areas near Bangalore. Additionally, it describes the alarming rise in disease prevalence among a tribe in Car Nicobar between 1986 and 2002 and emphasises the type and scope of new dangers. According to some epidemiologists, India will see a 20% increase in incidence over the next 20 years, with a total increase of 46 million cases of tuberculosis during that time, partly as a result of the HIV epidemic. Data are supplied and assessed on the government's attempts to intervene through the Revised National Tuberculosis Control Program (RNTCP) and to monitor the epidemiology of intervention by organising routine reporting. RNTCP must be utilised as a powerful tool to improve the epidemiological condition through rapid expansion and the accomplishment of a global goal. The purpose of the antituberculosis intervention efforts is discussed in the context of the current review's description of the worldwide tuberculosis situation. The epidemiological situation in India is discussed, along with the current trend and the efforts made to forecast the expected future burden of illness in India.<sup>[32]</sup>

The burden of tuberculosis sickness across the world continues to be alarmingly high. According to studies of the population, there are around 14 million people who are affected by the disease, which translates to approximately 10 million new cases each year, of which approximately six million are identified and treated. A key part of global and national tuberculosis care and prevention programs is finding the millions of people who are still lacking of treatment. This will help close the gap between the estimated number of people who have tuberculosis disease and the number of people who are receiving treatment. On the other hand, such measures rest on the assumption that we are fully aware of the extent to which tuberculosis sickness impacts society, which is probably definitely not the case. Instead, the estimations that are now being used most likely just measure the proverbial tip of the iceberg, which means that they could be missing potentially millions of additional people who have tuberculosis disease that is prevalent (that is, currently present).<sup>[7]</sup> To measure the number of people who suffer from what is known as active pulmonary tuberculosis disease, which is defined as bacteriologically confirmed tuberculosis from two sputum samples that are tested for the presence of *M. tuberculosis* using culture or PCR-based tests, current methods for estimating the prevalence of tuberculosis disease aim to count the number of people who have the so-called active form of the disease. In addition to the emergence of sputum-positive subclinical tuberculosis disease that is detected by current methods, we know that increasing the number or type (for example, induced sputum or Broncho alveolar lavage) of samples will identify additional sputum bacteriologically confirmed tuberculosis, be it clinical or subclinical. This is true regardless of whether the disease is detected by current methods. More crucially, sputum-negative people who breathe *M. tuberculosis* in high numbers and likely contribute to transmission have been detected through thorough bio aerosol sampling with face masks.<sup>[33]</sup>

In the first population-based national tuberculosis prevalence survey in Ethiopia from 2010 to 2011, which was published in 2014, the prevalence of bacteriologically positive pulmonary tuberculosis was found to be 277/100,000 population. This was lower than the estimated incidence of tuberculosis reported in the Global Tuberculosis Reports of 2011 and 2012. Senkoro and his colleagues did a national survey of the number of people with tuberculosis in Tanzania in 2012.<sup>[32]</sup> The United Republic of Tanzania is in the top 20 of the 30 countries with the most cases of tuberculosis. Bacteriologically positive pulmonary tuberculosis was found in 293 out of every 100,000 people. The number of people who had it was higher among men and in rural areas. The estimated number of cases of tuberculosis was much lower than the number of cases that were found in

the survey. Qadeer *et al.*<sup>[34]</sup> conducted a population-based national tuberculosis prevalence survey in Pakistan. They found that 398 out of every 100,000 people had bacteriologically positive pulmonary tuberculosis. The number of people with tuberculosis rose with age, and men had it 1.8 times more often than women.<sup>[32–34]</sup>

## CONCLUSION

Tuberculosis is one of the most lethal infectious diseases. Diseases have killed millions of people over the years and have been the cause of their deaths. Despite the fact that a considerable amount of work has been made towards reducing the global impact of tuberculosis throughout the course of the last decade, further efforts are still needed. Emerging problems, such as multidimensionality, resistant to drugs poses a risk of rolling back the gains that have been obtained. Concerning treatment and prevention of tuberculosis. The information repository for tuberculosis. Continues to be a field that is continuously growing, and worldwide rules are always being improved, for example by incorporating fresh information medicines against tuberculosis in an effort to combat issues of resistance.

## DECLARATION

### Acknowledgement

We, thank you to the R.C. Patel Institute of Pharmaceutical Education and Research Shirpur, Maharashtra, India, and METs, Institute of Pharmacy, Adgoan, Nashik, MH, India, for their constant support and providing all facilities to complete this work.

### Author Contributions

Pathan A wrote and revised the first draft. English editing was done by Ahire ED. Proofreading and editing of final draft was performed by Shelke RU and Keservani RK. All authors are agreed the final version and submitted the article.

### Funding

This review received no external funding.

### Ethics approval

Not applicable.

### Conflict of interest

Eknath D Ahire and Raj K Keservani are Editorial Board Members of the journal. The article was subject to the journal's standard procedures, with peer review handled independently of two editors and their research groups.

### Data sharing

Not applicable.

## REFERENCES

1. Nyarko RO, Prakash A, Kumar N, Saha P, Kumar R. Tuberculosis a globalized disease: review. *Asian J Pharm Res Dev.* 2021;9(1):198–201.
2. Stubbs B, Siddiqi K, Elsey H, *et al.* Tuberculosis and non-communicable disease multimorbidity: an analysis of the world health survey in 48 low- and middle-income countries. *Int J Environ Res Public Health.* 2021;18(5):2439.
3. Di Gennaro F, Gualano G, Timelli L, *et al.* Increase in tuberculosis diagnostic delay during first wave of the COVID-19 pandemic: data from an Italian infectious disease referral hospital. *Antibiotics.* 2021;10(3):272.
4. Sultana ZZ, Hoque FU, Beyene J, *et al.* HIV infection and multidrug resistant tuberculosis: a systematic review and meta-analysis. *BMC Infect Dis.* 2021;21(1):51.
5. Goletti D, Delogu G, Matteelli A, Migliori GB. The role of IGRA in the diagnosis of tuberculosis infection, differentiating from active tuberculosis, and decision making for initiating treatment or preventive therapy of tuberculosis infection. *Int J Infect Dis.* 2022;124(Suppl 1):S12–S19.
6. Yu WY, Wang YX, Mei JZ, Hu FX, Ji LC. Overview of tuberculosis. In: Yu Wy, Lu PX, Tan Wg, eds. *Tuberculosis Control in Migrating Population.* Springer; 2020:1-10.
7. Sinha P, Lönnroth K, Bhargava A, *et al.* Food for thought: addressing undernutrition to end tuberculosis. *Lancet Infect Dis.* 2021;21(10):e318–e325.
8. Kirtane AR, Verma M, Karandikar P, Furin J, Langer R, Traverso G. Nanotechnology approaches for global infectious diseases. *Nat Nanotechnol.* 2021;16(4):369–384.
9. Hermans SM, Zinyakatira N, Caldwell J, Cobelens FGJ, Boule A, Wood R. High rates of recurrent tuberculosis disease: a population-level cohort study. *Clin Infect Dis.* 2021;72(11):1919–1926.
10. Chakaya J, Petersen E, Nantanda R, *et al.* The WHO Global Tuberculosis 2021 Report - not so good news and turning the tide back to End TB. *Int J Infect Dis.* 2022;124(Suppl 1):S26–S29.
11. Behr MA, Kaufmann E, Duffin J, Edelstein PH, Ramakrishnan L. Latent tuberculosis: two centuries of confusion. *Am J Respir Crit Care Med.* 2021;204(2):142–148.
12. Huaman MA, De Cecco CN, Bittencourt MS, *et al.* Latent tuberculosis infection and subclinical coronary atherosclerosis in Peru and Uganda. *Clin Infect Dis.* 2021;73(9):e3384–e3390.
13. Migliori GB, Wu SJ, Matteelli A, *et al.* Clinical standards for the diagnosis, treatment and prevention of TB infection. *Int J Tuberc Lung Dis.* 2022;26(3):190–205.
14. Soko RN, Burke RM, Feasey HRA, *et al.* Effects of coronavirus disease pandemic on tuberculosis notifications, Malawi. *Emerg Infect Dis.* 2021;27(7):1831–1839.
15. Zimmer AJ, Kinton JS, Oga-Omenka C, *et al.* Tuberculosis in times of COVID-19. *J Epidemiol Community Health.* 2022;76(3):310–6.
16. Khulbe P, Singh DM, Aman A, Ahire ED, Keservani RK. The emergence of nanocarriers in the management of diseases and disorders. *Community Acquir Infect.* 2023;10.
17. Al-Hadrawy SK, Alhadrawi KK, Aljanaby IAJ, Aljanaby AAJ, Zabibah RS. Prevalence of pulmonary tuberculosis in Al-Najaf governorate, Iraq. *F1000 Res.* 2022;11:675.
18. Khawbung JL, Nath D, Chakraborty S. Drug resistant Tuberculosis: a review. *Comp Immunol Microbiol Infect Dis.* 2021;74:101574.
19. Rustage K, Lobe J, Hayward SE, *et al.* Initiation and completion of treatment for latent tuberculosis infection in migrants globally: a systematic review and meta-analysis. *Lancet Infect Dis.* 2021;21(12):1701–1712.
20. Surana KR, Parkhe AG, Ahire ED, *et al.* Current Therapeutic Targets for Neuropathic Pain. *Asian J Pharm Res.* 2022;12(1):96–104.

21. Ahire ED, Kshirsagar SJ. New hope in microbial multidrug resistance. *Community Acquir Infect.* 2022;9.
22. Al-Awadhi M, Ahmad S, Iqbal J. Current status and the epidemiology of malaria in the middle east region and beyond. *Microorganisms.* 2021;9(2):338.
23. Malenfant JH, Brewer TF. Rifampicin mono-resistant tuberculosis—a review of an uncommon but growing challenge for global tuberculosis control. *Open Forum Infect Dis.* 2021;8(2):ofab018.
24. Kinsella RL, Zhu DX, Harrison GA, *et al.* Perspectives and advances in the understanding of tuberculosis. *Annu Rev Pathol.* 2021;16:377–408.
25. Modjadji P. Communicable and non-communicable diseases coexisting in South Africa. *Lancet Glob Health.* 2021;9(7):e889–e890.
26. Asturiningtyas IP, Mulyantoro DK, Kusriani I, Ashar H. Non-communicable disease comorbidity and multimorbidity among people with tuberculosis in Indonesia. *Ann Trop Med Public Health.* 2021;24(1):24–191.
27. Kendall EA, Kitonsa PJ, Nalutaaya A, *et al.* The spectrum of tuberculosis disease in an urban Ugandan community and its health facilities. *Clin Infect Dis.* 2021;72(12):e1035–e1043.
28. Miller PB, Zalwango S, Galiwango R, *et al.* Association between tuberculosis in men and social network structure in Kampala, Uganda. *BMC Infect Dis.* 2021;21(1):1023.
29. de Mendonça EB, Schmaltz CA, Sant'Anna FM, *et al.* Correction: Anemia in tuberculosis cases: a biomarker of severity? *PLoS One.* 2021;16(3):e0249545.
30. Gong W, Wu X. Differential diagnosis of latent tuberculosis infection and active tuberculosis: a key to a successful tuberculosis control strategy. *Front Microbiol.* 2021;12:745592.
31. Noori T, Hargreaves S, Greenaway C, *et al.* Strengthening screening for infectious diseases and vaccination among migrants in Europe: what is needed to close the implementation gaps? *Travel Med Infect Dis.* 2021;39:101715.
32. Govender I, Karat AS, Olivier S, *et al.* Prevalence of *Mycobacterium tuberculosis* in sputum and reported symptoms among clinic attendees compared with a community survey in rural South Africa. *Clin Infect Dis.* 2022;75(2):314–322.
33. Thakur G, Thakur S, Thakur H. Status and challenges for tuberculosis control in India - Stakeholders' perspective. *Indian J Tuberc.* 2021;68(3):334–339.
34. Qadeer E, Fatima R, Yaqoob A, *et al.* Population based national tuberculosis prevalence survey among adults (> 15 years) in Pakistan, 2010–2011. *PLoS One.* 2016;11(2):e0148293.