



**THERAPEUTIC POTENTIAL OF CAMELLIA SINENSIS: PHYTOCHEMICAL
PROFILE AND DIVERSE BIOLOGICAL ACTIVITIES**

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ABSTRACT

Camellia sinensis is a commonly used medicinal plant that is abundant in bioactive phytochemicals having varied restorative qualities. Recent progress in research on natural products has brought an emphasis on its possible role in antitubercular therapy, both due to its general pharmacological action and to more recent findings of host-directed effects. The diverse phytoconstituents, such as phenols, tannins, terpenoids, alkaloids, and carbohydrates etc. All these phytochemical components are associated with potent antioxidant activity and a wide range of antimicrobial actions against a variety of Gram-positive and Gram-negative pathogens is further evidence of the therapeutic value of *C. sinensis*. This implies that the underlying mechanisms, the processes, which are common with pathways toward mycobacterial susceptibility, are membrane disruption, enzyme inhibition, and protein precipitation. During its discovery, notably, epigallocatechin-3-gallate (EGCG), the main catechin in green tea, has a proven antitubercular mechanism that is host directed, with transcription of TACO gene by Sp1 being suppressed by EGCG in macrophage. TACO down-regulation can be reversed, resulting in a process of phagosome-lysosome fusion, which results in the effective elimination of *Mycobacterium tuberculosis* in the cell. This process provides a complementary tool that has a potential to be used with standard TB treatment especially with the development of drug resistance. Other pharmacological action such as antitumor, antidiabetic and cytoprotective action further enhances the extensive bioactivity of *C. sinensis*. The toxicity tests always indicate that the level of toxicity is very high and there are no serious adverse effects at therapeutic and higher doses.

KEYWORDS: Camellia sinensis; Phytochemistry; Antioxidant activity; Anti-inflammatory; Antimicrobial; Anticancer; Antidiabetic.

1. INTRODUCTION

Camellia sinensis or green tea, is one of the most widely researched medicinal plants because of the abundance of bioactive phytochemicals and the long history of its use in traditional medicine. In addition to its cultural role as a commonly consumed drink, growing scientific interest has been given to the therapeutic value of its extracts of leaves especially the methanolic fraction which has been found to contain high levels of phenolics, catechins, tannins, alkaloids, terpenoids and other secondary metabolites.^[1]

These compounds contribute to a wide range of pharmacological properties, which comprise antioxidant, antimicrobial, anti-inflammatory, anti-tumour, metabolic and immunomodulatory properties. *Mycobacterium tuberculosis* causes tuberculosis (TB), which is a significant burden among the world population, with its well-known statistics being millions of cases every year.^[2] The TB burden has become so complicated with the rise of multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains, which has increased the length of the treatment process, hepatotoxicity of first-line medications, and host immunity.^[3,4]

These challenges contribute to the necessity of innovative therapies, in particular, the agents that are able to boost host immunity or supplement the current drug regimens. The bioactive compounds of plants have become credible options because they act multi-targetedly, are safe, and less likely to induce resistance. A number of *C. sinensis* components have overlapping pharmacological actions with TB pathophysiology.^[5] Phenolic substances and catechins have strong free-radical scavenging properties. TB requires the use of antioxidant defences since the cellular immunity of the infected macrophages is hampered by acute oxidative stress.

Green tea polyphenols induce a less favourable intracellular environment, which allows the macrophage to survive by neutralising reactive oxygen species, thereby supporting macrophage viability. The diverse data indicate that broad-spectrum antibacterial is achieved through a combination of multiple pathogenic bacteria, indicative of mechanisms including membrane disruption, enzyme inhibition and protein precipitation mechanisms, which are pertinent to the lipid-rich mycobacterial cell wall.^[6, 7]

More importantly, the main catechin of *C. sinensis*, epigallocatechin-3-gallate (EGCG), has been demonstrated to have a discriminating host-specific antitubercular action, in which EGCG inhibits the transcription factor Sp1, resulting in the down-regulation of TACO gene in macrophages. Since TACO inhibits the phagosome-lysosome fusion, its blockage recovers macrophage bactericidal activity.^[8] This mechanism makes *C. sinensis* resistant to traditional antibiotics by acting upon host pathways but not bacterial enzymes, limiting the chances of developing drug resistance.^[9]

The favourable blend of phytochemical abundance, pharmacological versatility and relevance in mechanisms, *Camellia sinensis* is an attractive natural source in adjunctive or host-directed research in

tuberculosis. This review examines its phytochemical profile, pharmacological data, and mechanisms of action that support its possible application in anti-tubercular treatment.^[10]

2. Phytochemical Profile of *Camellia sinensis*

The botanical source of green, black, white and oolong teas, *Camellia sinensis*, is prodigally endowed with a wide variety of bioactive phytoconstituents, which are ascribed to the pharmacological activity.^[11] The qualitative and quantitative composition of these metabolites depends on the maturity of the leaves, geographical source, climate, and processing techniques (fermentation/oxidation). The key phytochemical families found in *C. sinensis* are polyphenols, alkaloids, amino acids, volatile compounds, vitamins, minerals and saponins.^[12, 13] All these phytochemicals help the plant to have antioxidant, antimicrobial, immunomodulatory and antitubercular effects.

2.1. Polyphenols: The most important and largest set of phytochemicals in *C. sinensis* is composed of polyphenols. These are the active compounds that are central to the definition of the antioxidant capacity, sensorial and therapeutic properties of green tea. The significant subclasses of polyphenols in green tea are flavan-3-ols (catechins), flavonols, flavanones and small amounts of anthocyanidins and flavones.^[14]

2.1.1. Catechins ((Flavan-3-ols)): These contain very high levels of green tea, and consist of epigallocatechin gallate (EGCG), epigallocatechin (EGC), epicatechin gallate (ECG), and epicatechin (EC) as shown in **Figure 1**. EGCG is the most bioactive and the most abundant of these, playing an important role in the antioxidant, anti-inflammatory, antimicrobial, and anticancer actions of green tea.^[15] These catechins have multi-modes of action, including free-radical scavenging, metal ion chelation, oxidative enzyme inhibition and also in numerous ways on cellular signalling pathways such as NF- κ B, MAPK, and PI3K/Akt.

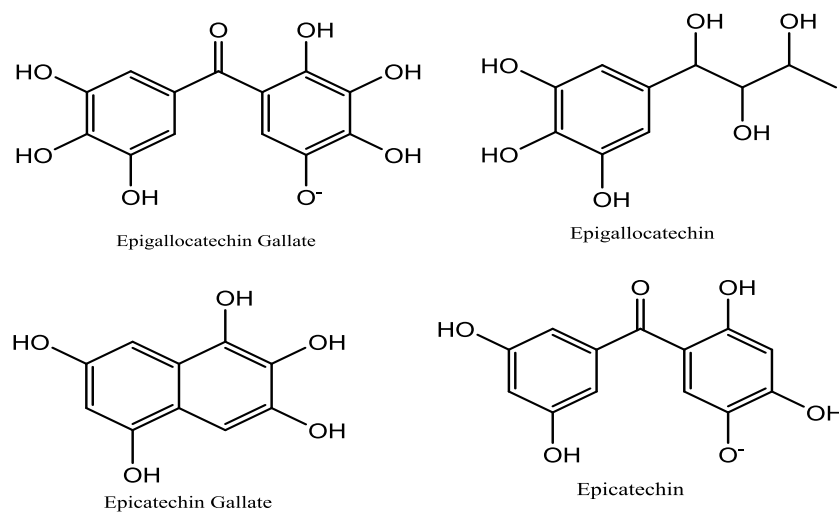


Figure 1: Major Polyphenols Present in *Camellia sinensis*.

This figure shows the main polyphenolic constituents of *Camellia sinensis*, with a focus on catechins, including EGCG, EGC, ECG and EC. These are the most abundant and pharmacologically active group of molecules of green tea.^[16] Their chemical structures, functional hydroxyl groups and aromatic rings that allow antioxidant, radical-scavenging and metal-chelating action are emphasised in the illustration. These functional characteristics are directly linked to biological processes that apply to tuberculosis, such as ROS neutralisation, immune modulation and inhibition of bacterial oxidative systems.^[17]

2.1.2. Flavonols: Green tea contains several flavonols and their glycosides, predominantly: Quercetin, Kaempferol and Myricetin. These compounds exist primarily as glycosidic derivatives in the fresh leaves and contribute to antioxidant and anti-inflammatory activities.^[18] Flavonols also play a structural and protective role in plant tissues, and their levels often increase in response to environmental stress such as UV exposure.

2.2. Alkaloids: *Camellia sinensis* leaves contain methylxanthine alkaloids that modulate CNS and cardiovascular activity.

2.2.1. Caffeine (1,3,7-trimethylxanthine)

The principal stimulant alkaloid varies with processing (high in black tea). The presence of caffeine, theobromine and theophylline contributes to the stimulant, diuretic, and antimicrobial effects of the plant. Caffeine exhibits mild antibacterial and membrane-modulatory activity, which, although weaker than polyphenols, enhances the overall antimicrobial potential of the extract. Xanthine alkaloids are known to inhibit bacterial topoisomerases, modulate intracellular cAMP and enhance macrophage metabolic activity. These mechanisms complement the host-directed action of EGCG in tuberculosis.^[19]

2.2.2. Theobromine and theophylline: Present in smaller quantities; influence smooth muscle relaxation and bronchodilation.

2.3. Tannins: Enhance astringency and also have metal-chelating and antioxidant activities. They inhibit bacterial enzymes, destabilise the membrane and proteins of the microbial cell wall because of their protein-precipitating capacity.^[20] Tannins are synergistic with catechins, and they increase antimicrobial activity against organisms with lipid-rich or protein-rich membranes **Figure 2**.

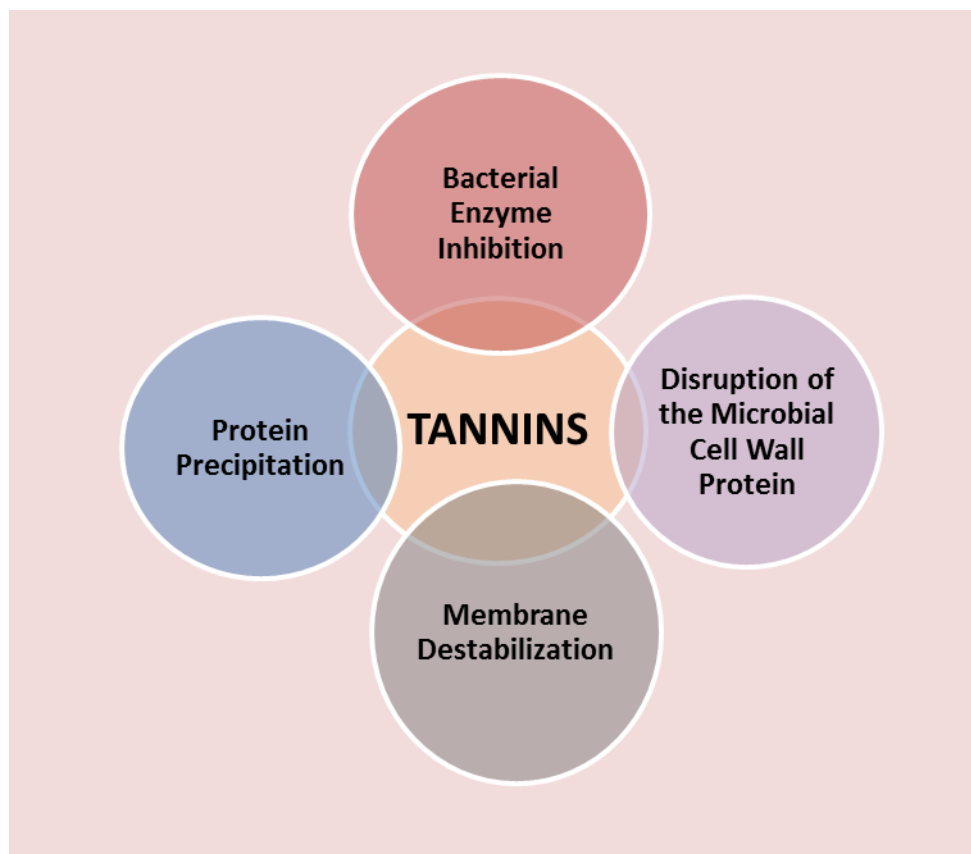


Figure 2: Tannins and Their Mechanisms of Antimicrobial Activity.

This figure represents the major antimicrobial mechanism linked with tannins in *Camellia sinensis*. As depicted in the illustration, tannins attach to proteins of

bacteria by forming hydrogen bonds, resulting in the precipitation and inactivation of essential enzymes in bacteria. Other panels emphasise that tannin can

destabilise the lipid bilayers, membrane proteins, and affect permeability to destabilise microbial cell membranes. Such interplay prevents bacterial adhesion, enzyme action and food assimilation.^[21] In the case of tuberculosis, the fatty cell wall of *Mycobacterium tuberculosis* has a high number of protein-lipid structures, which are prone to tannin-mediated disruption.

2.4. Terpenoids: The terpenoids present in green tea are an eclectic mix of terpenoids, with the most common classes being the monoterpene, sesquiterpene, and diterpene. The significant components are linalool, geraniol, nerol, citral, limonene, α -pinene, β -pinene, terpinen-4-ol, farnesol, and phytol (a degradation product of chlorophyll). These compounds are mainly related to the volatile portion of the tea leaf and differ depending on the cultivar, the age of the leaf and the conditions of the processing.^[22]

Terpenoids are the key functional components in the provision of the typical aroma and flavour profile of green tea. Linalool and geraniol are added to the floral sweet flavours, and limonene and citral are added to the citrus-like flavours. In addition to the sensory properties, a number of terpenoids have significant biological activities. The monoterpenes, limonene and linalool, are potent antioxidants of lipid-phase radical scavenging, and they supplement the aqueous-phase antioxidant effect of catechins. Also, terpenoids have anti-inflammatory effects by suppressing or regulating pro-inflammatory cytokines such as TNF- α , IL-6, and COX-2 or inhibiting the NF- κ B pathway.^[23]

Some terpenoids of green tea are also antimicrobial. The compounds of the linalool, geraniol and terpinen-4-ol interfere with the membrane integrity of microbes and lead to inhibitory action of gram-positive and gram-negative bacteria, as well as fungal pathogens. Moreover, diterpenes, such as phytol, and sesquiterpenes, such as farnesol, have chemopreventive properties (inducing apoptosis and blocking cancer cell proliferation), as well as by regulating essential signalling pathways (MAPK and PI3K/Akt).

2.5. Steroids: In green tea, steroidal constituents are found in the form of either phytosterols or triterpenoid steroids. The key phytosterols known are β -sitosterol, stigmasterol, and campesterol, though triterpenoid steroids, including lupeol, betulinic acid, oleanolic acid, and ursolic acid, are also mentioned.

Hypolipidemic activity of phytosterols has been related to competitive inhibition of intestinal cholesterol absorption in the diet, and hence adds lipid-lowering effect of green tea.^[24] Moreover, the steroidal triterpenoids have a strong anti-inflammatory effect, which is mostly caused by blocking the enzyme of cyclooxygenase, an inhibition of the pro-inflammatory cytokines. These compounds have also been

demonstrated to have a strong antioxidant activity that suppresses oxidative stress by eliminating lipid peroxidation of the cellular components.^[25]

Some of the steroids, such as ursolic acid and lupeol, have shown anticancer potential. Their modes include activation of apoptosis, suppression of tumour cell proliferation, alteration of intricate signalling pathways including NF- κ B, JNK, and PI3K/Akt. In addition, certain phytosterols are antimicrobial against microbes by disrupting the membrane structure of the microbes. The cardioprotective effect of steroidal constituents is also reported to enhance the endothelial activity and inhibit oxidative modification of low-density lipoproteins.^[26]

3. Relevance of Phytochemical Composition to Tubercular Research

Camellia sinensis phytochemical diversity goes beyond a descriptive tool to show a functional applicability to biological pathways that are involved in the pathogenesis of tuberculosis (TB) and control. Green tea contains phenolics and flavonoids that have powerful antioxidant and anti-inflammatory effects that are necessary in reducing oxidative stress and tissue damage that are typical of chronic *Mycobacterium tuberculosis* infection. The mode of action is that catechins, especially the epigallocatechin-3-gallate (EGCG), is a major host-directed modulator in stimulating macrophage activation, cytokine release control, and autophagic pathways in intracellular mycobacteria clearance. Tannins play a role in antimycobacterial activity because they have the capacity to bind and deactivate bacterial proteins, disrupting enzymatic processes required for the survival of mycobacteria.^[27] Another common group of green tea metabolites, terpenoids, have a membrane-disruptive nature, with the potential to disrupt cell envelope integrity of mycobacteria, which is a critical defence mechanism against intrinsic drug resistance. Further, alkaloids found in *C. sinensis* are also known to suppress enzymes that are linked to mycobacterial metabolic control and reproduction, which also indicates their promising therapeutic capabilities. Taken together, these phytochemical groups point to the mechanistic diversity with which green tea constituents can play a role in preventing direct antimicrobial effects as well as host-directed therapy in TB studies.^[28]

3.1. Antitubercular Evidence and Mechanistic Implications of *Camellia Sinensis*:

The antitubercular activity of *Camellia sinensis* has attracted interest over the last few years as a result of the growing burden of drug-resistant tuberculosis (TB) and the necessity of new low-toxicity therapeutic options. Even though green tea is historically taken in forms of antioxidant and immunomodulatory, recent experimental and mechanistic research suggests that phytochemical constituents of green tea can have a direct and indirect effect on *Mycobacterium tuberculosis* (Mtb). These

results highlight its applicability as an additional TB bioactive compound.^[29]

3.1.1. In Vitro Antimycobacterial Activity: It has been shown by several studies that extracts of *C. sinensis* have a measurable effect against *Mtb* and non-tuberculous mycobacteria (NTM). Bacteriostatic properties have been demonstrated by polyphenol-containing fractions (especially those high in catechins), with EGCG and EGC being prominent active components. These will disrupt important bacterial processes such as protein stability, enzymatic activity and membrane integrity.^[30] EGCG, specifically, has been demonstrated to prevent the FabG1 enzyme involved in the production of mycolic acid, a distinctive lipid pathway which is critical in the production of the complex mycobacterial cell wall.^[31] Further in vitro studies indicate that green tea catechins have the capability of paralysing *Mtb* proliferation by blocking dihydrofolate reductase, as well as disrupting redox homeostasis.

3.1.2. Membrane Disruption and Protein Inactivation: The *C. sinensis* tannins and terpenoids play a direct role in antimycobacterial activity, which is aimed at disrupting the structural integrity of the *Mtb* cell envelope. Terpenoids break lipid layers of mycobacterial membranes, making the bacteria permeable and rendering them unviable. These effects are potentiated by tannins, which bind to bacterial proteins and cause inactivation and structural destabilisation. All these actions together destabilise the inherent drug resistance of the highly hydrophobic and impermeable mycobacterial cell envelope.^[32]

3.1.3. Inhibition of Drug Resistance Mechanisms: Green tea phytochemicals also have the potential to regulate mechanisms linked to antibiotic resistance. Some flavonoids and catechins block *Mtb* efflux pumps, which are a significant source of the lowered effect of the first-line TB drugs. These compounds inhibit efflux activity and increase intracellular drug retention, and possibly, drug susceptibility is restored. Also, bacterial responses to stresses that result in drug tolerance can be reduced by the antioxidant effect of polyphenols.^[33]

3.1.4. Host-Directed Therapeutic Advantages: In addition to the direct antimycobacterial effects, *C. sinensis* also exhibits features that are of great use in host-directed therapy (HDT) of TB. It has been demonstrated that EGCG increases macrophage-mediated killing of mycobacteria by promoting autophagic flux, phagolysosomal fusion, and pattern-recognition receptor signalling. Its control on the production of cytokines, such as inhibition of excessive TNF- α and IL-6, aids in the maintenance of a balanced inflammatory state, inhibiting immune-mediated tissue destruction and aiding in the elimination of bacteria.^[34] These two activities suggest that green tea catechins are promising supplements to conventional TB chemotherapy.

3.1.5. Synergy With Conventional Antitubercular Drugs: There is some initial positive indication that *C. sinensis* components have the potential to interact with existing TB medications like isoniazid, rifampicin, and ethambutol. There are improved antimicrobial effects of catechins with first-line drugs, which may be explained by the better permeability of the membrane, changes to bacterial redox systems, or prevention of compensatory stress responses. These interactions can enable reduction of dose, reduction of treatment-related toxicity and multidrug regimens, particularly in cases of resistance.^[35]

3.2. Antidiabetic Evidence and Mechanistic Implications of Camellia Sinensis: Green tea and bioactive compounds in it, especially catechins, flavonoids, alkaloids, amino acids, and polysaccharides, have a multidimensional impact on glucose homeostasis, insulin activity, lipid metabolism, and oxidative stress. All these mechanisms are supportive in its use as a complementary treatment agent in the treatment of diabetes mellitus.^[36]

3.2.1. Modulation of Glucose Uptake and Insulin Sensitivity: The main role of improving the glucose uptake of the cell is localised to catechins, most notably, epigallocatechin-3-gallate (EGCG). Experimental works reveal that EGCG stimulates AMP-activated protein kinase (AMPK) induced translocation of glucose transporter type 4 (GLUT4) in skeletal muscle and adipose tissues. This enhances the use of peripheral glucose and helps in the enhancement of insulin sensitivity.^[37] Also, green tea polyphenols decrease insulin resistance by inhibiting inflammatory mediators and damaging insulin receptor signalling pathways.

3.2.2. Inhibition of Carbohydrate-Digesting Enzymes: Green tea extract has been demonstrated as an important inhibitor of α -amylase and α -glucosidase, which are enzymes that aid the digestion of carbohydrates and uptake of glucose in the intestine.^[26] There is retarded glucose release and reduced postprandial hyperglycemia, which results from the inhibition of these enzymes. The contribution of catechins and condensed tannins is regarded as the main contributor of this mechanism, which is similar to the effect of enzyme inhibitors used in a clinic, including acarbose.^[38]

3.2.3. Antioxidant Protection Against Diabetic Oxidative Stress: One of the leading causes of the development and the development of diabetic complications is oxidative stress. *C. sinensis* has a strong antioxidant capacity due to its catechins, phenolic acids, and flavonoids, which are effective in neutralising reactive oxygen species and the oxidative destruction of β -cells. Green tea maintains B-cell stability and increases the antioxidant enzyme activity endogenously, thereby contributing to the maintenance of insulin secretion and the minimisation of tissue damage associated with diabetes.^[39]

3.2.4. Modulation of Lipid Metabolism and Weight Regulation: Type 2 diabetes is a significant disease that is caused by abnormal lipid levels and obesity. It has been proven that green tea polyphenols could decrease total cholesterol, LDL cholesterol, and triglycerides and increase the HDL level.^[29] These effects are a result of inhibiting lipid absorption and activating β -oxidation, and increasing the energy expenditure. Thermogenesis, weight control, and better metabolic health are a combination of EGCG, caffeine, and theanine, which indirectly leads to better glycemic control.^[40]

3.2.5. Protection of Pancreatic β -Cells: Several *in vitro* and *in vivo* studies show the protective action of *C. sinensis* extracts on pancreatic β -cells. Catechins help to suppress Cdk5-induced glucotoxicity-induced apoptosis by modulating apoptotic signalling pathways, oxidative stress, and mitochondrial stabilisation. This maintains the mass of β -cells in aiding the normal secretion of insulin in diabetic diseases.^[41]

3.2.6. Influence on Gut Microbiota and Metabolic Homeostasis: Recent studies point to the interplay of phytochemicals of green tea and bowel flora. The tea polyphenols are prebiotic modulators, which enrich good bacteria, and optimise the synthesis of short-chain fatty acids (SCFA). The effects of these microbiotas include an increase in insulin sensitivity, glucose metabolism, and systemic inflammation.^[42,43] Emerging research highlights the interaction between green tea phytochemicals and gut microbiota. Tea polyphenols act as prebiotic modulators, enriching beneficial bacteria and enhancing the production of short-chain fatty acids (SCFAs). These microbiota-mediated effects contribute to improved insulin sensitivity, enhanced glucose metabolism, and reduced systemic inflammation.^[44]

3.2.7. Evidence From Clinical and Preclinical Studies
The animal models of type 1 and type 2 diabetes show

the hypoglycemic properties of green tea, which include the decrease in the levels of fasting blood glucose, HbA1c, and insulin resistance measures.^[45] Even though human clinical trials are variable in nature, they yield supporting evidence of the improvement of glycemic markers, lipid profile, and weight reduction with frequent intake of green tea. Variations in tea preparation, dosage, and time span help to create differences in the results but general orientation to the improvement of metabolism is well-established.^[46]

3.4. Antiproliferative and Antitumor Activity: Significance for Chronic Infection

A potato-disc bioassay data revealed complete inhibition of tumour formation induced by *Agrobacterium tumefaciens* at higher extract doses (750 μ L). While not TB-specific, this finding indicates cytotoxicity against hyperproliferative cells, inhibition of bacterial-induced oncogenic transformation and disruption of cell-signalling pathways.^[47] These cytoprotective properties emphasise the biochemical potency of green tea constituents and support their therapeutic potential beyond antimicrobial activity.^[48]

3.5. Direct Antitubercular Evidence- the EGCG-TACO Interaction: The strongest antitubercular evidence arises from the EGCG-macrophage interaction pathway, where EGCG modulates host cellular mechanisms rather than exerting direct bactericidal pressure.^[49] This host-directed activity includes the downregulation of the *TACO* gene through *Sp1*-mediated transcriptional control, thereby preventing phagosome maturation arrest and enabling effective intracellular killing of *Mycobacterium tuberculosis*.^[50] This pathway represents the most robust and well-characterised mechanism underlying green tea's antitubercular potential **Table 1**.

Table 1. Key findings of EGCG-macrophage interaction pathway.

1. EGCG suppresses Sp1-mediated TACO expression The TACO protein (coronin-1A) prevents normal phagosome maturation. <i>Mtb</i> exploits TACO to avoid lysosomal killing.
EGCG inhibits the Sp1 transcription factor, reducing TACO gene transcription. \downarrow TACO \rightarrow \uparrow phagosome maturation \rightarrow \uparrow <i>Mtb</i> clearance.
2. EGCG reduces intracellular <i>Mtb</i> survival Macrophages treated with EGCG before infection show significantly reduced CFU counts.
3. EGCG restores phagosome-lysosome fusion Confocal imaging confirmed increased colocalization of lysosomal markers with ingested bacilli.

Most plant extracts show *in vitro* antimicrobial activity, whereas EGCG shows host-directed therapy, a cutting-edge approach endorsed by the WHO for TB.^[51] It does not target the bacterium directly but corrects the immune defect that *Mtb* exploits, and this mechanism uniquely positions *C. sinensis* in TB drug discovery pipelines **Figure 3**.

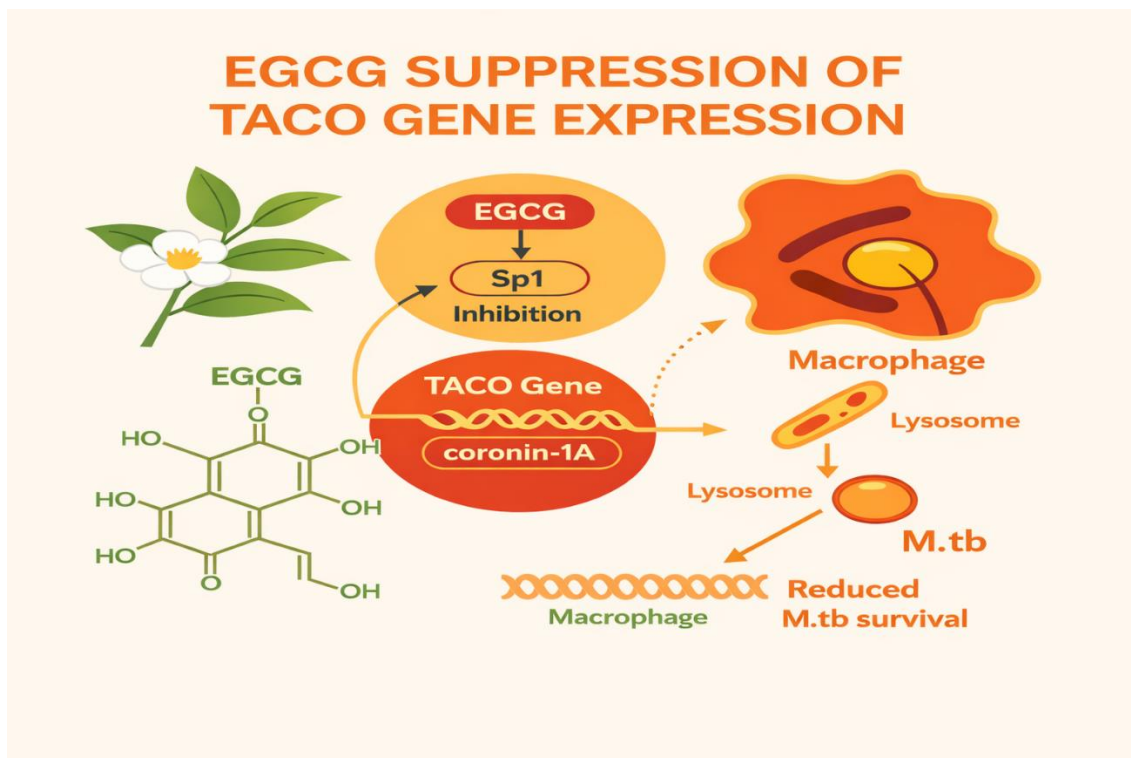


Figure 3: EGCG-Mediated Suppression of TACO Gene Expression.

This figure illustrates the molecular pathway through which EGCG exerts host-directed antitubercular effects. It shows EGCG inhibiting transcription factor Sp1, thereby reducing expression of the TACO (coronin-1A) gene in macrophages. With TACO downregulated, phagosomes can finally fuse with lysosomes, leading to effective killing of *Mycobacterium tuberculosis*. Confocal microscopy representations indicating colocalization of lysosomal markers with engulfed bacilli highlight the restored phagolysosomal fusion.

3.6. Toxicity and Safety: Essential for TB Adjunct Therapy: No mortality at 2000 mg/kg (acute), No significant histopathology changes in liver, kidney, heart, or spleen, Reversible biochemical fluctuations at high doses and no organ damage under subchronic treatment. For TB, which already strains liver function due to isoniazid and rifampicin, safety is as important as efficacy. *C. sinensis* exhibits a favourable safety margin suitable for long-term adjunct therapy.^[52]

3.7. Limitations of Current Antitubercular Evidence: The present body of evidence is limited by several factors, despite the increased interest in host-directed and phytochemical adjuncts for tuberculosis therapy. The majority of studies rely on the lack of *in vivo* TB animal model studies, the absence of direct Mtb MIC testing (MABA/REMA), the lack of fractionation-based identification of active principles other than EGCG, the variability in extract preparation across studies, the limited investigation of synergy with TB drugs (INH, RIF), and the lack of molecular docking data for tannins, flavonoids, or terpenoids.^[53]

4. Mechanistic Insights as Antifungal Potential of *Camellia sinensis*

The mechanistic basis of the antitubercular potential of *Camellia sinensis* can only be understood by combining its phytochemical diversity with immunological, biochemical and microbiological pathways involved in *Mycobacterium tuberculosis* (Mtb). The articles contained in your uploaded materials, especially the mechanistic assessment of EGCG in macrophages as well as the phytochemical and functional-group analysis, depict a consistent image of how the constituents of the plant have an effect on the host cells and the microbial structures.^[54] Unlike conventional antibiotics, which target bacterial enzymes or cell-wall components directly, green tea exhibits a dual mechanism of modulation, affecting host immune pathways (host-directed therapy) and biochemical interference with microbial viability through antioxidant, enzyme-inhibitory, and membrane-disruptive actions.^[55]

4.1. Host-Directed Therapy: The Central Mechanism of EGCG: The most important mechanistic discovery is the interaction of epigallocatechin-3-gallate (EGCG) with macrophage regulatory pathways that determine the fate of ingested Mtb. Unlike antibiotics, which attempt to kill bacilli directly, EGCG corrects the macrophage defect created by the bacterium.^[56]

4.1.1. The TACO gene: A barrier to phagosome maturation: *M. tuberculosis* survives inside macrophages by preventing normal phagosome–lysosome fusion. It does this primarily by upregulating the TACO gene (tryptophan-aspartate containing coat protein), also known as coronin-1A, and recruiting the

TACO protein to the phagosomal membrane. TACO acts as a molecular shield, preventing lysosomal enzymes from entering the phagosome. If TACO expression is not suppressed, the bacterium remains inside an immature phagosome, protected from immune destruction.^[57]

4.1.2. EGCG suppresses TACO via Sp1 inhibition:

EGCG inhibits transcription factor Sp1, Sp1 normally promotes TACO gene transcription, inhibiting Sp1 \Rightarrow downregulation of TACO mRNA, decreasing TACO

protein \Rightarrow restored phagosome–lysosome fusion, resulting in enhanced intracellular killing of Mtb. This mechanism is supported by RT-PCR evidence of decreased TACO transcripts, confocal microscopy showing increased fusion of phagosomes with lysosomes, and reduced Mtb CFU in EGCG-treated macrophages.^[58] This makes EGCG one of the rare plant-derived compounds with validated host-directed antitubercular activity **Figure 4**.

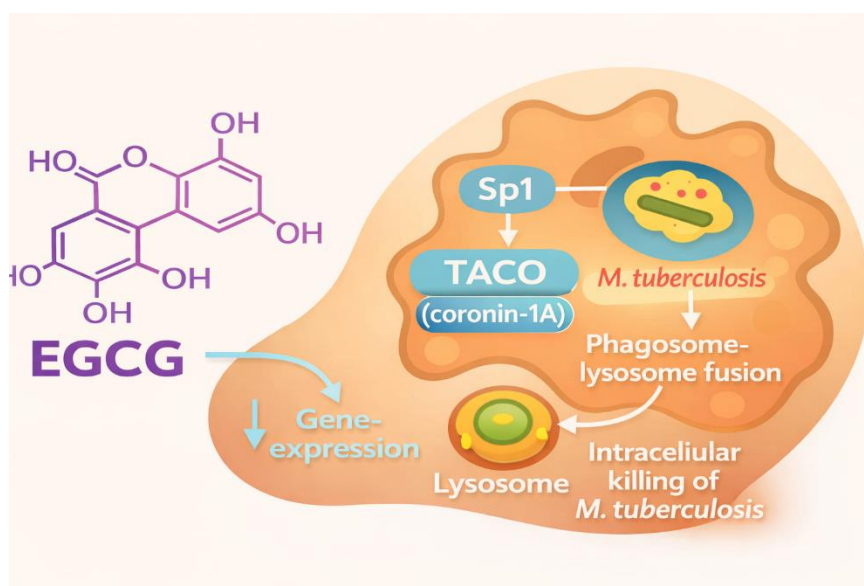


Figure 4: Integrated Host-Directed Mechanism of EGCG in Macrophages.

This figure unifies the complete host-directed mechanism triggered by EGCG. It highlights the molecular cascade starting from EGCG entry into macrophages, suppression of Sp1, down-regulation of TACO expression, restoration of phagosome maturation and ultimately enhanced intracellular killing of Mtb. Additional elements illustrate macrophage survival, reduced ROS burden, and improved immune signalling. The diagram shows how EGCG simultaneously strengthens host defence while disabling Mtb's evasive strategies. This integrated model bridges immunology, molecular biology and phytochemistry, offering readers a complete understanding of how one plant compound can reshape macrophage pathogen interactions.

4.2. Antioxidant Mechanisms and Redox Modulation:

TB infection causes extreme oxidative stress within lung tissue and macrophages. Excess ROS damages host tissue, impairs immune function and creates an environment that favours Mtb persistence.^[59]

4.2.1. Phenolics and catechins as ROS neutralisers:

The FTIR spectrum of *C. sinensis* shows abundant phenolic O–H groups—well-known for electron-donating ability. Catechins (EGCG, EGC, EC, ECG) are potent scavengers of H_2O_2 , hydroxyl radicals, peroxynitrite and DPPH free radicals.^[60]

4.2.2. Antioxidants protect macrophages: By reducing oxidative burden, *C. sinensis* protects macrophages from ROS-mediated apoptosis, preserves phagocytic function and enhances intracellular clearance potential.^[61] This indirect antitubercular mechanism improves host resilience while reducing tissue damage.

4.3. Membrane Disruption and Cell-Wall Interference:

The lipid-rich mycolic acid cell wall of Mtb is one of the main reasons for its drug resistance.

Phytochemicals in *C. sinensis*, especially tannins and terpenoids, interact with microbial membranes in ways relevant to TB.^[62]

4.3.1. Terpenoids and lipid insertion: Terpenoids (confirmed strongly by Salkowski test) integrate into lipid bilayers, altering membrane fluidity, proton gradients and ion permeability.^[63,64] This effect is particularly potent against bacteria with high lipid content, like Mtb.

4.3.2. Tannins disrupt protein–lipid structures: Tannins precipitate proteins, including membrane proteins, cell-wall enzymes and virulence factors. This results in enzyme inactivation, impaired cell wall integrity and inhibited bacterial adhesion.^[65] Together,

these mechanisms complement the host-directed actions of EGCG.

4.4. Enzyme Inhibition and Metabolic Stress Induction: Several phytochemicals—especially alkaloids, flavonoids, and phenolics act on bacterial enzymes, target pathways relevant to Mtb Topoisomerase inhibition (alkaloids), Fatty-acid synthase inhibition (phenolics), Energy metabolism disruption via NADH oxidase interference and ATP depletion through membrane-potential collapse. These effects cause metabolic stress, making intracellular Mtb more vulnerable.^[66,67]

4.5. Immune Modulation and Cytokine Balance: Polyphenols are known to modulate immune responses at the cytokine and transcriptional levels. Immune-modulating effects, including decreasing TNF- α and IL-6 (excessive inflammation worsens TB), increasing IL-12 and IFN- γ (enhances macrophage activation), promoting autophagy pathways, and enhancing antigen presentation. These immunomodulatory effects support macrophage-mediated Mtb clearance.^[68]

4.6. Synergistic Multi-Target Mechanisms: The most important insight is that *C. sinensis* does not rely on a single mechanism.^[69,70] Instead, its phytochemicals act synergistically across multiple pathways as presented in Table 2.

Table 2: Phytochemical action of synergistically multiple pathways.

Phytochemical Class	Mechanistic Effects
Catechins (EGCG)	Host-directed therapy → Suppresses TACO → Allows phagolysosomes fusion
Phenolics	Strong antioxidants → Reduce ROS → Protect immune cells
Tannins	Protein precipitation → Disrupt cell-wall enzyme systems
Terpenoids	Membrane disruption → Collapse proton gradients
Alkaloids	Topoisomerase inhibition → Impaired replication
Flavonoids	ROS modulation → Anti-inflammatory balance

This multi-targeted nature mirrors modern drug discovery strategies, where synergistic mechanisms are

preferred over single-target drugs for chronic infections like TB in Figure 5.

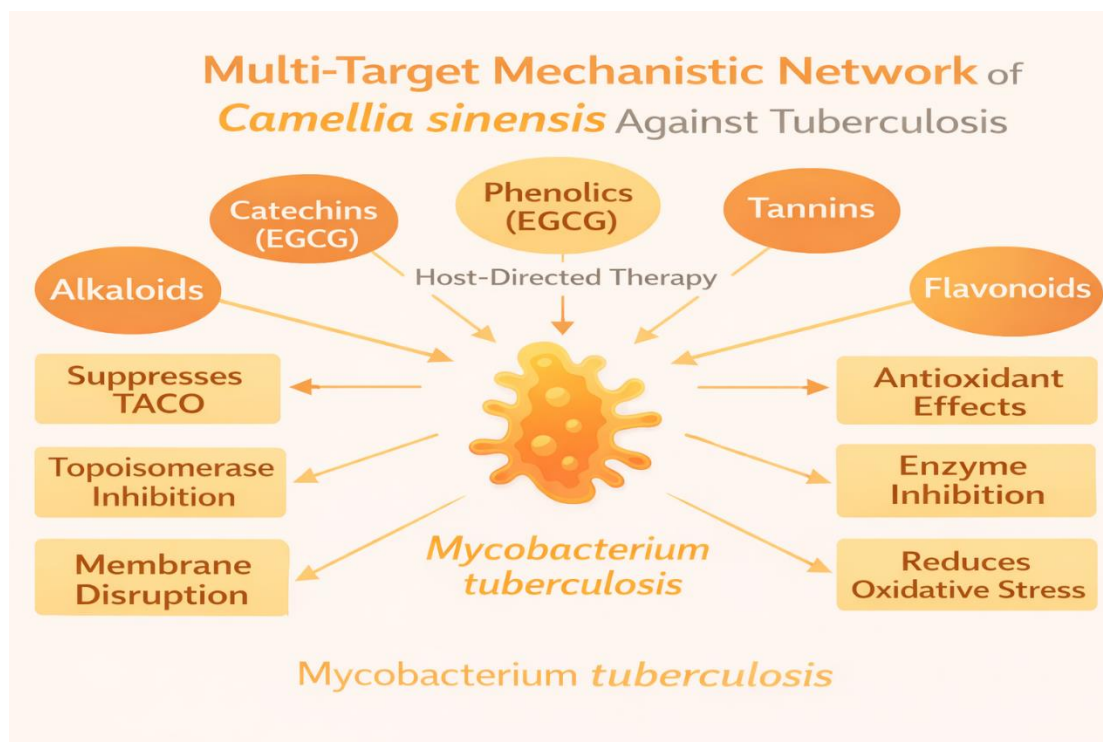


Figure 5: Multi-Target Mechanistic Network of *Camellia sinensis* Against Tuberculosis.

This figure overlaid all the key mechanistic pathways by which *Camellia sinensis* affects the pathology of tuberculosis. It combines antioxidant, enzyme inhibitors, topoisomerase disruption, disrupting membranes, immune modulation, and host-directed TACO suppression. All the phytochemical groups, catechins,

phenolics, tannins, terpenoids, flavonoids, and alkaloids, are related to certain biological pathways. Arrows depict the synergistic interactions, including the effect of antioxidant protection in increasing macrophage bactericidal ability or the effect of membrane

perturbation complementing the effect of enzyme inhibition.

4.7. Mechanistic Advantages Over Conventional TB

Drugs: Conventional antitubercular drugs have several critical drawbacks. Isoniazid is linked to a high risk of hepatotoxicity, mainly because of the formation of toxic metabolites during hepatic metabolism. Rifampicin accelerates the development of drug resistance, especially when treatment adherence is suboptimal or when used without sufficient combination therapy.^[71] Ethambutol is known to cause optic neuritis, which results in visual disturbances and impaired colour vision. Additionally, Pyrazinamide is linked to severe liver injury, often necessitating treatment modification or drug withdrawal.^[72] These adverse effects collectively underscore the need for safer and more effective therapeutic alternatives in tuberculosis management. Green tea exhibits several mechanistic advantages that differentiate it from conventional antimicrobial agents. Instead of exerting direct bactericidal pressure, its bioactive constituents—particularly EGCG—primarily function by enhancing host defence, instead of killing bacteria directly, reducing resistance risk.^[73,74] Antioxidant and immunomodulatory effects may reduce drug-induced toxicity and have no cross-resistance with antibiotic pathways because EGCG acts on macrophage genes (TACO/Sp1), not Metabolic enzymes.^[75]

5. CONCLUSION

The present review reveals that *Camellia sinensis* has a highly enriched and diverse phytochemical constituent, which directly or indirectly leads to its antibacterial effects on tuberculosis. Screening by qualitative methods continually showed high levels of phenolics, tannins, terpenoids, alkaloids and carbohydrates, which were confirmed by functional groups reported in FTIR and characterised as hydroxylated polyphenols, aromatic structures and lipid-active terpenoid moieties. These elements are the basis of the strong antioxidant, antimicrobial, antitumor, metabolic and immunomodulatory effect of the extract and create a solid pharmacological base applicable to tuberculosis studies. Antioxidant tests, especially the DPPH and the H₂O₂ scavenging tests, indicate a high capacity to neutralise free radicals, which is essential in reducing the oxidative stress of macrophages in infection by *Mycobacterium tuberculosis*. *C. sinensis* lowers the levels of ROS and maintains the integrity of immune cells, and assists the host against infections. Broad-spectrum antibacterial effect seen against many pathogenic bacteria, but not specific to *Mtb* (which is thought to occur by a combination of protein precipitation, membrane disruption and enzyme inhibition), acting on the lipid-rich mycobacterial cell wall. The strongest evidence comes out as a result of mechanistic research on epigallocatechin-3-gallate (EGCG). As opposed to the traditional antibiotics, which act on bacterial pathways, EGCG has a host-based

antitubercular effect, in which it inhibits transcription of the TACO gene through the action of Sp1. TACO down-regulation restores phagosome-lysosome fusion in macrophages, allowing them to successfully kill *Mtb* intracellularly. The pathway is particularly relevant in the context of increasing antimicrobial resistance, in which case host-directed therapies lower the selective pressure of bacterial populations and supplement current TB pharmacotherapy. Other phytochemicals may also have a supportive action by membrane interference, metabolic upset and redox and are part of a multi-target pharmacophenotype. The toxicological evaluation also indicates the safety of *C. sinensis* extracts, as no mortality or significant organ pathology was observed at elevated doses. This positive safety profile is critical to prolonged adjunct therapy in the treatment of TB, in which hepatotoxicity of first-line drugs is a major problem. The findings emphasise *Camellia sinensis* as a potential source of antitubercular research using plants, which have synergistic antioxidant, antimicrobial, and host-directed synergies. Nevertheless, major gaps still exist, such as the lack of direct *Mtb* MIC testing (MABA/REMA), only fractionation studies of EGCG, and *in vivo* confirmation. Future research should focus on isolating bioactive compounds, making predictions about molecular docking, utilising macrophage transcriptomics, and conducting combinatorial research with current TB medications.

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Conflicts of Interest

The authors declare no conflict of interest.

Abbreviations

Abbreviation	Definition
EGCG	Epigallocatechin-3-gallate
TACO	Tryptophan Aspartate Containing Coating Protein
TNF- α	Tumor Necrosis Factor
DPPH	2,2-diphenyl-1-picrylhydrazyl
IFN- γ	Interferon gamma
NADH	Nicotinamide Adenine Dinucleotide
MABA	Microplate Alamar Blue Assay
REMA	Resazurin Microtiter Assay
ROS	Reactive Oxygen Species
AMPK	AMP-activated protein kinase
CFU	Colony Forming Units
SCFA	short-chain fatty acids
Mtb	<i>Mycobacterium tuberculosis</i>
WHO	World Health Organization

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