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Exploring plant-based therapies for tuberculosis: A comprehensive review of natural alternatives for effective treatment

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Abstract

Tuberculosis (TB), is caused by Mycobacterium tuberculosis, continues to challenge global health systems, compounded by the emergence of multidrug-resistant strains and the limitations of existing therapies. In response, plant-based approaches offer a promising alternative, leveraging bioactive compounds with anti-microbial, anti-inflammatory, and immunomodulatory properties. This review highlights the pharmacological potential of medicinal plants, including Curcuma longa, Azadirachta indica, and Acalypha indica, in combating TB. These plants exhibit mechanisms such as disrupting mycobacterium cell wall synthesis, enhancing host immunity, and reducing inflammation. By integrating plant-derived compounds with conventional therapies, the potential for more effective, sustainable, and accessible TB treatments emerges. Further research into these bioactive molecules and their mechanisms of action is essential for overcoming current therapeutic barriers and advancing global TB control.

Keywords: Tuberculosis; Medicinal plants; Multidrug resistance-TB; Mycobacterium tuberculosis

1. Introduction :[1-8]

Tuberculosis (TB) is an airborne infectious disease caused by the bacteria *Mycobacterium tuberculosis* (Mtb), which is part of the *Mycobacterium tuberculosis complex* (MTBC) that includes other mycobacterium such as *M. bovis, M. africanum, M. canettii,* and *M. microti.* It is transmitted when a person with infectious TB expels droplet nuclei (airborne particles about 1-5 microns) through actions like coughing, sneezing, shouting, or singing, and these droplets are inhaled, reaching the alveoli of the lungs via the nasal passages, respiratory tract, and bronchi. Globally, TB has infected an estimated 2 billion people (about one-third of the world's population), with 8.7 million new cases and 1.4 million deaths reported in 2011, of which 13% were co-infected with HIV. The disease primarily affects the lungs (pulmonary TB) but can also spread through the blood to other organs like the kidneys, spine, or brain (extra pulmonary TB). Pulmonary TB is infectious, whereas extra pulmonary TB is not. While TB is preventable and treatable, it remains one of the deadliest infectious diseases. After a decline in cases during 2020, TB cases have been increasing since 2021, with 2023reporting the highest numbers since 2013, including 9,633 cases in the United States 15.6% increase from 2022. Enhanced strategies to diagnose and treat latent TB and active TB are crucial to reversing this trend and achieving the goal of TB elimination. Although the primary mode of transmission is person-to-person through airborne particles, TB can occasionally spread to humans from infected cows via unsterilized milk, though this plays a minor role in the disease's overall epidemiology.

Although it was anticipated that tuberculosis (TB) would be increasingly brought under control in many countries as the year 2000 approached, the last decade of the 20th century witnessed a global resurgence of the disease. In 1997,

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the World Health Organization (WHO) estimated 7.96 million TB cases worldwide, with half being highly infectious, and over 1.9 million deaths, making TB the fifth leading cause of death from a single infectious agent. By 1998, approximately1.9 billion individuals one-third of the global population were infected with the tubercle bacillus, with more than 95% of cases occurring in Africa, Asia, and Latin America. However, TB cases increased in nearly every country, leading to its declaration as a "global emergency." The re-emergence of TB was attributed to several factors, including rising poverty, not only in developing nations but also among marginalized groups in industrialized countries; demographic growth, with a high number of children born in TB-endemic regions reaching age groups where the disease is more prevalent; the AIDS epidemic, which escalated TB cases in regions heavily affected by HIV; negligence by health authorities in prioritizing TB control; and the lack of effective National Tuberculosis Programmers (NTPs) or reliance on outdated strategies that led to chaotic treatment and multidrug-resistant TB. WHO projected that by 2000,TB cases would surpass 8.4 million, causing over 2 million deaths annually, and without significant improvements in TB control, the following decade could see 80 million new cases and nearly 20 million deaths, predominantly among individuals aged 20-49 years, the most economically active population group. Nonetheless, model TB control programmers, supported by WHO and the International Union Against Tuberculosis.

2. Etiology of Tuberculosis (TB)

Tuberculosis (TB) is a contagious complaint primarily caused by *Mycobacterium tuberculosis*. It generally affects the lungs, though it can involve any organ in the body. The bacterium is transmitted from person to person through airborne patches when an individual with pulmonary TB coughs, sneezes, or speaks. This airborne transmission allows the bacteria to reach the lungs of a susceptible existent, where they may be getting infection.

The development of active TB depends on several factors, including the vulnerable status of the existent. When an individual inhales the bacteria, the body's vulnerable system responds by trying to contain the infection. In healthy individualities, the vulnerable system can frequently limit the spread of the bacteria, leading to a TB infection (LTBI). Still, in people with weakened vulnerable systems, similar as those with HIV/AIDS, malnutrition, or those witnessing immunosuppressive treatments, the bacteria can multiply, leading to active TB complaint.

Threat factors for TB also include dragged close contact with an infected person, living or working in crowded conditions, and being in regions with high TB frequency. Also, inheritable factors may play a part in vulnerability to TB, as some individualities may have inherited vulnerable system characteristics that make them more prone to developing active TB after exposure.

In addition to *M. tuberculosis*, other mycobacterium species can begetting TB such like conditions, including *Mycobacterium Bovis*, which primarily affects creatures but can also infect humans, generally through the consumption of unpasteurized milk or direct contact with infected creatures.

The pathogenesis of TB involves the original entry of *M. tuberculosis* into the lungs, where it's phagocytosed by macrophages. The bacterium is suitable to survive and replicate within these vulnerable cells, ultimately leading to granuloma conformation. This granuloma represents a controlled vulnerable response aimed at containing the infection. Still, if the granuloma fails to contain the bacteria, or if the vulnerable system is compromised, the bacteria can spread, leading to active complaint. Lung Disease in countries like Benin, China, Guinea, Nicaragua, Peru, and Tanzania, demonstrated effective strategies for combating the complaint.

3. Pathophysiology

Tuberculosis infections begin with the inhalation of *M. tuberculosis bacilli* into the airways, where they spread to the lungs, lymph bumps, and distant spots via the bloodstream. During haematogenous dispersion, mycobacterium can deposit near the ventricles or subarachnoid space, leading to granuloma conformation. Postmortem examination studies show this granuloma in the CNS, indeed in those without suspected CNS involvement. Rich and McCordock proposed that rupture of this granuloma, known as a "Rich focus," triggers the seditious response and initiates tuberculosis meningitis. Recent exploration suggests that variations in host impunity and M. tuberculosis strains may impact this process.

CNS infections by *M. tuberculosis* most frequently present as sub acute or habitual meningitis, or as tuberculoma, which can beget space- enwrapping lesions. CNS involvement can do alone or with pulmonary or circulated tuberculosis, the ultimate appertained to as Miliary tuberculosis. The vulnerable response to granuloma rupture leads to tuberculous exudates accumulation, which can beget vasculitis and infarctions in the cerebral arterial system, contributing to

neurological poverties. Cranial whim-whams impairment can affect from infarction or contraction. In a mouse model, increased TNF- nascence product in the exudates was linked to the complaint's progression. Hydrocephalus is more common and progressive in children than grown-ups with tuberculous meningitis.

4. Opinion

4.1. Treatment of TB complaint involves the following:

- Signs and symptoms harmonious with TB
- Casket-ray
- Clinical judgment
- Bacteriology, including
- AFB smear microscopy
- Nucleic Acid Modification Testing (NAAT)
- Culture and identification
- medicine vulnerability testing (DST)

5. Epidemiology [09]

Despite a recent worldwide decline in incident cases, tuberculosis remains a leading cause of death encyclopedically. One- third of the world's population is infected with Mycobacterium tuberculosis, which progresses to active complaint in roughly 10 of individualities. In 2015, the World Health Organization estimated 10.4 million incident cases of tuberculosis worldwide, with 10 being in children and 11 in individualities living with mortal immunodeficiency contagion (HIV). Tuberculous meningitis accounts for 1 – 2 of active tuberculosis cases, and in regions with concurrent HIV and tuberculosis pandemics, M. tuberculosis has come a leading cause of bacterial meningitis, alongside pathogens like Neisseria meningitidis, Haemophilus influenza, and Streptococcus pneumoniae. Unfortunately, about half of all tuberculous meningitis cases affect in severe disability or death.

6. Tb treatments [10-13]

6.1. TB treatment rules include

- TB Infection (LTBI) treatment options
- 9 months of Isoniazid
- 4 months of Rifampin
- 3 months of isoniazid plus Rifapentine
- TB complaint (pulmonary, medicine-susceptible TB)
- 6- month standard authority
- ferocious phase 2 months of Isoniazid, Rifampin, Ethambutol, and Pyrazinamide
- Durability phase 4 months of Isoniazid and Rifampin.

7. Treatments

7.1. Medicine-Resistant Tuberculous Meningitis

The global challenge of medicine- resistant tuberculosis is aggravated by the specific pitfalls posed by tuberculous meningitis, which has advanced case-casualty rate than other forms of tuberculosis. Treatment for known or suspected medicine-resistant tuberculous meningitis can be supported by remedial medicine monitoring, given the variable pharmacokinetics and narrower remedial indicator of numerous alternate-line anti-tuberculosis medicines.

7.2. Isoniazid Resistance

Isoniazid distributes freely into cerebrospinal fluid, both in the presence and absence of inflamed meninges, and demonstrates bactericidal exertion against *M.tuberculosis*. These pharmacokinetic and pharmacodynamic parcels punctuate its central part in treating tuberculous meningitis. Encyclopedically, Isoniazid nonresistance is the most current form of medicine- resistant tuberculosis. Among 1614 tuberculous meningitis cases in the USA over a 12- time

period, the odds of death before completing treatment were doubly as high among cases with Isoniazid- resistant complaint. The association between original Isoniazid resistance and death was also significant among 186 HIV-infected tuberculous meningitis cases in Vietnam, with the fresh observation that redundant mortality due to Isoniazid resistance didn't appear until after the first 60 days of treatment. Interestingly, this time-dependent relationship between Isoniazid resistance and death was also observed among tuberculous meningitis cases in New York City, USA.

Therefore, Isoniazid- resistant tuberculous meningitis may offer an occasion to impact the course of the complaint by early intensification of remedy. While boosted remedy with high- cure Rifampin and a fluoroquinolone didn't show an overall survival benefit among Vietnamese cases, there was a survival benefit among HIV- uninfected tuberculous meningitis cases with Isoniazid- resistant complaint. Importantly, the survival benefit of boosted treatment was topmost when the intensification passed at the launch of tuberculosis treatment, rather than in response to medicine vulnerability testing. Enforcing these findings in clinical practice would bear either a rapid-fire individual test for Isoniazid resistance in tuberculous meningitis cases or the use of threat factors for Isoniazid resistance, similar as tuberculosis contact history or the epidemiology of Isoniazid- resistant complaint in the original population.

7.3. Multidrug Resistance

Multidrug- resistant tuberculous meningitis, defined as resistance to both Isoniazid and Rifampin with or without resistance to other agents, carries a poor prognostic. The pronounced increase in mortality of tuberculous meningitis when rifampin resistance is added to isoniazid resistance underscores the significance of rifampin in the treatment authority, despite its limited penetration into cerebrospinal fluid compared to isoniazid. Over a 12- time period in the USA, 19 of 26(73) cases with multidrug- resistant tuberculous meningitis failed before completing tuberculosis treatment. Among 16 cases with multidrug- resistant tuberculous meningitis in the boosted remedy trial in Vietnam (including a single case with rifampin- resistant, isoniazid-susceptible complaint), 11 failed before completing treatment, with a median time to death of 27 days from treatment inauguration. Successful treatment of multidrug-resistant tuberculous meningitis is primarily reported in case studies, including the use of intrathecal administration of levofloxacin and amikacin. Linezolid may also be a useful alternate- line medicine grounded on recent clinical tests in Chinese children and grown-ups.

7.4. Long- Term Neurologic issues Among Survivors

Neurologic sequela performing from tuberculous meningitis includes hydrocephalus, stroke, cranial whim-whams paralysis, seizures, and mass lesions, with the threat of these complications adding with individual detainments. In a clinical trial of dexamethasone in Vietnam, 14 of actors had severe disability at 5 times of follow- up, with no difference between the dexamethasone and placebo arms. Among HIV- uninfected tuberculous meningitis cases in New York City who successfully completed treatment, there was no fresh mortality compared to age- and coitus- matched controls over a 10- time follow-up period, although differences in neurologic morbidities, which didn't impact survival, weren't measured. A cohort study of 806 US tuberculous meningitis cases using executive claims data linked rates of neurologic complications at 1-time following opinion stroke (15), seizures (12), and visual impairment (19). Almost of these complications passed during the original hospitalization. A methodical review of 19 pediatrics tuberculous meningitis studies, which included an aggregate of 1636 children treated for tuberculous meningitis, reported a 19-mortality rate, with 54 of survivors passing neurologic complications. Further exploration is demanded to completely understand the burden of tuberculous meningitis in high- prevalence settings, especially the long- term consequences of endless disabilities among nonwage.

8. Types of tuberculosis

Tuberculosis (TB) is a contagious complaint primarily caused by Mycobacterium tuberculosis. TB is classified into different types grounded on colorful factors similar as the point of infection, the form of the complaint, and the resistance to treatment. The primary orders of tuberculosis include pulmonary TB, extra pulmonary TB, medicine-resistant TB, and idle TB. Each of these orders can be further divided into more specific types depending on the clinical and microbiological characteristics.

8.1. Pulmonary Tuberculosis (PTB)

Pulmonary tuberculosis is the most common form of TB, and it primarily affects the lungs. The complaint is transmitted through airborne driblets when a person with active TB coughs or sneezes. Symptoms of pulmonary TB generally include patient cough, haemoptysis (coughing up blood), night sweats, weight loss, fever, and fatigue.

- **Cavitary TB** In some cases of pulmonary TB, the infection leads to the conformation of depressions in the lungs due to towel destruction. This form is more severe and increases the threat of transmission to others.
- **Miliary TB** This form occurs when the bacteria spread to other corridor of the body through the bloodstream. Its ca begets wide infection, affecting organs similar as the liver, spleen, and bone gist.

8.2. Extra pulmonary Tuberculosis (EPTB)

Extra pulmonary TB occurs when the infection affects organs outside the lungs. Extra pulmonary TB is more common in individualities with weakened vulnerable systems, similar as those with HIV/ AIDS.

- **Lymphatic TB** This is one of the most common forms of extra pulmonary TB. It generally involves the lymph bumps, which come enlarged and tender. It's frequently seen in children and in immune compromised cases.
- **Bone and common TB Tuberculosis** can infect bones and joints, causing osteoarticular TB. The chine (Pott's complaint) is the most generally affected area. It leads to reverse pain, disfigurement, and, in severe cases, palsy.
- **Genitourinary TB** This affects the feathers, bladder, and reproductive organs. Symptoms may include haematuria (blood in the urine), dysuria (painful urination), and pelvic pain.
- **TB of the CNS (Central Nervous System) Tuberculosis** can affect the brain and meninges, leading to conditions similar as tuberculous meningitis, which can beget neurological poverties, confusion, and seizures.
- **Peritoneal TB** Involves the peritoneum, the filling of the abdominal depression, causing abdominal pain, ascites, and peritonitis.

8.3. Idle Tuberculosis

Idle tuberculosis infection (LTBI) occurs when a person has been infected with M. tuberculosis but doesn't show symptoms and isn't contagious. In idle TB, the bacteria remain dormant within the body and can come active if the vulnerable system weakens. People with idle TB are generally detected through a positive tuberculin skin test (TST) or interferon- gamma release assay (IGRA). Without treatment, idle TB can progress to active TB complaint.

8.4. Medicine- Resistant Tuberculosis (DR-TB)

Medicine- resistant tuberculosis is a form of TB that doesn't respond to the standard treatment authority, which generally includes a combination of antibiotics similar as isoniazid and rifampin. Medicine resistance arises due to mutations in the bacteria, and it's frequently the result of indecorous treatment rules or non-compliance with remedy.

- **Multidrug- Resistant TB (MDR- TB)** this form of TB is resistant to at least isoniazid and Rifampin, the two most potent first- line anti-TB medicines. MDR- TB requires longer treatment with alternate- line medicines, which may be less effective and more precious.
- **Considerably medicine- Resistant TB (XDR- TB)** This is an indeed more resistant form of TB that's resistant to at least four of the core anti-TB medicines, including any fluoroquinolone and one of the alternates- line injectable medicines. XDR- TB is delicate to treat and has a lower cure rate than MDR- TB.
- **Completely medicine- Resistant TB (TDR- TB)** this is an extremely rare form of TB that shows resistance to all known anti-TB medicines, including both first- line and alternate- line specifics. Treatment options for TDR- TB are limited and frequently ineffective.

8.5. Primary Tuberculosis

Primary tuberculosis occurs when a person is infected with M. tuberculosis for the first time. In almost cases, the body's vulnerable system is suitable to control the infection and help it from progressing to active complaint. Still, in some individuality, the infection may develop into primary progressive tuberculosis, where the bacteria multiply and beget active complaint in the lungs or other organs.

8.6. Secondary Tuberculosis

Secondary tuberculosis, also known as reactivation TB, occurs when latent TB becomes active, frequently due to a weakened vulnerable system. This can be times after the original infection, frequently touched off by conditions similar as HIV/ AIDS, malnutrition, or immunosuppressive treatments. Secondary TB is most frequently pulmonary, but it can also involve extra pulmonary spots.

8.7. Circulated Tuberculosis

Circulated tuberculosis refers to the wide spread of M. tuberculosis throughout the body, frequently involving multiple organs. This type of TB is generally seen in immune compromised individualities, particularly those with advanced HIV/ AIDS. Miliary TB is a form of circulated TB.

8.8. Tuberculosis in Children

Tuberculosis in children frequently presents else than in grown-ups. Pediatrics TB is more likely to be extra pulmonary and is frequently diagnosed latterly due to the absence of classic symptoms like coughs and foam product. Lymph knot TB, abdominal TB, and TB meningitis are common forms in children. Also, children fewer than five times old are at lesser threat of developing severe forms of TB, similar as TB meningitis and circulated TB.

9. Drug classification :[14-21]

Tuberculosis (TB) treatment relies on a combination of drugs to ensure effectiveness and prevent the development of resistance. TB drugs are classified into different categories based on their mechanisms of action, and these include first-line, second-line, and other drugs used in special cases like drug-resistant TB. Understanding the mechanisms of these drugs helps to guide their appropriate use and enhance their efficacy in treating TB.

9.1. First-Line Drugs

First-line drugs are the core drugs used in the treatment of tuberculosis. These drugs are highly effective against *Mycobacterium tuberculosis* and are typically used in combination for the initial treatment phase. They are considered the most potent drugs for treating TB and have the least risk of causing drug resistance when used properly.

- Isoniazid (INH)
    - Mechanism of Action: Isoniazid is a bactericidal drug that inhibits the synthesis of mycolic acids, which are essential components of the bacterial cell wall in *M. tuberculosis*. This disruption leads to the weakening and eventual death of the bacteria. Isoniazid is especially effective during the rapid division phase of the bacteria.
    - Resistance: Resistance can develop due to mutations in the *in-hA* gene, which affects the target enzyme, or mutations in the *katG* gene, which codes for catalase-peroxidase, an enzyme involved in the activation of isoniazid.
- Rifampin (RIF)
    - Mechanism of Action: Rifampin is a bactericidal drug that works by inhibiting bacterial RNA polymerase, thus preventing the transcription of bacterial DNA into RNA. This inhibition prevents the bacteria from synthesizing essential proteins, leading to bacterial death. Rifampin is highly effective in both active and latent TB.
    - Resistance: Resistance to Rifampin generally occurs through mutations in the *probe* gene, which encodes the RNA polymerase beta-subunit, the target of Rifampin.
- Pyrazinamide (PZA)
    - Mechanism of Action: Pyrazinamide is bactericidal in acidic environments, such as those found within macrophages or in the intracellular environment. It is thought to inhibit the synthesis of mycolic acids, as well as disrupt the function of bacterial membrane transport proteins. The exact mechanism remains not fully understood.
    - Resistance: Resistance is typically caused by mutations in the *pncA* gene, which encodes the enzyme Pyrazinamide, responsible for converting Pyrazinamide into its active form.
- Ethambutol (EMB)
    - Mechanism of Action: Ethambutol is bacteriostatic and inhibits the synthesis of the mycobacterium cell wall by blocking the enzyme arabinosyl transferase. This inhibition prevents the formation of arabinogalactan, a critical component of the mycobacterium cell wall, leading to bacterial cell death.
    - Resistance: Resistance occurs due to mutations in the *embB* gene, which encodes the arabinosyl transferase enzyme.
- Streptomycin (SM)
    - Mechanism of Action: Streptomycin is an amino glycoside antibiotic that inhibits protein synthesis by binding to the 30S ribosomal subunit of *M. tuberculosis*, leading to misreading of the mRNA and the production of dysfunctional proteins, ultimately killing the bacteria.
    - Resistance: Resistance to streptomycin is primarily due to mutations in the *rpsL* and *rrs* genes, which encode components of the ribosomal subunit.

9.2. Second-Line Drugs

Second-line drugs are used when the first-line drugs are ineffective, either due to resistance or intolerance. These drugs are generally more toxic and less effective than first-line drugs, and their use should be carefully monitored.

- **Fluoroquinolone** (e.g., Levofloxacin, Moxifloxacin, Gatifloxacin)
    - **Mechanism of Action**: Fluoroquinolone are bactericidal and inhibit DNA gyrase and topoisomerase IV, enzymes responsible for unwinding DNA during replication. By inhibiting these enzymes, fluoroquinolone prevent DNA replication and repair, leading to bacterial death.
    - **Resistance**: Resistance to fluoroquinolone occurs through mutations in the *gyrA* and *parC* genes, which affect the target enzymes.
- **Kanamycin and Amikacin**
    - **Mechanism of Action**: Both kanamycin and amikacin are amino glycosides, similar to streptomycin. They inhibit protein synthesis by binding to the 30S ribosomal subunit, leading to misreading of mRNA and the production of defective proteins.
    - **Resistance**: Resistance to these drugs is typically due to enzymatic modification of the drug, mediated by amino glycoside-modifying enzymes.
- **Capreomycin**
    - **Mechanism of Action**: Capreomycin is another amino glycoside that inhibits protein synthesis by binding to the 70S ribosomal subunit. It interferes with the formation of the ribosomal complex, thereby inhibiting protein translation.
    - **Resistance**: Resistance to capreomycin occurs through mutations in the *rpsL* gene or through the acquisition of resistance genes that modify the drug.
- **Clofazimine**
    - **Mechanism of Action**: Clofazimine is a riminophenazine derivative that binds to DNA and interferes with bacterial DNA replication and protein synthesis. It also exhibits anti-inflammatory properties, which may help in the treatment of drug-resistant TB.
    - **Resistance**: Resistance to clofazimine is rare but can occur due to mutations in the *mycobacterium* DNA repair mechanisms.
- **Ethionamide**
    - **Mechanism of Action**: Ethionamide is similar to isoniazid in its mechanism of action, as it inhibits the synthesis of mycolic acids in the mycobacterium cell wall.
    - **Resistance**: Resistance to ethionamide is generally caused by mutations in the *inhA* gene or other genes related to cell wall synthesis.
- **Cycloserine**
    - **Mechanism of Action**: Cycloserine inhibits the synthesis of the bacterial cell wall by interfering with the formation of peptidoglycan cross-links. It is bacteriostatic and is used as a second-line agent in the treatment of multidrug-resistant TB.
    - **Resistance**: Resistance to cycloserine arises through mutations in the *alr* and *d*-cycloserine genes involved in cell wall synthesis.

9.3. Other Medicines in Special Cases

These medicines are generally used in the treatment of resistant TB or in cases with specific requirements, similar as those with expensive medicine resistance or co-infection with HIV.

- **Bedaquiline** Medium of Action Bedaquiline is a new medicine that inhibits the ATP synthase enzyme in M. tuberculosis, dismembering the bacteria's energy product. This leads to the eventual death of the bacteria.
- **Resistance:** Resistance to bedaquiline is primarily caused by mutations in the atpE gene, which encodes the target enzyme.
- **Delamanid** Medium of Action Delamanid is a nitroimidazole that inhibits the conflation of mycolic acids in the bacterial cell wall by inhibiting the enzyme InhA. It also affects the bacterial respiratory chain, dismembering energy product.
- **Resistance:** It is caused by mutations in the in A gene and other targets involved in cell wall biosynthesis.

9.4. Mechanisms of Drug Resistance in TB

Drug resistance in TB arises through mutations in specific genes within M. tuberculosis. These mutations can confer resistance to one or further medicines, making treatment more delicate. Some of the most common mechanisms of resistance include.

Target Enzyme Mutations numerous TB medicines work by inhibiting specific enzymes(e.g., RNA polymerase for rifampin, mycolic acid conflation for isoniazid). Mutations in the genes garbling these enzymes can reduce the medicine's effectiveness.

Efflux Pumps M. tuberculosis can have efflux pumps that laboriously transport medicines out of the bacterial cell, reducing the medicine's intracellular attention.

Some resistance mechanisms involve the revision of the medicine itself through enzymatic action similar as the acetylating of isoniazid or the revision of amino glycosides like kanamycin.

10. Operation of Tuberculosis (TB)

The operation of tuberculosis (TB) involves a combination of pharmacological treatment, applicable individual procedures, and addressing public health strategies to control its spread. The foundation of TB treatment is antimicrobial remedy, which requires the use of multiple medicines to help the development of medicine-resistant strains. The World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC) give detailed guidelines for the treatment of both medicine-susceptible and medicine- resistant TB.

10.1. First- Line medicine Treatment for medicine-Susceptible

The treatment of medicine-susceptible TB generally involves a six- month authority of four first-line medicines. These medicines

- **Isoniazid (INH)** An antibiotic that inhibits the conflation of mycolic acids in the bacterial cell wall.
- **Rifampin (RIF)** A broad- diapason antibiotic that interferes with bacterial RNA conflation by binding to bacterial RNA polymerase.
- **Pyrazinamide (PZA)** A pro drug that's converted into its active form inside the acidic terrain of the phagosome, targeting the bacteria during the intracellular phase.
- **Ethambutol (EMB)** A medicine that inhibits the conflation of the bacterial cell wall by snooping with arabinogalactan conflation.

This authority is recommended for cases with recently diagnosed, medicine-susceptible TB. The first two months of treatment, known as the ferocious phase, correspond of all four medicines, while the remaining four months, known as the durability phase, involve a combination of isoniazid and rifampin.

10.2. Medicine- Resistant TB

The treatment of multidrug- resistant TB (MDR- TB), which is resistant to at least isoniazid and rifampin, and considerably medicine- resistant TB (XDR- TB), which is also resistant to fluoroquinolone and alternate- line injectable medicines, is more complicated. MDR- TB and XDR- TB bear the use of alternate- line medicines, which may include

- **Fluoroquinolone** (e.g., levofloxacin, moxifloxacin)
- **Injectable agents** (e.g., amikacin, kanamycin, capreomycin)
- **Bedaquiline** A newer medicine that inhibits the ATP synthase of the mycobacterium, making it effective against resistant strains.
- **Delamanid** Another newer medicine that inhibits mycobacterium cell wall conflation.

Treatment rules for MDR- TB or XDR- TB are more prolonged, frequently lasting 18- 24 months, and have an advanced threat of adverse goods. Also, the success rate for these treatments is lower than that of medicine-susceptible TB.

10.3. Adherence to Treatment

Adherence to the TB treatment authority is pivotal for precluding the development of medicine resistance and icing effective treatment. Directly observed remedy (DOT) is a strategy used to cover adherence, where healthcare providers observe cases taking their specifics to ensure the full course of treatment is completed. DOT has been shown to ameliorate adherence and cure rates in TB treatment.

10.4. Operation of idle TB Infection (LTBI)

Idle TB infection occurs when the vulnerable system contains the Mycobacterium tuberculosis bacteria but doesn't allow them to multiply, therefore precluding the development of active TB complaint. LTBI can be treated with preventative remedy to reduce the threat of progression to active complaint. The most generally used treatments for LTBI include

- Isoniazid monotherapy for 6- 9 months
- Rifampin monotherapy for 4 months
- Isoniazid and Rifapentine formerly daily for 3 months (the 3HP authority)

These curatives are recommended for individualities at high threat of developing TB, similar as those with HIV, close connections of TB cases, and people with compromised vulnerable systems.

10.5. Probative Care and Management of Side goods

TB treatment can affect in adverse goods due to the dragged use of multiple medicines. Common side goods include hepatotoxicity, gastrointestinal disturbances, and supplemental neuropathy, especially from isoniazid. probative care, including characteristic treatment and monitoring for medicine- related venom, is an essential part of TB operation. In some cases, cases may need to discontinue or switch specifics if adverse responses do.

10.6. Surgical Intervention

Surgical intervention in TB is reserved for cases where medical treatment is inadequate or complications arise. Surgery may be necessary in the case of expansive lung damage, pleural TB, or medicine- resistant TB where drug alone cannot achieve a cure. Surgical options may include resection of diseased lung towel or pleurectomy for cases with complicated TB.

10.7. Public Health and Infection Control

Public health measures are also critical to TB operation, particularly in high- frequency settings. This includes insulation of cases with active TB, especially those with medicine- resistant TB, until they're no longer contagious. Contact dogging and webbing of individualities who may have been exposed to a person with TB is essential for precluding farther transmission (WHO, 2020). In healthcare settings, infection control measures similar as proper ventilation and particular defensive outfit (PPE) for healthcare workers are necessary to help the spread of TB.

10.8. New Developments and Research

Recent exploration has concentrated on perfecting the opinion, treatment, and forestallment of TB. New individual tools, including rapid-fire molecular tests similar as Gene pert, allow for faster discovery of TB and medicine resistance (Boehme et al., 2010). New medicine campaigners, similar as bedaquiline and delamanid, offer stopgap for cases with medicine-resistant TB. Also, there's ongoing exploration into the development of a TB vaccine to give a more effective preventative measure.

11. Sign and symptoms of tuberculosis (TB) [22-30]

Tuberculosis (TB) is a serious contagious complaint primarily caused by Mycobacterium tuberculosis. TB affects the lungs in the maturity of cases, but it can also affect other corridor of the body, leading to extra pulmonary TB. The signs and symptoms of TB vary depending on whether the complaint is pulmonary or extra pulmonary, and whether it's active or idle. This section will bandy the common signs and symptoms of TB, pressing the differences between active and idle forms, as well as pulmonary and extra pulmonary instantiations.

11.1. Signs and Symptoms of Pulmonary Tuberculosis

Pulmonary tuberculosis (PTB) is the most common form of TB and primarily affects the lungs. The symptoms of PTB frequently develop gradationally, and the complaint may be asymptomatic in its early stages. still, as the infection progresses, the following signs and symptoms are generally observed.

- **Patient Cough;** A dry or productive cough lasting for further than three weeks is one of the hallmark signs of pulmonary TB. The cough may start off mild but can worsen as the complaint progresses.
- **Haemoptysis (Coughing Up Blood);** In more advanced stages, individualities with pulmonary TB may cough up small quantities of blood or foam mixed with blood. Haemoptysis is a more serious symptom and generally indicates a more severe form of TB infection.
- **Fever Low- grade fever;** Is generally present in TB cases. The fever is generally intermittent and may be accompanied by chills. It tends to worsen in the evening or night.
- **Night Sweats;** Inordinate sweating at night, frequently soaking through bed clothes, is a classic symptom of TB. This occurs due to the body's response to the infection.
- **Fatigue and Weakness;** As the body fights the infection, individualities frequently witness extreme frazzle and lack of energy. This can lead to reduced physical exertion and an overall feeling of malaise.
- **Weight Loss Significant and unexplained;** Weight loss is generally seen in people with active TB. This weight loss is associated with the body's increased metabolic demands due to the infection.
- **Casket Pain;** Some individualities may witness casket pain, which can range from mild discomfort to severe pain. The pain is frequently a result of inflammation in the lungs or pleura (the filling around the lungs).
- **Briefness of Breath Difficulty breathing;** Especially when TB has caused significant lung damage or if the infection has spread to other corridor of the body.
- **Appetite Loss;** Individualities with TB frequently witness a loss of appetite, which contributes to weight loss and general weakness.

11.2. Signs and Symptoms of Extra pulmonary Tuberculosis

Extra pulmonary TB occurs when the infection spreads beyond the lungs to other corridor of the body. The symptoms of extra pulmonary TB depend on the organs involved. Common spots for extra pulmonary TB include the lymph bumps, bones, joints, gastrointestinal system, central nervous system, and genitourinary system. The symptoms associated with extra pulmonary TB vary grounded on the position of the infection;

- **Lymphatic Tuberculosis** This form of TB involves the lymph bumps, most frequently in the neck. The lymph bumps may come enlarged, tender, and establishment. The condition is frequently appertained to as "scrofula."
- **Bone and common Tuberculosis** It can affect the bones and joints, causing pain, swelling, and limited range of stir. The chine is the most generally affected area, leading to a condition known as" Pott's complaint." In severe cases, disfigurement, palsy, or abscess conformation may do.
- **Genitourinary Tuberculosis** When TB affects the feathers, bladder, and reproductive organs, symptoms similar as blood in the urine(haematuria), painful urination(dysuria), pelvic pain, and frequent urination can do.
- **Abdominal Tuberculosis** Abdominal TB can beget symptoms similar as abdominal pain, distension, nausea, puking, and weight loss. It can lead to ascites (fluid build-up in the tummy) and peritonitis (inflammation of the abdominal filling).
- **Meningeal Tuberculosis (Tuberculous Meningitis)** When TB affects the central nervous system; it can beget meningitis, which leads to symptoms like headaches, stiff neck, confusion, puking, perceptivity to light (photophobia), and seizures. In severe cases, it may lead to coma or death.
- **Pericardial Tuberculosis** This occurs when the infection spreads to the pericardium, the sac girding the heart. Symptoms include casket pain, difficulty breathing, and fatigue. In severe cases, pericardial effusion (fluid build-up around the heart) can do, leading to heart failure.
- **Pulmonary Tuberculosis** Complicated by Pleural effusion, a condition in which fluid accumulates in the pleura, can be a complication of TB. It leads to symptoms similar as briefness of breath, casket pain, and difficulty breathing.

11.3. Signs and Symptoms of Latent Tuberculosis

Latent tuberculosis (LTBI) occurs when a person has been infected with *Mycobacterium tuberculosis*, but the bacteria remain dormant in the body and do not cause active disease. Individuals with latent TB do not exhibit symptoms and cannot transmit the bacteria to others. However, the bacteria can become active at any time, particularly if the immune system becomes weakened.

- **No Symptoms**: People with latent TB are asymptomatic and may not be aware they are infected. This form of TB is typically detected through skin tests or blood tests such as the tuberculin skin test (TST) or interferon-gamma release assays (IGRA).
- **Risk of Progression**: If left untreated, latent TB may progress to active TB, particularly in individuals with weakened immune systems (e.g., those with HIV, diabetes, or those on immunosuppressive medications).

11.4. Signs and Symptoms of Drug-Resistant Tuberculosis

Drug-resistant TB (DR-TB) refers to TB infections that are resistant to one or more of the first-line drugs used to treat the disease. Symptoms of drug-resistant TB are similar to those of drug-susceptible TB but may persist or worsen despite treatment with first-line medications. The main forms of drug-resistant TB include multidrug-resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB).

- **Delayed Response to Treatment**: One of the most obvious signs of drug-resistant TB is the lack of improvement or worsening of symptoms despite appropriate treatment with first-line drugs.
- **Prolonged Duration of Symptoms**: The cough, fever, and weight loss may persist for an extended period in cases of drug-resistant TB, often requiring longer and more complex treatment regimens.

11.5. General Symptoms of TB:

Some general symptoms that may be present in any form of tuberculosis, whether pulmonary or extra pulmonary, include:

- **Chills**: Some individuals with TB experience chills that are often accompanied by fever and night sweats.
- **Fever**: A common sign in both pulmonary and extra pulmonary TB, fever is typically low-grade and intermittent.
- **Malaise**: General discomfort or unease is common in individuals with TB, as the body responds to the infection.

11.6. Increased Risk of Complications:

Drug-resistant TB is associated with a higher risk of complications such as lung damage, respiratory failure, and death due to the reduced efficacy of standard treatments.

So that we use traditional medicinal plants to cure Tuberculosis (TB) and reduce the mortality due to Tuberculosis (TB)

Table 1 Adverse effect of Anti-TB Drugs [31]

S. NO	DRUGS	ADVERSE EFFECTS
01.	Isoniazid	Skin rash, hepatitis
02.	Rifampicin	Abdominal pain, hepatitis, thrombocytopenic purpura
03.	Pyrazinamide	Arthralgia, hepatitis
04.	Streptomycin	Vestibular and auditory nerve damage, renal damage
05.	Ethambutol	Retro bulbar neuritis, ocular side effects
06.	Thioacetazone	Skin rash, exfoliative dermatitis
07.	PASA	Anorexia, nausea, vomiting, hypersensitivity reactions
08.	Kanamycin	Vertigo, auditory nerve damage, Nephrotoxicity

09.	Ethionamide	Diarrhoea, abdominal pain, hepatotoxicity
10.	Cycloserine	Dizziness, headache, depression, psychosis, convulsions
11.	Capreomycin	Ototoxicity, nephrotoxicity, electrolyte disturbances
12.	Bedaquiline	Nausea, arthralgia, hepatotoxicity, QT prolongation
13.	Delamanid	Nausea, vomiting, QT prolongation, hypokalemia
14.	Clofazimine	Skin discoloration, abdominal pain, diarrhoea, phototoxicity
15.	Amikacin	Nephrotoxicity, ototoxicity, vestibular dysfunction
16.	Levofloxacin	Nausea, dizziness, tendonitis, QT prolongation
17.	Moxifloxacin	Nausea, headache, QT prolongation, phototoxicity
18.	Linezolid	Bone marrow suppression, peripheral neuropathy, optic neuropathy
19.	Pretomanid	Nausea, vomiting, hepatotoxicity, peripheral neuropathy
20.	Fluoroquinolone	CNS affects, tendon rupture, tendinitis

Table 2 List of Anti-Tubercular plants from Ayurveda [31]

S. No	Botanical/ Family name	Ayurvedic name	Part used	Chemical constituents	Other biological activities
01.	Acalypha indica, Euphorbiaceae	Kuppi	Leaves	Kaempferol, acalyphamide and other amides, quinone, sterols, cyanogenic glycoside	Antibacterial, used in bronchitis, asthma
02.	Adhatoda vasica. Acanthaceae	Vaasaa	Leaves	Quinazoline alkaloid	Expectorant (used in bronchial asthma)
03.	Allium cepa, Liliaceae	Palaandu	Bulbs	Volatile oil with sulphurous constituents, including allylpropyldisulphide, sulphurcontaining compounds, including allicin, alliin, flavonoids; phenolic acids and sterols	Antibiotic, antibacterial, antisclerotic, anticoagulant
04.	Allium sativum, Liliaceae	Lashuna	Bulbs	Sulphurcontaining amino acids known as alliin	Antibiotic, bacteriostatic, fungicide, anthelmintic, antithrombic, hypotensive, hypoglycaemic, hypocholesterolaem ic
05.	Aloe vera, Liliaceae	Ghrit kumaarika	Leaves, gel from leaves	Anthraquinone glycosides, known as aloin	Purgative
06.	Vitex negundo, Verbenaceae	Nirgundi	Leaves, seeds	Iridoid glycosides, isomeric flavanones and flavonoids	Anti-inflammatory, analgesic

07.	Trichosanthes dioica, Cucurbitaceae	Patola	Roots, fruits	Free amino acids, nicotinic acid, riboflavin, vitamin C, thiamine, 5-hydroxytryptamine	Cathartic, febrifuge
08.	Tinospora cordifolia, Menispermaceae	Guduuchi	Stem, leaves	Alkaloidal constituents, including berberine; bitter principles, including columbin, chasmanthin, palmarin and tinosporon, tinosporic acid and tinosporo	Antipyretic, antiperiodic, anti-inflammatory
09.	Caesalpinia pulcherrima, Caesapiniacae	Padangam	Leaves, flowers	Flavonoid, myricitroside	Laxative, antipyretic
10.	Prunus armeniaca, Rosaceae	Peetaalu	Kernels	Salicylic acid,organic acids tannins and potassium salts. Protocatechuic,p coumaric, ferulic and diferculic acids	Antitussive, antiasthmatic
11.	Ocimum sanctum, Labiatae	Tulasi	Leaves, flowers, Seeds	Ursolic acid, apigenin, orientin luteolin, apigenin-7 Oglucuronide, luteolin-7-O glucuronide	Carminative, stomachic, antispasmodic, antiasthmatic, antirheumatic, expectorant, hepatoprotective, antiperiodic,
12.	Morinda citrifolia, Rubiaceae	Ashyuka	Leaves, roots, fruits	Anthraquinones alizarin and its glycosides,nor damnacanthol. Ursolic acid and β sitosterol. asperuloside and caproic acid	Antileucorrhoeic,antid ysenteric emmenagogue
13.	Myrtus communis, Myrtaceae	Muurad-daan	Fruits	Tannins (pyrogallol derivative), flavonoids (including myricetin, kaempferol, quercetin glycosides; volatile oil containing α-pinene, cineole, myrtenol, nerol, geraniol and dipentene	Antimicrobial, antiparasitic antiseptic
14.	Canscora decussate, Gentianaceae	Daakuni	Roots	β-amyrin, friedelin,genianine mangiferin, Xanthones	Anticonvulsant, CNS depressant, anti-inflammatory, hepatoprotective.
15.	Piper species, Piperac	Pippali	Fruits	Aristolactams, dioxoaporphines long chain isobutyl amide, lignans, longamide, pluviatilol, methyl	Digestive, appetizer and carminative

				pluviatilol (fargesin), sesamin, asarinine, piperine	
16.	Vitex trifolia, Verbenaceae	Sinduvaara	Leaves, roots, fruits	Flavonoids-artemetin, luteolin, orientin, casticin; and iridoid glycosides, aucubin and agnuside. alkaloid, vitricin	Febrifuge, antibacterial, anthelmintic, cytotoxic
17.	Mallotus philippensis, Euphorbiacae	Kampillaka	Gland and hair of fruit	Phloroglucinol derivatives; rottlerin, isorottlerin, iso allorottlerin	Purgative, anthelmintic, styptic
18.	Colebrookea oppositifolia, Lamiaceae	binda	Leaves, fruits, roots	Flavonoids	Antiinflammatory
19.	Rumex hastatus, Polygonaceae	Katambal	Root and bark	Tannins	Astringent
20.	Mimosa pudica, Mimosaceae	Laajavanti	Leaves, roots	Mimosine and turgorin	Astringent, alterative
21.	Kalanchoe integra, Crassulaceae	Parnabija	Leaves	Triterpenoids-friedelin, taraxerol and glutinol and a mixture of long chain hydrocarbons	Hypotensive, antiarrhythmic.
22.	Flacourtia ramontchii, Flacourtiacae	Vikankata	Leaves, roots, bark, fruits Quinazoline alkaloid	Phenolic glucoside ester, (−)-flacourtin, ramontoside, β-sitosterol and its β D-glucopyranoside	Anticholerin
23.	Annona squamosa, annonaceae	Sitaphal/ sharifa	Leaf, seed and bark extract	Flavonoids, squamocin, annonin, annonaceous aceteogenins	Anti-diabetic, neuroprotective, anti-cancer
24.	Cedrus deodara, pinaceae	devadaru	Wood, leaves	Atlantone, cedrol, deodarone	Insecticidal, anti-cancer

Table 3 List of Anti-Tubercular plants of foreign origin [31]

S. No	Botanical name	Family	Extract	Chemical constituents
1.	*Clavijaprocera*	Theophrastaceae	Ethanolic	Oleananetriterpenoid(aegicerin)
2.	*Rhodomyrtus tomentosa*	Myrtaceae	Alcoholic	Rhodomyrtone
3.	*Aristolochia taliscana*	Aristolochiaceae	Hexane	Neolignans
4.	*Astraeuspteridis*	Astraeaceae	Ethanolic	Lanostanetriterpenesandphenylalanine
5.	*Byrsonimacrassa*	Malpighiaceae	Chloroform	Triterpenes: α-amyrin,β-amyrinand theiracetates,lupeol,oleanolicacid, ursolic acid and α-amyrinone
6.	*Galeniaafricana*	Asteraceae	Ethanolic	Flavonoids

7.	*Gentianopsis paludosa*	Gentianaceae	Ethanolic	1,7,8-Trihydroxy-3-methoxyxanthone, luteolin-7-O-glucoside
8.	*Cryptocarya latifolia*	Lauraceae	Acetone,water	Coumarins
9.	*Eucleanatalensis*	Ebenaceae	Acetone,water	Naphthoquinones
10.	*Helichrysum melanacme*	Asteraceae	Acetone,water	Essentialoils
11.	*Nidorella anomala*	Asteraceae	Acetone,water	Naphthoquinones
12.	*Thymusvulgaris*	Lamiaceae	Acetone,water	Flavonoids,essentialoils
13.	*Buddlejasaligna*	Scrophulariaceae	Alcoholic	Non-cytotoxictriterpenoidsoleanolic
14.	*Leysera gnaphalodes*	Asteraceae	Alcoholic	Non-cytotoxictriterpenoidsoleanolic
15.	*Laggera pterodonta*	Asteraceae	Methanolic	Flavonoids
16.	*Laggeraaurita*	Asteraceae	Methanolic	Flavonoids
17.	*Salviahypargeia*	Lamiaceae	Alcoholic	Diterpene
18.	*Salviasclarea*	Lamiaceae	Alcoholic	Diterpene
19.	*Angiopteris evecta*	Marattiaceae	-	Lactones,coumarins
20.	*Costusspeciosus*	Costaceae	-	Flavonoids
21.	*Plucheaindica*	Asteraceae	-	Phenolics
22.	*Tabernaemontana coronaria*	Apocynaceae	-	Alkaloids
23.	*Pelargonium reniforme*	Geraniaceae	Ethanolic, acetone	Phenolics
24.	*Pelargonium sidoides*	Geraniaceae	Ethanolic, acetone	Phenolics
25.	*Quinchamalium majus*	Santalaceae	Methanolic	Triterpenes
26.	*Senecio chionophilus*	Asteraceae	Hexane, dichloromethane	Sesquiterpenoids
27.	*Evodiaelleryana*	Rutaceae	Hexane, ethyl acetate, methanol	Alkaloid, quinoline
28.	*Piper methysticum*	piperaceae	Kava/ kava lactones	Alkaloids, flavokavains, kavalacetone(methysticin)

Table 4 Medicinal plants and natural products showing in vitro anti-TB activity [32]

S.No	Plant species	Plant family	Plant used	Extract	Compound class	Active constituents
01.	Abrusprecatorius	Fabaceae	Aerial parts	Dichloro Methane fraction	Isoflavanquinoe	Abruquinone,Methyl gallate
02.	Acaciafarnesiana	Mimosaceae	Fruit	Methanolic extract	Parabens, flavanones	(2*S*)-Naringenin 7-*O*-*β*-galloylglucopyranoside (3) showed activity against multidrug resistant *M. t*uberculosi*s* G122 with MIC of 50 μg/ml by MABA
03.	Aglaiaforbesii	Meliaceae	Leaf	Dichloromethanefraction	Benzopyranflavaglines	Desacetylpyramidaglain D against *M.* tuberculosis Ra with MIC of 25 μg/ml by MABA
04.	Allanblackia floribunda	Guttiferae	Root bark	Successivelymaceratedin dichloromethane methanol (1:1) and methanol for4h	Biflavonoids	Morelloflavonewith the MIC of 19.53 and 39.06 μg/ ml against *M.* smegmatis and *M.* tuberculosis, respectively, by MABA
05.	Allium neapolitanum	Alliaceae	Bulb	Chloroformextract	Canthinone	Canthin-6-one, 8-hydroxy-canthin-6-one
06.	Alliumsativum	Liliaceae	Bulb	Petroleumetherextract	Fattyacids	Lauric acid and myristic acid
07.	Allophylusedulis	Sapindaceae	Leaf	Hydrodistillation	Cycloprop[e]azulen-4-ol	Viridiflorol
08.	Alnusincana	Betulaceae	Bark	Methanolextract	Triterpenes	Betulin, betulinic acid, betulone
09.	Alpinia katsumadi	Zingiberaceae	Seed	n-Hexane	Diarylheptanoids	Trans, trans-1,7-diphenylhepta-4,6-dien-3-one
10.	Amphipterygium astringens	Anacardiaceae	Stem, bark	Dichloromethane/ methanol (1:1)	Tirucallanes	(14*β*, 24E)-3-oxolanosta-7,24-dien-26-oic acid (16) and (14*β*,24E)-3-hydroxylanosta-7,24-dien-26-oic acid

Table 5 Important anti-TB traditional medicinal plants in literature by the systemic survey on the prescribed formula (32)

S. No	Species number	Family number	Main families	Country/region
01.	90	44	Fabaceae (13), Asteraceae (7), Moraceae (5), Rutaceae (4)	Districts of Mpigi and Butambala, Uganda
02.	35	22	Fabaceae (5), Rutaceae (4), Apocynaceae (3), Menispermaceae (3),	Madhya Pradesh, India
03.	132	45	Annonaceae (14), Zingiberaceae (12), Rutaceae (10), Annonaceae(10), Asteraceae(8), Euphorbiaceae(8), Fabaceae(7)	Southeast Asian
04.	10	8	Fabaceae (3), Cannellaceae(1), Rubiaceae(1), Anacardiaceae(1), Rutaceae(1), Mirtaceae(1), Merlucciidae(1), Guttiferae(1)	Lake Victoria Basin (Uganda, Kenya and Tanzania)
05.	14	8	Euphorbiaceae (4), Verbenaceae (3), Rutaceae (2)	Lake Victoria region and the Samburu community
06.	2	2	Achillea millefolium (1), Dryopteris Stewartii (1)	Kell village, Neelum valley, Azad Kashmir, Pakistan
07.	4	3	Amaryllidaceae (1), Lauraceae (1), Amaranthaceae (1), Asteraceae (01)	Sulaymaniyah province, Kurdistan, Iraq,
08.	22	18	Liliaceae (3), Euphorbiaceae(2), Verbenaceae(2),	India
09.	6	6	Vitaceae (1), Poaceae(1), Pinaceae(1), Musaceae(1), Rosaceae(1), Leguminosae(1)	Arabian Peninsula
10.	2	2	Asteraceae (1) Dryopteridaceae(1)	Pakistan
11.	70	44	Arecaceae (4), Euphorbiaceae (4), Fabaceae (3), Piperaceae (3),	Malaysia

12. Conclusion

Tuberculosis (TB) remains a critical global health issue, worsened by drug resistance and complex disease presentations. Although synthetic drugs are effective, their side effects have driven interest in safer, plant-based alternatives. Medicinal plants offer a cost-effective and widely accessible source of potential treatments, yet many remain underexplored for clinical application. The rich diversity of bioactive compounds in these plants presents an opportunity for innovative therapies. Integrating traditional knowledge with modern medicine could provide safer and more effective solutions. Advancing research in this field is vital for overcoming TB's challenges and improving global health outcomes.

Compliance with ethical standards

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The author(s) declare that they have no competing interests.

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