



Anti-tubercular drugs: Mechanism of action, resistance and recent advances

Khushboo Vaghela^{1*}, Kavya Patel², Mahi Patel², Smruti Bhatt², Devarpi Patel², Divyakant Patel³

¹ Professor, Department of Pharmacognosy, Sharda School of Pharmacy, affiliated to Gujarat Technological University, Pethapur, Gandhinagar, Gujarat, India

² Department of Pharmacognosy, Sharda School of Pharmacy, affiliated to Gujarat Technological University, Pethapur, Gandhinagar, Gujarat, India

³ Head of the Institute, Sharda School of Pharmacy, affiliated to Gujarat Technological University, Pethapur, Gandhinagar, Gujarat, India

DOI: <https://doi.org/10.66856/ijpsr.2026.11.3.11041>

Abstract

Tuberculosis (TB) remains one of the leading infectious diseases worldwide and is primarily caused by *Mycobacterium tuberculosis*, predominantly affecting the lungs. Although standard chemotherapy with first-line anti-tubercular agents such as isoniazid, pyrazinamide, and ethambutol has significantly improved treatment outcomes, prolonged therapy is often associated with serious adverse drug reactions, poor patient compliance, and the increasing emergence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains. These challenges highlight the urgent need for alternative or adjunct therapeutic strategies. In recent years, medicinal plants have gained considerable attention due to their diverse bioactive phytoconstituents and potential antimycobacterial properties. Several herbal extracts and isolated compounds have demonstrated promising inhibitory activity against *M. tuberculosis* in preclinical studies. Additionally, certain plant-derived compounds exhibit immunomodulatory and anti-inflammatory effects, which may enhance host defense mechanisms and improve treatment outcomes. This review aims to summarize the pathophysiology of TB, limitations of current synthetic therapies, and recent advances in herbal anti-tubercular agents, emphasizing their mechanisms of action, therapeutic potential, and future prospects in TB management. The use of allopathic medicine in complex disease like tuberculosis is associated with the problem of cross resistance and herbal drugs have proven to be most effective in this context.

Keywords: *Mycobacterium tuberculosis*, herbal medicine, phytoconstituents, antimycobacterial activity, alternative therapy

Introduction

Tuberculosis (TB) is a Chronic Granulomatous disease caused by *Mycobacterium tuberculosis* an acid-fast bacillus (AFB). Mycobacterial infections require prolonged treatment. Antitubercular medications are a group of drugs used to treat tuberculosis^[1]. History suggests that TB appeared long ago and remained unpredictable until the eighteenth century^[2]. It then, at that point, reached pandemic proportions among the increasing population and overwhelming daily situations. In the 20th century, the TB-afflicted population started to reduce rapidly in developed countries with progress in prosperity, food, and dwelling conditions. TB cases have decreased since the Bacillus Calmette–Guérin (BCG) vaccine was made available in 1921 and antimicrobial drugs such as streptomycin, isoniazid, and rifampicin were made available by prescription in 1943, 1952, and 1963. MTB infects approximately 8,000,000 people annually, and about 2–3 million die from the disease. It is estimated that 33% of the absolute people are infected with latent TB, with 40% coming from India^[3]. With a yearly rate of 2,000,000 new cases, more than 40% of India's population is infected with latent TB. For about 50 years, counter-TB drugs have been depleted. The emergence of multi-drug-resistant (MDR) and extensively drug-resistant tuberculosis (XDR-TB) strains is a significant challenge in the fight against TB. With the emergence of COVID-19, the number of TB cases has been increasing abruptly, as it has directly impacted TB treatment and diagnosis. With the current situation in mind, the World Health Organization (WHO) has moved the goal of eradicating TB from the world from 2025 to 2035^[4].

Biochemistry of Tuberculosis

It needs oxygen to grow, which is a requirement for *M. tuberculosis*. Because of the high lipid content of its wall, it does not require any bacteriological stain and is never classified as either Gram-positive or Gram-negative. Ziehl-Neelsen staining, also known as acid-fast staining, is therefore utilized. The lack of an outer cell membrane causes the mycobacteria to be classed as acid-fast Gram-positive bacteria even though they do not appear to fall under the empirically defined category of Gram-positive bacteria (i.e., they do not maintain the crystal violet stain). In comparison to other bacteria, whose division periods are often measured in minutes, *M. tuberculosis* replicates every 15-20 hours, which is incredibly slowly. Early endosomal auto antigen 1 (EEA1), a bridge molecule, is specifically blocked by *M. tuberculosis*, however this blockage does not stop the fusion of vesicles filled with nutrients. Consequently, the bacteria multiply unchecked within the macrophage. The bacteria also carried the UreC gene, which prevents acidification of the phagosome. The bacteria also evade macrophage-killing by causing the neutralization of reactive nitrogen intermediates^[5].

Pathophysiology of TB

TB is a severe infectious disease that primarily impacts on the pulmonary system. Moreover, it can impact other body parts, including the kidneys, spine, bones, lymph nodes and brain and is attributed to *Mycobacterium tuberculosis* (Mtb) (Figure 1), a highly specialized human pathogen^[6].

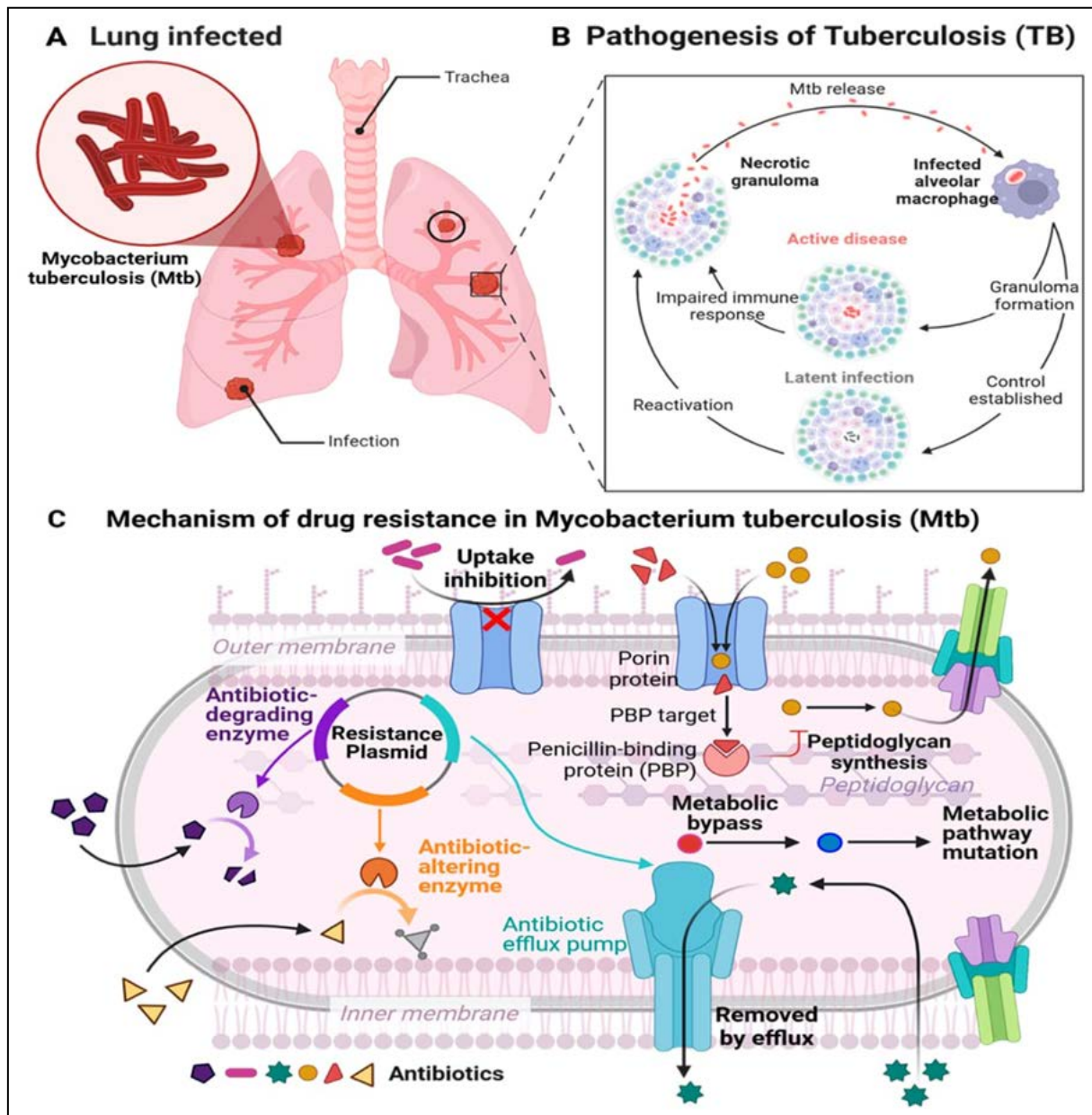


Fig 1: Pathophysiology and transmission mechanism of *Mycobacterium tuberculosis* infection.

Mtb transmission occurs via airborne particles expelled by individuals who cough, sneeze or spit, thereby distributing the infectious organism. The bacteria can persist in the air for several hours, rapidly infecting others [7]. Following inhalation, *Mtb* is primarily engulfed by resident alveolar macrophages in the lungs [7]. Macrophages internalize pathogens via phagocytosis, which is facilitated by ligand-receptor interactions involving mannose receptors, scavenger receptors, complement receptors (CR1, CR3, CR4), Fc receptors, and surfactant protein receptors [8]. *Mtb* skillfully circumvents the bactericidal strategies of macrophages through mechanisms that include the prevention of bacterial vacuole acidification, inhibition of phagolysosome formation and the obstruction of apoptosis and autophagy in infected macrophages [8, 9]. After internalization, *Mtb* inhibits phagosome maturation, allowing it to merge with lysosome and persist within macrophages. Alternatively, it replicates intracellularly by preventing phagosome-lysosome fusion and transforming macrophages into a protective environment for the

pathogen [10]. After infection of alveolar macrophages within the lower respiratory tract, *Mtb* infiltrates the interstitium of the lung(s), thereby advancing the course of the infection process. The incursion of pathogens into the parenchyma triggers an immune response, resulting in the mobilization of T and B cells to the locus of infection. This recruitment precipitates a complex multicellular response by the host called granulomatous inflammation. Granulomas serve the purpose of efficiently confining the *Mtb* pathogen at the site(s) of infection, thus inhibiting spreading in the host. Nonetheless, the progression of dysfunctional granulomas may lead to the persistence of pathogens, considerable tissue damage and insufficient responses to treatment. *Mtb* utilizes granulomas (Figure 1) as focal points of infection, where phagocytic cells congregate, and providing ample nutrients for replication [8]. Infected individuals who do not present with symptoms of the disease cannot transmit *Mtb*; however, the bacteria persist within the body in an inactive or latent form, which if left untreated, can advance to become active TB (Figure 1) [7].

Classification	Herb	Botanical Name	Therapeutic Contribution in Tuberculosis
 Direct Anti-mycobacterial Herbs	Neem	<i>Azadirachta indica</i>	Suppresses the growth of <i>Mycobacterium tuberculosis</i> .
	Garlic	<i>Allium sativum</i>	Exhibits antibacterial properties and enhances immune defense.
	Turmeric	<i>Curcuma longa</i>	Offers anti-inflammatory and antimicrobial benefits.
	Tulsi	<i>Ocimum sanctum</i>	Shows antimicrobial activity and supports respiratory function.
 Immunomodulatory Herbs	Ashwagandha	<i>Withania somnifera</i>	Enhances immune function and improves physical resilience.
	Guduchi	<i>Tinospora cordifolia</i>	Modulates immune response and promotes host resistance.
 Expectorant / Respiratory Herbs	Vasaka	<i>Adhatoda vasica</i>	Helps relieve cough and eases bronchial congestion.
	Mulethi	<i>Glycyrrhiza glabra</i>	Soothes irritation and reduces airway inflammation.
 General Tonic / Supportive Herbs	Amla	<i>Phyllanthus emblica</i>	Rich in antioxidants and improves nutritional status.
	Shatavari	<i>Asparagus racemosus</i>	Supports body strength and aids in recovery.

Fig 2: Classification of herbal anti-tubercular drugs based on their mechanism of action.

1. Garlic: As Shown in Figure 2, garlic (*Allium sativum* Linn.) is classified under direct anti-mycobacterial herbs.

Synonym: *Allium sativum* Linn.

Family: Amaryllidaceae

Biological Source: Garlic consists of the fresh or dried bulbs of *Allium sativum* Linn.

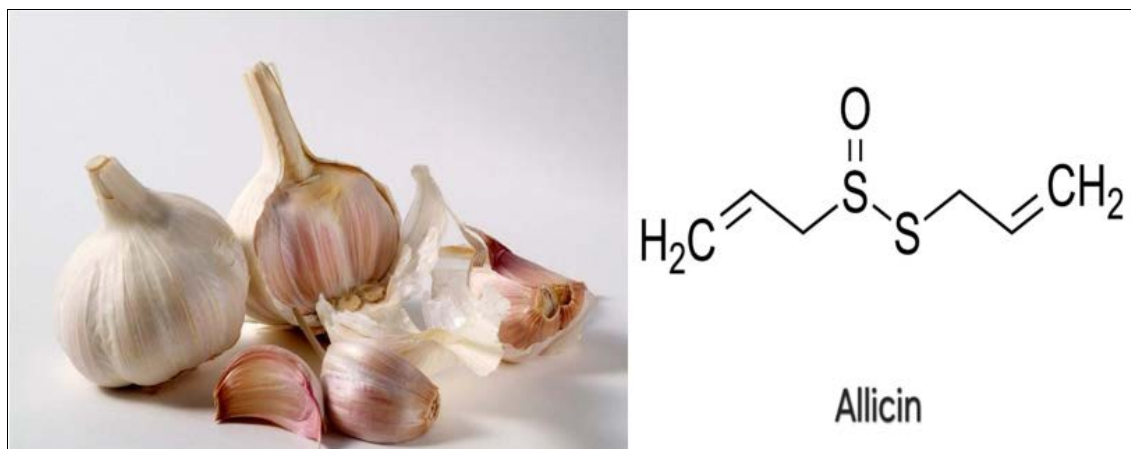


Fig 3: Chemical structure and key bioactive constituents of *Allium sativum* (Garlic).

Chemical Constituents

Allium sativum bulb contains sulfur compounds such as ajoene and derivatives, allicin, allyl-methyl trisulfide and derivatives, diallyl disulfide and derivatives, dimethyl sulfide, allyl-methyl disulfide and derivatives, dimethyl thiosulfonate and derivatives, amino acids derived from cysteine and anthocyanins, sulphurated amino acids: alliin (by enzymatic oxidation it becomes allicin, intermediate in the formation of allyl disulfide derivatives, final constituents of essential oil), 5-butyl-cysteine sulfoxide and derivatives, lipids such as cerebrosides, steroids, prostaglandin A, B, E, and F; alkaloids such as phosphatidylcholine, nicotinic acid, diterpenes (gibberellin A-3 and A-7); car-bohydrates such as allium fructans; derivatives of erubioside, sativoside, and tigonine [12].

Mechanism of Action:

As Mentioned in figure [4], Allicin exerts its antimycobacterial activity primarily through inhibition of sulfhydryl (–SH) containing enzymes essential for bacterial metabolism. It interacts with thiol groups of key metabolic enzymes, leading to enzyme inactivation and disruption of cellular function. Additionally, allicin induces oxidative stress within *Mycobacterium tuberculosis* by modulating reactive oxygen species (ROS) pathways. It enhances host immune defense by stimulating macrophage activation and promoting Th1-mediated immune responses. Allicin has also been reported to increase glutathione peroxidase activity and interfere with intracellular survival and entry of *M. tuberculosis*, thereby reducing bacterial burden [13].

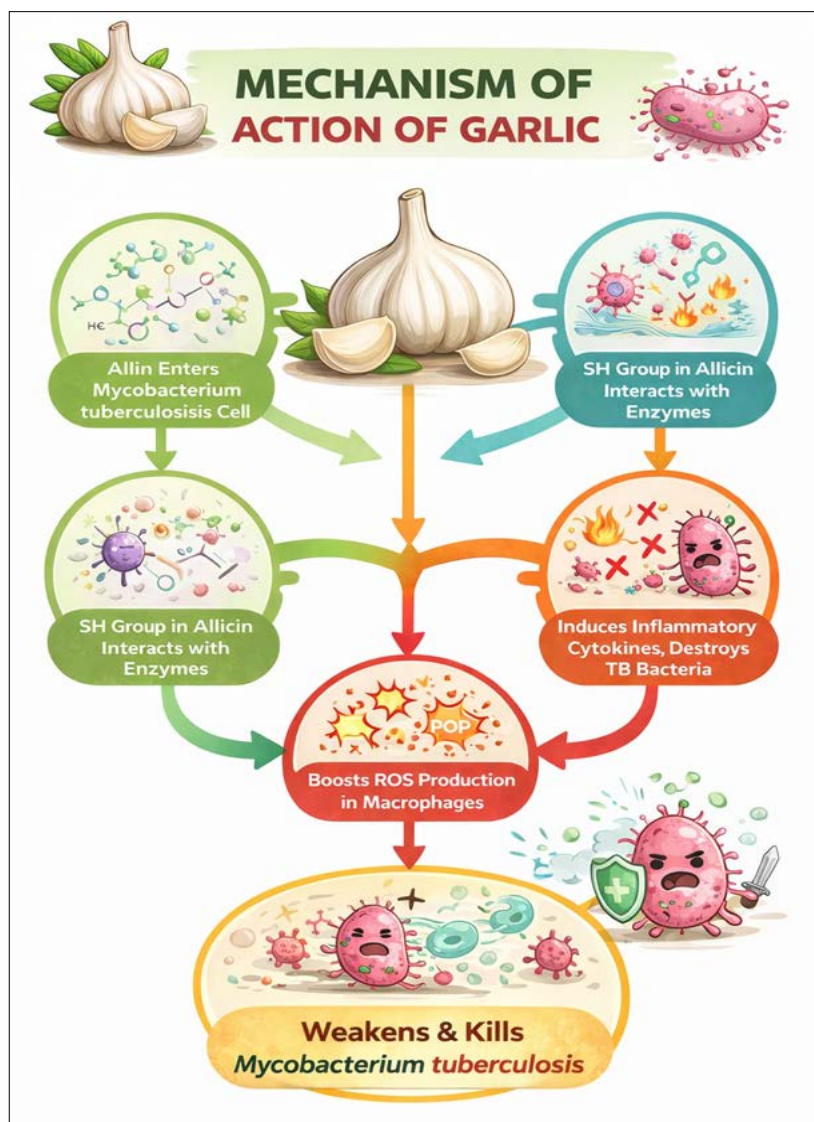


Fig 4: Mechanism of action of allicin against *Mycobacterium tuberculosis*.

Uses

Garlic has been used as herbal medicine to treat asthma, bronchitis, influenza, and colds. Besides, it serves as a treatment for respiratory infections^[14]. It is commonly used to treat bronchopulmonary diseases as expectorant antiseptic and asthma. Likewise, it is used as a hypotensive, diaphoretic, and vermifuge^[15].

2. URMERIC: As shown in Figure 2, turmeric (*Curcuma longa* Linn.) is classified under direct anti-mycobacterial herbs.

Synonym: *Curcuma domestica* Valetton

Family: Zingiberaceae (ginger)

Biological Source: Turmeric consists of the dried or fresh rhizomes (underground stems) of *Curcuma longa* Linn.

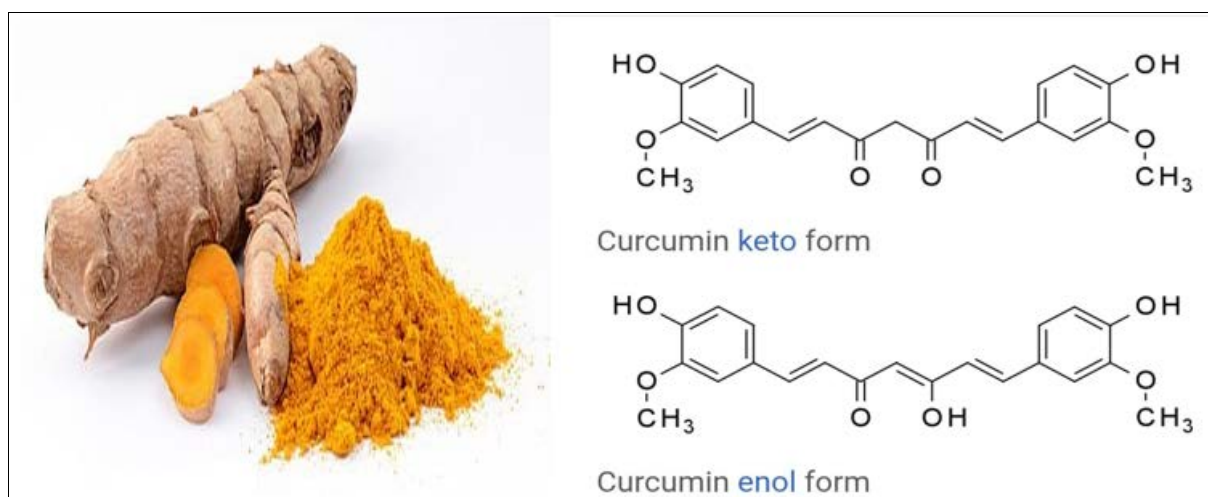


Fig 5: Chemical structure and active constituents of *Curcuma longa* (Turmeric).

Chemical Constituents

The medicinal properties of turmeric, the supply of curcumin, were regarded from many years; but, the capability to decide the exact mechanism(s) of action and to decide the bioactive compounds have only these days been investigated as follows: Curcumin is also referred as diferuloylmethane, is the principle herbal polyphenol observed inside the rhizome of *Curcuma longa* (turmeric) and in others *Curcuma* spp^[16]. *Curcuma longa* has been historically used in Asian countries as a medical herb because of its antioxidant, antimutagenic, antimicrobial^[17] and anticancer properties^[18]. The energetic parts of turmeric are the flavonoid Curcuminoids that aggregates curcumin (diferuloylmethane), monodemethoxycurcumin and bisdemethoxycurcumin in Curcumin makes up approximately 90% of the curcuminoid content material in turmeric. Other elements encompass sugars, proteins, and resins. The best-researched energetic constituent is curcumin, which comprises 35.4% of uncooked turmeric^[19].

Mechanism of Action

As Mentioned in Figure^[6], Inhibition of Pro-inflammatory Signaling, Curcumin downregulates the NF-KB signaling

pathway, leading to decreased expression of inflammatory mediators such as: Cyclooxygenase-2 (COX-2), Inducible nitric oxide synthase (iNOS). By suppressing NF-κB activation, curcumin reduces chronic inflammation, which plays a crucial role in TB pathogenesis.

Activation of Antioxidant Pathways

Curcumin upregulates the Nrf2 pathway, enhancing cellular antioxidant defenses. This reduces oxidative stress-induced tissue damage during TB infection.

Induction of Apoptosis and Autophagy

Curcumin promotes apoptosis in infected macrophages, facilitating intracellular killing of *M. tuberculosis*. Additionally, it enhances autophagy-related gene expression, improving bacterial clearance mechanisms.

Direct Antimicrobial Activity

Studies demonstrate dose-dependent inhibition of intracellular growth of *M. tuberculosis* (H37Rv strain), suggesting both host-directed and direct antibacterial effects (20 to 22).

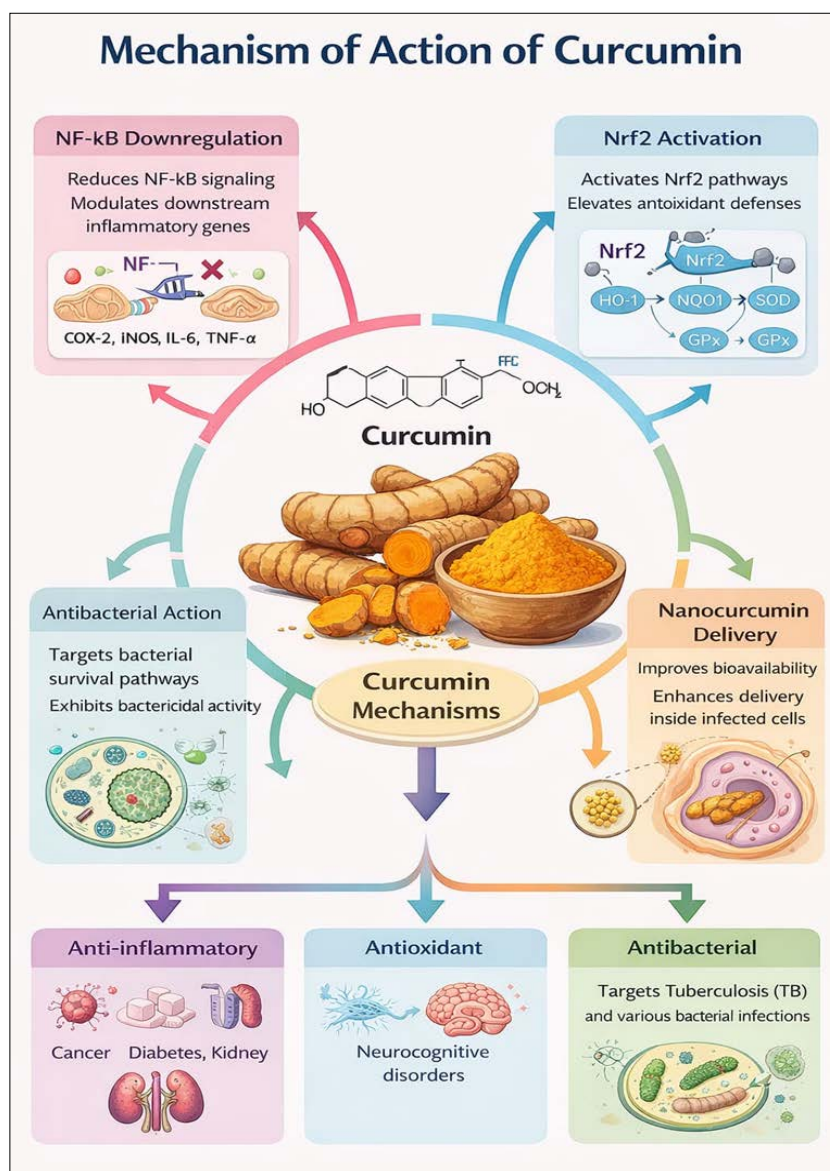


Fig 6: Mechanism of action of curcumin in tuberculosis management.

Uses

Turmeric is widely used for medicinal purpose such as helps to relieve gas, improves digestion, dispelling worms, regulates menstruation, relieves arthritis, dissolving gallstones and overall energy of the body. Turmeric and its paste are also used in many ayurvedic applications like blood purification, remedial for skin conditions in Indian country. Turmeric paste is beneficial for hair to remove superfluous hair.

In some parts of the -India, Pakistan, Bangladesh turmeric is applied on the bride and groom skin before marriage to

make skin glow, shine, and helps to keep harmful bacteria away from the body^[23].

3. Guduchi: As shown in Figure 2, Guduchi (*Tinospora cordifolia* Linn.) is classified under immunomodulatory herbs.

Synonym: *Tinospora cordifolia* (Willd.) Miers.

Family: Menispermaceae

Biological source: Guduchi (giloy) is derived from the dried or fresh stem of *Tinospora cordifolia* (willed). Miers, a climbing shrub belonging to the family Menispermaceae.

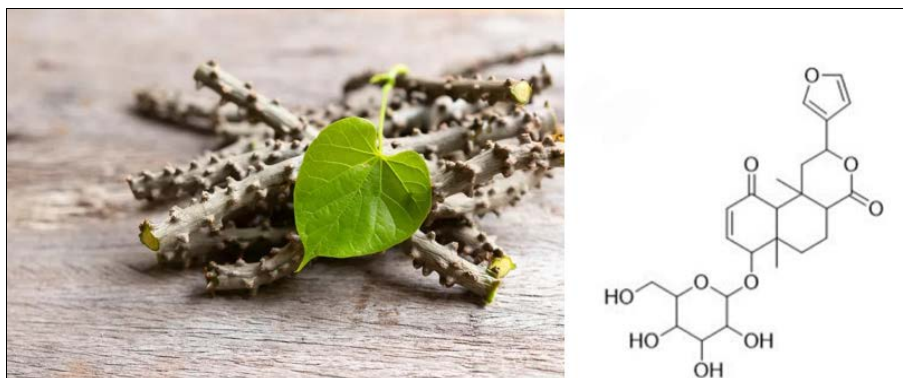


Fig 7: Phytochemical constituents of *Tinospora cordifolia* (Guduchi).

Chemical Constituents

The different classes of compounds which are found in this plant are classed in groups like alkaloids, steroids, Terpenoids, polysaccharides, glycosides and different aromatic and aliphatic compounds that are present in their phytoactive form that are responsible for the wide range of medicinal and therapeutic properties. The presence of these compound is found in various plant parts but highly concentrated in the stem, leaves and root part of the plant^[24].

Mechanism of Action

As Mentioned in figure^[8],

1. Active Phytochemicals Glycosides: Cordifolioside A, B, Cordioside, Cordiol

Others: Syringin, Magnoflorine, Tinocordiside

Novel compounds: N-formylannonain, 11-hydroxymuskatone^[25]

Effect:

↑ IgG antibody production, ↑ Neutrophil & macrophage phagocytosis, ↑ Reactive oxygen species (ROS)

2. Polysaccharides G1-4A (Arabinogalactan) Targets:

Macrophages (major), B-cells (minor) Acts as TLR4 agonist Pathway: TLR4 → MyD88 → NF-κB / MAPKs (ERK, JNK, p38)^[26].

Effects: ↑ Th1 cytokines (TNF-α, IL-1β), ↓ Th2 response, ↑ M1 macrophage activation (↑ MHC-II, CD86), ↓ TB bacterial load

3. β-Glucans Bind receptors: CD11b, TLR2, TLR6^[27]

Effect: Promote Th1 immune response.

4. Guduchi immunomodulatory Protein:

Effects:

↑ Splenocyte & thymocyte proliferation, ↑ Macrophage phagocytic & bactericidal activity

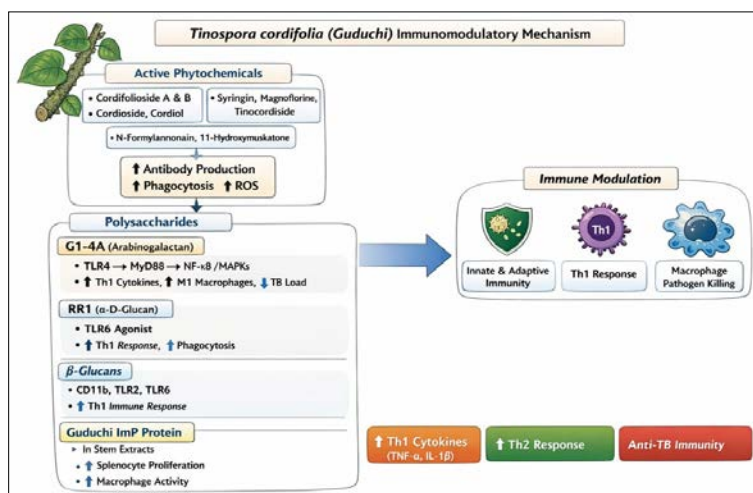


Fig 8: Immunomodulatory mechanism of *Tinospora cordifolia* in tuberculosis.

Uses

Tinospora cordifolia (willed). Miers, commonly known as guduchi is one of the highly valued vine useful in a wide range of diseases. Satva is aqueous extractable starchy substance collected from herbal drug. Guduchi Satva is commonly used and prescribed Ayurvedic medicine in Daha (burning sensation), Pitta predominant disorders. Its method

of preparation has been mentioned initially in yoga Ratnakara, followed by Rasa Yoga, Sagara, Sidhha Yoga Sangraha, Bhavprakash, etc; however, no text emphasizes on the impact of the season on quantitative and qualitative aspects of Satva [28].

Glance of Herbal anti-tubercular drugs

Botanical Name	Family	Chemical Constituents	Activity	Ayurvedic Name
<i>Allium cepa</i>	Liliaceae	allylpropyldisulphide, sulphurcontaining compounds, including allicin, alliin, flavonoids; phenolic acids and sterols	Antibiotic, antibacterial, antisclerotic, anticoagulant	Palaandu
<i>Aloe vera</i>	Liliaceae	aloin	Purgative	Ghritkumaarika
<i>Trichosanthes dioica</i>	Cucurbitaceae	nicotinic acid, riboflavin, vitamin C, thiamine, 5-hydroxytryptamine	Cathartic, febrifuge	Patola
<i>Prunus armeniaca</i>	Rosaceae	Protocatechuic, pcoumaric, Ferulic acid	antiasthmatic	Peetaalu
<i>Ocimum sanctum</i>	Labiatae	apigenin, orientin luteolin, apigenin-7-Oglucuronide	Carminative, stomachic, antispasmodic, antiasthmatic, antirheumatic, expectorant, hepatoprotective, antiperiodic	Tulsi

Fig 9: Overview of herbal anti-tubercular drugs and their therapeutic roles.

Current Drug Therapy

To boost the body's ability to adapt to care and decrease the period of treatment, current pharmaceutical therapy utilizes a cocktail of drugs. Two of the most often given first-line treatments are rifampicin and isoniazid. Rifampicin is a more significant part of the regimen since it reduces recovery time and assures the best results. A nine-month therapy includes rifampicin and isoniazid, as well as streptomycin and ethambutol [33]. According to one study done by the Medical Research Council of the United Kingdom, utilizing pyrazinamide in the first two months of the therapy regimen reduces the treatment period to six months while maintaining a cure rate of 95% or better [34]. Stopping the virus from spreading is the most effective way to eliminate tuberculosis, which may be achieved by prioritizing treatment for sputum smear-positive patients or anyone who can spread the illness. DOTS (Directly Observed Treatment) may be used to assess and manage these individuals. The short course is a very successful and cost-efficient strategy to TB care that is widely recommended across the globe [35]. DOTS consists of five elements: ongoing political and financial commitment, quality-assured diagnostic protocols, consistent delivery of superior anti-TB medications, supervised documentation and reporting, and systematic short-course (SCC) anti-TB care provided under clear and supportive observation (DOT) [36].

Problems with the Current Drug Therapy

Patient compliance is hampered due to the extended period of TB treatment, and the large number of drugs utilized will make it difficult for patients to take their regular dosages. The present treatment is still prone to side effects and has been demonstrated to interact negatively with other drugs. In addition, the lengthy operation involves a large financial commitment in the pharmaceuticals required for treatment. The new multidrug therapy has little to no effect on latent tuberculosis. It has also led in the development of MDR-TB [37]. When the improper or inefficient medications are utilised, drug tolerance develops. This might be due to drug delivery confusion, stopping the process in the middle, or leaving just one of the medicines delivered in the protocol (standard treatment is at least two drugs) [38]. MDR-TB may be treated with second-line drugs such as fluoroquinolones, aminoglycosides, and others, but they are ineffective, dangerous, and costly. Second-line drugs are more expensive than first-line drugs, but the main issue is their treatment duration, which is nearly double that of standard TB treatment, making it much more difficult for some patients to afford treatment, continue treatment for the full duration, and increase the risk of disease spread, leading to further resistance development [39].

Conclusion

With increasing rates of tuberculosis worldwide and the rise of MDR-TB and XDR-TB, there is a need for novel antimycobacterial agents. Using the MRA as a screening

tool, this study assessed the antimycobacterial properties of traditional medicinal plants^[40]. The side effects associated with the allopathic drugs have remarkably necessities the need of herbal drugs. In this review, authors have tried to make a complete description of all the antitubercular drugs around the globe. The various constituents present in plants makes them as an effective antitubercular drug. The discovery of new drugs has finally begin to emerge, the standard of care for tuberculosis might become possible soon, and the framework for classification of drug-resistant cases will need to be explored. Although the prevalence of TB in the society exists from large decades, still research needs to be explored for the benefit of the society. Herbal medicinal plants used for treatment and alleviate the many diseases^[41]. The World Health Organization (WHO) has estimated that up to 70% of the world's population is purely depends on the traditional system of medicine for their preliminary health care. A number of factors can be attributed by dependence of large portion of the population on traditional medicine like: comparatively good approachability to the plants and wide-ranging of local information and knowledge amongst the societies^[42].

References

- Jassal M, Bishai WR. Extensively drug-resistant tuberculosis. The Lancet infectious diseases,2009;9(1):19-30.
- Barberis I, Bragazzi NL, Galluzzo L, Martini M. The history of tuberculosis: from the first historical records to the isolation of Koch's bacillus. Journal of preventive medicine and hygiene,2017;58(1):E9.
- Montes M, Phillips C. Nonreactive tuberculosis. American Review of Tuberculosis and Pulmonary Diseases,1959;79(3):362-70.
- World Health Organization. Global tuberculosis report 2021: supplementary material. World Health Organization, 2022.
- Burcher S, Ho MW. Prac. Global strategy for traditional medicine research, 1996, 142-4.
- Sparrow A, Smith-Torino M, Shamamba SM, Chirakarhula B, Lwaboshi MA, Benn CS, et al. A risk management approach to global pandemics of infectious disease and anti-microbial resistance. Tropical medicine and infectious disease,2024;9(11):280.
- Sierra CJ. Tuberculosis Infection, Demographic and Clinical Factors, and Post-Transplant Outcomes in Adult Liver Transplant Patient in Florida (Doctoral dissertation, University of Miami).
- Omoteso OA, Fadaka AO, Walker RB, Khamanga SM. Innovative strategies for combating multidrug-resistant tuberculosis: advances in drug delivery systems and treatment. Microorganisms,2025;13(4):722.
- Bekraki AI. Liposomes-and niosomes-based drug delivery systems for tuberculosis treatment. InNanotechnology based approaches for tuberculosis treatment. Academic Press, 2020, 107-122.
- De Chastellier C. The many niches and strategies used by pathogenic mycobacteria for survival within host macrophages. Immunobiology,2009;214(7):526-42.
- Mistry KS, Sanghvi Z, Parmar G, Shah S. The antimicrobial activity of *Azadirachta indica*, *Mimusops elengi*, *Tinospora cordifolia*, *Ocimum sanctum* and 2% chlorhexidine gluconate on common endodontic pathogens: An in vitro study. European journal of dentistry,2014;8(02):172-7.
- Vakhariya RR, Talokar S, Dhole AR, Mohite SK, Magdum CS. Comparative Standardization Study of Two Marketed Shatavari Churna Formulation. Asian Journal of Pharmaceutical Analysis, 2016, 6(1).
- Choi JA, Cho SN, Lim YJ, Lee J, Go D, Kim SH, et al. Enhancement of the antimycobacterial activity of macrophages by ajoene. Innate Immunity,2018;24(1):79-88.
- Jasim FA, Al-Hilu HS. Chemical Datasets, Antioxidant, Free Radicals Scavenger activities estimate in Aqueous Garlic (*Allium sativum*) extract. Res. J. Pharm. Technol.,2021;14(10):5157-62.
- Villasana AP. Global evolution history, biogeographical distribution and emergence of drug resistance by the Latin American and Mediterranean (L4. 3) family of *Mycobacterium tuberculosis* (Doctoral dissertation).
- Bharti AC. Anticancer potential of curcumin: preclinical and clinical studies. Anticancer research, 2003.
- Reddy RC, Vatsala PG, Keshamouni VG, Padmanaban G, Rangarajan PN. Curcumin for malaria therapy. Biochemical and biophysical research communications,2005;326(2):472-4.
- Wright LE, Frye JB, Gorti B, Timmermann BN, Funk JL. Bioactivity of turmeric-derived curcuminoids and related metabolites in breast cancer. Current pharmaceutical design,2013;19(34):6218-25.
- Heath DD, Khwaja F, Rock CL. Curcumin content of turmeric and curry powders. InFaseb Journal. 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA: FEDERATION AMER SOC EXP BIOL.,2004;18(4):A125-A125.
- Rao CV. Regulation of COX and LOX by curcumin. The molecular targets and therapeutic uses of curcumin in health and disease, 2007, 213-26.
- Jobin C, Bradham CA, Russo MP, Juma B, Narula AS, Brenner DA, et al. Curcumin blocks cytokine-mediated NF- κ B activation and proinflammatory gene expression by inhibiting inhibitory factor I- κ B kinase activity. The Journal of Immunology,1999;163(6):3474-83.
- Gupta PK, Kulkarni S, Rajan R. Inhibition of intracellular survival of multi drug resistant clinical isolates of *Mycobacterium tuberculosis* in macrophages by curcumin. Open Antimicrob Agents J.,2013;4(1):1-5.
- Araújo MC, da Luz Dias F, Takahashi CS. Potentiation by turmeric and curcumin of γ -radiation-induced chromosome aberrations in Chinese hamster ovary cells. Teratogenesis, carcinogenesis, and mutagenesis,1999;19(1):9-18.
- Sharma PC, Yelne MB, Dennis TJ, Joshi A. Database on medicinal plants used in Ayurveda. New Delhi: CCRAS, 2005.
- Maurya R, Handa SS. Tinocordifolin, a sesquiterpene from *Tinospora cordifolia*. Phytochemistry,1998;49(5):1343-5.
- Mishra R, Kaur G. Aqueous ethanolic extract of *Tinospora cordifolia* as a potential candidate for differentiation based therapy of glioblastomas. PLoS One,2013;8(10):e78764.

27. Nair PR, Melnick SJ, Ramachandran R, Escalon E, Ramachandran C. Mechanism of macrophage activation by (1, 4)- α -D-glucan isolated from *Tinospora cordifolia*. International Immunopharmacology, 2006;6(12):1815-24.
28. YT A. Charaka Samhita of Agnivesha; Kalpasthana; Madanakalpa. Reprint ed., 2011, 653.
29. Gabbrielli G, Loggini F, Cioni PL, Giannaccini B, Mancuso E. Activity of lavandino essential oil against non-tubercular opportunistic rapid grown mycobacteria. Pharmacological research communications, 1988;20:37-40.
30. Sehlapelo BM, Drewes SE, Scott-Shaw R. A 6-substituted 5, 6-dihydro- α -pyrone from two species of *Cryptocarya*. Phytochemistry, 1994;37(3):847-9.
31. Koyama J. Anti-infective quinone derivatives of recent patents. Recent Patents on Anti-Infective Drug Discovery, 2006;1(1):113-25.
32. Imelouane B, Amhamdi H, Wathelet JP, Ankit M, Khedid K, El Bachiri A. Chemical composition and antimicrobial activity of essential oil of thyme (*Thymus vulgaris*) from Eastern Morocco. Int. J. Agric. Biol., 2009;11(2):205-8.
33. Chhajed SS, Upasani CD, Wadher SJ, Mahapatra DK. Medicinal Chemistry. Nashik: Career Publications Private Limited., 2017, 71-2.
34. Chhajed SS, Bastikar V, Bastikar AV, Mahapatra DK. Computer aided drug design. Pune: Everest Publishing House., 2019, 68.
35. Baheti JR, Mahapatra DK, Borkar SS, Wakodkar III SB. Pharmacology-III. Nagpur: ABD Publications Private Limited., 2020, 137.
36. Puranik MP, Mahapatra III DK. Medicinal chemistry-III. Nagpur: ABD Publications Private Limited., 2020, 141.
37. Mitnick CD, Shin SS, Seung KJ, Rich ML, Atwood SS, Furin JJ, et al. Comprehensive treatment of extensively drug-resistant tuberculosis. New England Journal of Medicine, 2008;359(6):563-74.
38. Caminero JA, Sotgiu G, Zumla A, Migliori GB. Best drug treatment for multidrug-resistant and extensively drug-resistant tuberculosis. The Lancet infectious diseases, 2010;10(9):621-9.
39. LoBue P. Extensively drug-resistant tuberculosis. Current opinion in infectious diseases, 2009;22(2):167-73.
40. Jethva KD, Bhatt DR, Zaveri MN. Antimycobacterial screening of selected medicinal plants using mtt and the microplate resazurin assay. Int. J Pharm Sci & Res., 2021;12(3):1537-45.
41. Vaghela K, Bhatt D, Zaveri M. Effect of ethanomedicinal plants on *Mycobacterium smegmatis* Tubercular bacilli using mycobacteria growth indicator tube assay.
42. Jethva K, Bhatt D, Zaveri M. Epidemiology of non-tuberculous mycobacteria in India: a review. J pharmacogn phytochem., 2019;8(3):954.