ISSN: 2579-079X

International Digital Organization for Scientific Research IDOSR JST/26/1130000
IDOSR JOURNAL OF SCIENCE AND TECHNOLOGY 12(1):40-45, 2026.
https://doi.org/10.59298/IDOSR/JST/26/113.4045

Redox-Immune Crosstalk in Tuberculosis: The Role of Antioxidant Phytochemicals in Combating Oxidative Stress

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ABSTRACT

Tuberculosis (TB) unfolds within a chemically reactive battlefield where host-derived reactive oxygen and nitrogen species (ROS/RNS) intersect with immune signaling to shape disease outcomes. While oxidative stress is essential for mycobacterial control, sustained redox imbalance damages host tissues, disrupts granuloma integrity, fuels inflammation, and may even enhance Mycobacterium tuberculosis (Mtb) persistence. This review synthesizes current understanding of redox–immune crosstalk in TB pathogenesis and critically evaluates antioxidant phytochemicals as host-directed therapy (HDT) candidates. We outline how key pathways-Nrf2/Keap1, NF-κB, HIF-1α, MAPK, and JAK/STAT-integrate redox signals to calibrate macrophage activation, autophagy, antigen presentation, and adaptive immunity. We then survey major phytochemical classes (polyphenols, terpenoids, organosulfur compounds, alkaloids) with reported effects on ROS/RNS homeostasis, inflammasome activity, mitochondrial function, and immunometabolism. Emerging data suggest that select compounds can (i) enhance bactericidal mechanisms without exacerbating tissue injury, (ii) temper pathological inflammation, (iii) promote autophagy and phagosome maturation, and (iv) mitigate anti-TB drug toxicities. We highlight formulation advances that improve bioavailability, summarize preclinical/early clinical signals, and discuss safety, pharmacokinetic, and drug–drug interaction considerations. Finally, we identify knowledge gaps and propose a translational roadmap for integrating rigorously characterized phytochemicals into TB HDT regimens.

Keywords: Tuberculosis; Oxidative stress; Nrf2; Host-directed therapy; Phytochemicals

INTRODUCTION

TB remains a leading infectious killer [1]. Control of Mtb depends on a delicately balanced inflammatory response: enough oxidative and nitrosative pressure to restrict bacilli, but not so much that it drives immunopathology and cavitary disease [2]. Oxidative stress-classically attributed to NADPH oxidase-derived superoxide, inducible nitric oxide synthase (iNOS)-derived NO, and downstream radicals-interacts with cytokine networks, metabolism, and cell death programs [3]. Mtb exploits this terrain with robust antioxidant defenses, redox-sensing transcriptional programs, and strategies that subvert phagolysosomal killing. Host-directed strategies that recalibrate (rather than blunt) redox tone are therefore attractive adjuncts to standard chemotherapy, particularly for drug-resistant TB and patients with inflammatory comorbidities [4].

Oxidative Stress in TB Pathogenesis

Early innate phase. Upon encountering Mycobacterium tuberculosis, alveolar macrophages internalize bacilli and rapidly assemble the NOX2 complex, producing superoxide that dismutates to hydrogen peroxide [5]. Inducible nitric oxide synthase generates nitric oxide, which reacts with superoxide to form peroxynitrite capable of damaging bacterial proteins, lipids, and DNA [6]. These oxidants also signal for cytokine production and phagosome maturation. Yet an unchecked neutrophil influx amplifies extracellular ROS, degrading matrix, exposing new tissue niches, and propagating necrotic cell death that can favor bacillary spread [7].

Granuloma dynamics. Granulomas constrain infection but are redox-mosaic structures [8]. Hypoxia and ongoing NADPH oxidase activity elevate ROS, while lipid-laden foamy macrophages exhibit mitochondrial dysfunction, impaired β-oxidation, and heightened oxidative tone [9]. Persistent ROS/RNS oxidize lipids and proteins, yielding damage-associated molecular patterns that reinforce chemokine gradients and recruit additional neutrophils [10]. Oxidative injury stiffens the collagenous capsule, impairs vascular supply, and promotes caseation, all of which hinder antibiotic penetration and create sanctuaries for persisting organisms [11].

Systemic consequences. Chronic redox stress perturbs iron and zinc handling, disrupts epithelial barrier function, and skews antigen processing, diminishing T-cell priming and effector quality [12]. When unrestrained, this imbalance converts protective inflammation into tissue-destructive pathology.

Antioxidant Phytochemicals: Classes and Canonical Actions

Polyphenols (flavonoids and phenolic acids). Quercetin, epigallocatechin gallate, curcumin, resveratrol, and baicalin commonly activate Nrf2, dampen NF-κB signaling, modulate MAPKs, scavenge radicals, and promote autophagy and mitophagy [13]. Limited direct antimycobacterial activity is reported in vitro; their chief value lies in redox and inflammatory recalibration.

Terpenoids. Carotenoids, monoterpenes such as carvacrol, and triterpenoids including oleanolic and ursolic acids stabilize membranes, modulate ROS generation, and in some models enhance macrophage bactericidal pathways or inhibit efflux pumps, supporting host-directed activity [14].

Organosulfur compounds. Allicin and related thiosulfinates exert redox-active sulfur chemistry that can disrupt microbial thiol homeostasis while upregulating host antioxidant defenses, thereby lowering damaging oxidative tone without abolishing antimicrobial signaling [15].

Alkaloids. Berberine and piperine influence AMPK and NF-κB axes, stimulate autophagy, and fortify epithelial barriers [16]. Piperine also increases oral bioavailability of co-administered agents, a practical advantage for combination phytochemical strategies [17].

Others. Lignans such as silymarin/silibinin and selected coumarins provide hepatoprotective antioxidant effects with potential to mitigate isoniazid- and rifampicin-associated liver injury, enabling sustained chemotherapy while restraining collateral oxidative damage [18].

Mechanistic Interfaces with TB Immunobiology

Tuning ROS/RNS without immunoparesis. The aim is to narrow the oxidative "aperture," not close it. Mild activation of Nrf2 boosts glutathione synthesis, HO-1, and phase II detoxification, buffering host tissues while leaving NOX2- and iNOS-driven antimycobacterial mechanisms intact [19]. Temporal control matters: early containment benefits from preserved oxidants; later, Nrf2 help curbs destructive neutrophil-driven damage [20].

Autophagy enhancement. Curcumin, resveratrol, and berberine engage AMPK and inhibit mTOR, activating ULK1 and Beclin-1 to promote autophagy and mitophagy [21]. This improves phagosome–lysosome fusion, delivers antimicrobial peptides, and clears damaged mitochondria that otherwise leak ROS and DAMPs, improving intracellular control of Mtb.

Inflammasome calibration. Polyphenols modulate NLRP3 priming and activation by reducing mitochondrial ROS and preserving potassium homeostasis. The result is restrained IL-1β and IL-18 release that maintains antimicrobial tone while avoiding pyroptosis-driven tissue injury and neutrophil hyper-recruitment [22].

Immunometabolism. By nudging the AMPK–mTOR–HIF-1α axis, phytochemicals rebalance glycolysis and oxidative phosphorylation in macrophages and T cells [23]. This supports bactericidal functions such as nitric oxide production while preventing the pathological, lactate-heavy state that fuels chronic inflammation and granuloma breakdown.

Mitochondrial quality control. Agents that induce PGC-1α and mitophagy enhance biogenesis of healthy mitochondria, restore membrane potential, and limit release of cardiolipin and mitochondrial DNA that amplify inflammation [24]. Better mitochondrial fitness sustains macrophage endurance during prolonged infection.

Barrier and microbiome effects. Certain compounds strengthen epithelial tight junctions, reduce mucosal oxidative injury, and may shift gut microbial metabolites toward short-chain fatty acids that promote regulatory yet competent lung immunity [25]. The gut–lung axis is emerging as a lever to tune TB inflammation [26].

Evidence Landscape

In vitro macrophage systems consistently show that curcumin, resveratrol, quercetin, epigallocatechin gallate, and berberine reduce lipid peroxidation, restore glutathione, and enhance phagolysosomal maturation, yielding modest decreases in intracellular Mtb [27]. These effects track with Nrf2 induction, NF-κB attenuation, and AMPK activation. Murine models extend these findings: adjunct phytochemicals can shrink lesion size, lower bacterial burden, and limit caseation while improving hepatic histology during isoniazid and rifampicin exposure [28]; silymarin is notably hepatoprotective [29]. However, dosing ranges, extract standardization, and formulation strategies vary widely, complicating comparisons.

Human data remain sparse and heterogeneous. Small, short-duration adjunct studies report improvements in oxidative stress biomarkers such as malondialdehyde and reduced glutathione, stabilization of liver enzymes, and better symptom scores, but effects on time to culture conversion, radiographic healing, relapse, and mortality are not yet definitive. Overall, the translational signal is one of biological plausibility with promising surrogate outcomes, pending rigorously powered, biomarker-stratified trials using standardized preparations and validated pharmacokinetics.

Formulation and Pharmacokinetics

Many phytochemicals are highly lipophilic, chemically labile, and subject to rapid phase II conjugation, yielding low and variable oral bioavailability [30]. Nanoformulations such as liposomes, solid lipid nanoparticles, nanoemulsions, polymeric micelles, and cyclodextrin inclusion complexes improve apparent solubility, protect against degradation, and prolong circulation [31]. Ligand-decorated carriers (for example, mannose or Fc fragments) can exploit macrophage receptors to enrich drug within granulomas, while pH- or ROS-responsive polymers trigger release in acidic, oxidant-rich lesion cores [32]. Co-loaded systems that pair a phytochemical with isoniazid or rifampicin may harmonize intralesional exposure and reduce dosing complexity [33]. Phospholipid complexes (phytosomes) and amorphous solid dispersions increase Cmax and AUC by enhancing membrane permeability and reducing crystallinity; salt forms, prodrugs, and cocrystals are complementary tools that tune lipophilicity and metabolic stability [34]. Adjuvants like piperine or quercetin analogs can attenuate first-pass metabolism and efflux, but their own interaction liabilities require careful selection [35].

Pulmonary delivery is especially attractive for tuberculosis. Inhalable dry powders and nebulized nanosuspensions concentrate actives in the bronchial tree and alveoli, limit systemic exposure, and may achieve therapeutic levels in caseous material that oral dosing poorly reaches [36]. Mucus-penetrating particles and ultrafine aerosols improve epithelial translocation and macrophage uptake [37]. For any platform, pharmacokinetic endpoints should include plasma and epithelial lining fluid concentrations, lesion and caseum penetration, intracellular macrophage levels, and half-life within lung tissue. Food effects, protein binding, and enterohepatic recycling can meaningfully shift exposure; population PK that accounts for cachexia, HIV co-infection, and diabetes is essential before moving to dose-ranging trials.

Drug–Drug Interactions (DDIs) and Safety

Polyphenols and related compounds variably modulate CYP1A2, 2C9, 2C19, 2D6, and 3A4, as well as UGTs and transporters such as P-gp and BCRP [38]. Rifampicin is a strong inducer of 3A and several transporters, potentially lowering phytochemical exposure [39]; conversely, phytochemical inhibition of CYPs or P-gp could alter levels of isoniazid, bedaquiline, linezolid, clofazimine, or azoles [40]. Thorough DDI screening and staggered dosing plans are prudent. Silymarin and related lignans may mitigate drug-induced liver injury, but perceived protection can delay recognition of rising aminotransferases; routine monitoring of ALT, AST, bilirubin, and clinical symptoms remains mandatory [41]. Excessive suppression of NF-κB or ROS risks blunting bactericidal immunity; timing and dose should favor tissue protection without compromising early antimicrobial signaling. Comorbidities shape safety and efficacy: malnutrition alters protein binding [42]; diabetes and HIV change redox tone and enzyme expression [43]; antiretroviral regimens introduce additional CYP and transporter interactions. Genetic variability in NAT2, GST, and Nrf2 pathways may further stratify responders [44]. Finally, quality control matters: standardized extracts, contaminant testing for heavy metals and mycotoxins, and clear labeling of active content are nonnegotiable for clinical translation, especially in pregnancy or hepatic disease where safety margins are narrow [45, 46].

CONCLUSION

TB pathogenesis is inseparable from redox biology. The same oxidants that help control Mtb can, when dysregulated, drive lung destruction and favor bacterial persistence. Antioxidant phytochemicals offer a nuanced means to recalibrate this landscape by activating cytoprotective programs (Nrf2), fine-tuning inflammatory signaling (NF-κB/MAPK), supporting autophagy, and preserving mitochondrial function—while potentially mitigating drug toxicities. The promise is real but conditional: standardized compounds, targeted delivery, biomarker-guided selection, and rigorous clinical trials are essential to translate biochemical plausibility into durable patient benefit. As part of a broader HDT strategy, well-characterized phytochemicals could help tip the redox–immune balance toward bacterial clearance with less collateral damage.

REFERENCES

1. World Health Organization: WHO, World Health Organization: WHO. Tuberculosis. 2025. Available from: https://www.who.int/news-room/fact-sheets/detail/tuberculosis
2. Rahman F. Characterizing the immune response to Mycobacterium tuberculosis: a comprehensive narrative review and implications in disease relapse. Frontiers in Immunology. 2024;15. doi:10.3389/fimmu.2024.1437901
3. Uhuo E N, Egba S I, Nwuke P C, Obike C A and Kelechi G K. Antioxidative properties of Adansonia digitata L. (baobab) leaf extractexert protective effect on doxorubicin induced cardiac toxicity in Wistar rats. Clinical Nutrition Open Science 2022; 45:3-16
4. Dong W, Wang G, Bai Y, Li Y, Zhao L, Lu W, et al. Repurposing an Antioxidant to Kill Mycobacterium tuberculosis by Targeting the 50S Subunit of the Ribosome. Biomolecules. 2023;13(12):1793. doi:10.3390/biom13121793

5. Cohen SB, Gern BH, Delahaye JL, Adams KN, Plumlee CR, Winkler JK, et al. Alveolar Macrophages Provide an Early Mycobacterium tuberculosis Niche and Initiate Dissemination. Cell Host & Microbe. 2018;24(3):439-446.e4. doi:10.1016/j.chom.2018.08.001
6. Alum, E.U., Uti, D.E. & Offor, C.E. Redox Signaling Disruption and Antioxidants in Toxicology: From Precision Therapy to Potential Hazards. *Cell Biochem Biophys* (2025). https://doi.org/10.1007/s12013-025-01846-8
7. Aroca-Crevillén A, Vicanolo T, Ovadia S, Hidalgo A. Neutrophils in physiology and Pathology. Annual Review of Pathology Mechanisms of Disease. 2024;19(1):227–59. doi:10.1146/annurev-pathmechdis-051222-015009
8. Menshchikova E, Zenkov N, Tkachev V, Potapova O, Cherdantseva L, Shkurupiy V. Oxidative stress and Free-Radical oxidation in BCG granulomatosis development. Oxidative Medicine and Cellular Longevity. 2013;2013:1–8. doi:10.1155/2013/452546
9. Liu S, Liu J, Wang Y, Deng F, Deng Z. Oxidative stress: signaling pathways, biological functions, and disease. MedComm. 2025;6(7). doi:10.1002/mco2.70268
10. Manoharan RR, Prasad A, Pospíšil P, Kzhyshkowska J. ROS signaling in innate immunity via oxidative protein modifications. Frontiers in Immunology. 2024;15. doi:10.3389/fimmu.2024.1359600
11. Ugwu, CE., Sure, SM., Dike, CC., Okpoga, NA and Egba, SI. Phytochemical and *in vitro* antioxidant activities of methanol leave extract of *Alternanthera basiliana*. Journal of Pharmacy Research, 2018; 12(6): 835-839
12. Kim EH, Baek SM, Kim D, Choi S, Kim W, Bian Y, et al. Iron overload and oxidative stress participated in zinc oxide nanoparticles-induced blood-brain barrier dysfunction. Ecotoxicology and Environmental Safety. 2025;301:118528. doi:10.1016/j.ecoenv.2025.118528
13. Cione E, La Torre C, Cannataro R, Caroleo MC, Plastina P, Gallelli L. Quercetin, epigallocatechin gallate, curcumin, and resveratrol: from dietary sources to human MicroRNA modulation. Molecules. 2019;25(1):63. doi:10.3390/molecules25010063
14. López-García S, Castañeda-Sanchez J, Jiménez-Arellanes A, Domínguez-López L, Castro-Mussot M, Hernández-Sanchéz J, et al. Macrophage Activation by Ursolic and Oleanolic Acids during Mycobacterial Infection. Molecules. 2015;20(8):14348–64. doi:10.3390/molecules200814348
15. Okada Y, Tanaka K, Fujita I, Sato E, Okajima H. Antiodidant activity of thiosulfinates derived from garlic. Redox Report. 2005;10(2):96–102. doi:10.1179/135100005x38851
16. Li W, Hua B, Saud SM, Lin H, Hou W, Matter MS, et al. Berberine regulates AMP-activated protein kinase signaling pathways and inhibits colon tumorigenesis in mice. Molecular Carcinogenesis. 2014;54(10):1096–109. doi:10.1002/mc.22179
17. Tiwari A, Mahadik KR, Gabhe SY. Piperine: A comprehensive review of methods of isolation, purification, and biological properties. Medicine in Drug Discovery. 2020;7:100027. doi:10.1016/j.medidd.2020.100027
18. Eminzade S, Uras F, Izzettin FV. Silymarin protects liver against toxic effects of anti-tuberculosis drugs in experimental animals. Nutrition & Metabolism. 2008;5(1). doi:10.1186/1743-7075-5-18
19. Zhang Q, Liu J, Duan H, Li R, Peng W, Wu C. Activation of Nrf2/HO-1 signaling: An important molecular mechanism of herbal medicine in the treatment of atherosclerosis via the protection of vascular endothelial cells from oxidative stress. Journal of Advanced Research. 2021;34:43–63. doi:10.1016/j.jare.2021.06.023
20. Xie T, Xu J, Liu X, Yu Y, Lu Y, Wang T, et al. Mechanistic insights into Nrf2-driven pathogenesis and therapeutic targeting in spinal cord injury. Frontiers in Immunology. 2025;16. doi:10.3389/fimmu.2025.1574834
21. McCubrey JA, Lertpiriyapong K, Steelman LS, Abrams SL, Yang LV, Murata RM, et al. Effects of resveratrol, curcumin, berberine and other nutraceuticals on aging, cancer development, cancer stem cells and microRNAs. Aging. 2017;9(6):1477–536. doi:10.18632/aging.101250
22. Nani A, Tehami W. Targeting inflammasome pathway by polyphenols as a strategy for pancreatitis, gastrointestinal and liver diseases management: an updated review. Frontiers in Nutrition. 2023;10. doi:10.3389/fnut.2023.1157572
23. Wang T, Liu H, Lian G, Zhang SY, Wang X, Jiang C. HIF1α-Induced glycolysis metabolism is essential to the activation of inflammatory macrophages. Mediators of Inflammation. 2017;2017:1–10. doi:10.1155/2017/9029327
24. Fontecha-Barriuso M, Martin-Sanchez D, Martinez-Moreno J, Monsalve M, Ramos A, Sanchez-Niño M, et al. The role of PGC-1α and mitochondrial biogenesis in kidney diseases. Biomolecules. 2020;10(2):347. doi:10.3390/biom10020347
25. Yang S, Liu H, Liu Y. Advances in intestinal epithelium and gut microbiota interaction. Frontiers in Microbiology. 2025;16. doi:10.3389/fmicb.2025.1499202
26. Enjeti A, Sathkumara HD, Kupz A. Impact of the gut-lung axis on tuberculosis susceptibility and progression. Frontiers in Microbiology. 2023;14. doi:10.3389/fmicb.2023.1209932

27. Li C, Xu Y, Zhang J, Zhang Y, He W, Ju J, et al. The effect of resveratrol, curcumin and quercetin combination on immuno-suppression of tumor microenvironment for breast tumor-bearing mice. Scientific Reports. 2023;13(1). doi:10.1038/s41598-023-39279-z
28. Fatima S, Kumari A, Dwivedi VP. Advances in adjunct therapy against tuberculosis: Deciphering the emerging role of phytochemicals. MedComm. 2021;2(4):494–513. doi:10.1002/mco2.82
29. Jaffar HM, Al-Asmari F, Khan FA, Rahim MA, Zongo E. Silymarin: Unveiling its pharmacological spectrum and therapeutic potential in liver diseases—A comprehensive narrative review. Food Science & Nutrition. 2024;12(5):3097–111. doi:10.1002/fsn3.4010
30. Alum EU, Uti DE, Egba SI, Ugwu OP-C, Aja PM. The Role of Phytochemicals in Age-Related Cognitive Decline: A Natural Solution for Brain Health. *Natural Product Communications*. 2025;20(6). doi:10.1177/1934578X251350761
31. Nwuruku, O.A., Ugwu, O.P.-C., Uti, D.E., Alum, B.N., Edwin, N.: Harnessing nature: plant-derived nanocarriers for targeted drug delivery in cancer therapy. Phytomedicine Plus. 5, 100828 (2025). https://doi.org/10.1016/j.phyplu.2025.100828
32. Paurević M, Gajdošik MŠ, Ribić R. Mannose ligands for mannose receptor targeting. International Journal of Molecular Sciences. 2024;25(3):1370. doi:10.3390/ijms25031370
33. Akkerman OW, Dijkwel RDC, Kerstjens H a. M, Van Der Werf TS, Srivastava S, Sturkenboom MGG, et al. Isoniazid and rifampicin exposure during treatment in drug-susceptible TB. The International Journal of Tuberculosis and Lung Disease. 2023;27(10):772–7. doi:10.5588/ijtld.22.0698
34. Lu M, Qiu Q, Luo X, Liu X, Sun J, Wang C, et al. Phyto-phospholipid complexes (phytosomes): A novel strategy to improve the bioavailability of active constituents. Asian Journal of Pharmaceutical Sciences. 2018;14(3):265–74. doi:10.1016/j.ajps.2018.05.011
35. Kesarwani K, Gupta R. Bioavailability enhancers of herbal origin: An overview. Asian Pacific Journal of Tropical Biomedicine. 2013;3(4):253–66. doi:10.1016/s2221-1691(13)60060-x
36. Chan HW, Chow S, Zhang X, Zhao Y, Tong HHY, Chow SF. Inhalable nanoparticle-based dry powder formulations for respiratory diseases: Challenges and strategies for Translational research. AAPS PharmSciTech. 2023;24(4). doi:10.1208/s12249-023-02559-y
37. Ensign LM, Schneider C, Suk JS, Cone R, Hanes J. Mucus penetrating nanoparticles: biophysical tool and method of drug and gene delivery. Advanced Materials. 2012;24(28):3887–94. doi:10.1002/adma.201201800
38. Korobkova EA. Effect of natural polyphenols on CYP metabolism: Implications for diseases. Chemical Research in Toxicology. 2015;28(7):1359–90. doi:10.1021/acs.chemrestox.5b00121
39. Chattopadhyay N, Kanacher T, Casjens M, Frechen S, Ligges S, Zimmermann T, et al. CYP3A4-mediated effects of rifampicin on the pharmacokinetics of vilaprisan and its UGT1A1-mediated effects on bilirubin glucuronidation in humans. British Journal of Clinical Pharmacology. 2018;84(12):2857–66. doi:10.1111/bcp.13750
40. Desta Z, Soukhova NV, Flockhart DA. Inhibition of cytochrome P450 (CYP450) isoforms by isoniazid: potent inhibition of CYP2C19 and CYP3A. Antimicrobial Agents and Chemotherapy. 2001;45(2):382–92. doi:10.1128/aac.45.2.382-392.2001
41. De Avelar CR, Pereira EM, De Farias Costa PR, De Jesus RP, De Oliveira LPM. Effect of silymarin on biochemical indicators in patients with liver disease: Systematic review with meta-analysis. World Journal of Gastroenterology. 2017;23(27):5004. doi:10.3748/wjg.v23.i27.5004
42. Ngcobo NN. Malnutrition and its Effect on drug pharmacokinetics: A Clinical perspective. Clinical Pharmacokinetics. 2025. doi:10.1007/s40262-025-01558-5
43. Lan X, Cheng K, Chandel N, Lederman R, Jhaveri A, Husain M, et al. High glucose enhances HIV entry into T cells through upregulation of CXCR4. Journal of Leukocyte Biology. 2013;94(4):769–77. doi:10.1189/jlb.0313142
44. Malinga TVB, Othman H, Paximadis M, Tiemessen CT, Ramsay M, Hazelhurst S, et al. Characterization of NAT, GST, and CYP2E1 genetic variation in Sub-Saharan African populations: Implications for treatment of tuberculosis and other diseases. Clinical Pharmacology & Therapeutics. 2025. doi:10.1002/cpt.3557
45. Manjula, V.S., Uti, D.E., Echegu, D.A., Ugwu, O.P.C., Egba, S.I., Agu, P.C. (2025). Metabolomics-Driven Standardization of Herbal Medicine: Advances, Applications, and Sustainability Considerations. *Natural Product Communications*. 2025;20(8). doi:10.1177/1934578X251367650
46. Singh J, Mehta A. Rapid and sensitive detection of mycotoxins by advanced and emerging analytical methods: A review. Food Science & Nutrition. 2020;8(5):2183–204. doi:10.1002/fsn3.1474

CITE AS: Mugisha Emmanuel K. (2026). Redox-Immune Crosstalk in Tuberculosis: The Role of Antioxidant Phytochemicals in Combating Oxidative Stress.
IDOSR JOURNAL OF SCIENCE AND TECHNOLOGY 12(1):40-45. https://doi.org/10.59298/IDOSR/JST/26/113.4045