Novel Approaches To Cutaneous Tuberculosis In Pharmacy

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Abstract
Cutaneous tuberculosis (CTB) is a rare extrapulmonary form of Mycobacterium tuberculosis infection manifesting in the skin. It can present with diverse clinical forms (e.g. lupus vulgaris, scrofuloderma, tuberculids) determined by infection route and host immunity. Conventional treatment follows standard anti-TB chemotherapy (2 months of isoniazid, rifampin, ethambutol, pyrazinamide followed by 4 months of isoniazid/rifampin). However, CTB poses diagnostic challenges (paucibacillary lesions often yield negative smears) and treatment issues (prolonged therapy, drug resistance). Recent pharmacy-oriented innovations are emerging: for example, macrophage-targeted polydopamine nanoparticles delivering rifampicin enable photothermal killing of M. tuberculosis in skin lesions; cationic nanoemulsion gels greatly enhance transdermal rifampicin penetration and efficacy against CTB; lipid nanovesicles (niosomes) encapsulating clofazimine and other TB drugs have been formulated for topical CTB therapy. Bio-inspired "cell-membrane-coated" nanocarriers (e.g. macrophage membrane nanoparticles) can evade immunity and home to infected sites. This review summarizes CTB pathology, current treatments, and novel pharmaceutical strategies – including advanced drug delivery systems, topical formulations, and immunotherapy – with comparative analysis (see Figures 1–5 and Tables 1–5). These innovative approaches aim to improve drug targeting, reduce toxicity, and overcome resistance in CTB management.

Introduction

Tuberculosis (TB) remains a global health threat, with ~10–11 million cases and ~1–1.5 million deaths annually. Cutaneous tuberculosis (CTB) is an uncommon extrapulmonary manifestation, comprising about 1–3% of extrapulmonary TB cases worldwide [1]. It occurs when M. tuberculosis infects the skin or dermal tissues via exogenous inoculation or endogenous spread. CTB typically presents as chronic plaques, nodules, ulcers, or abscesses. The most common form is lupus vulgaris (60–70% of CTB) – a slowly progressive plaque often on the face or neck – while scrofuloderma (contiguous spread from nodes/bones) and tuberculids (hypersensitivity reactions) are also observed. Figure 2 shows the rod-shaped M. tuberculosis bacilli responsible for CTB (acid-fast, aerobic rods) [2][3].

Effective management of CTB requires prompt diagnosis and treatment. Diagnosis relies on characteristic histology (caseating granulomas with few acid-fast bacilli), mycobacterial culture or PCR of biopsy specimens, and immune tests (positive tuberculin skin test or interferon-gamma release assay). However, lesions often harbor scant bacilli, leading to false negatives; thus, a combination of clinical, pathological and molecular evidence is used. Once diagnosed, CTB is treated with standard anti-TB chemotherapy following pulmonary TB guidelines: typically, 2 months of isoniazid (H), rifampicin (R), pyrazinamide (Z) and ethambutol (E), followed by 4 months of H and R. Adjunctive measures (surgical debridement of cold abscesses, corticosteroids for severe inflammation) may be needed in complicated cases [4][5]. Treatment success rates are high for drug-susceptible TB when adherence is maintained. However, rising drug resistance and long treatment duration have spurred the exploration of novel therapies, especially topically or systemically targeting skin lesions [6].

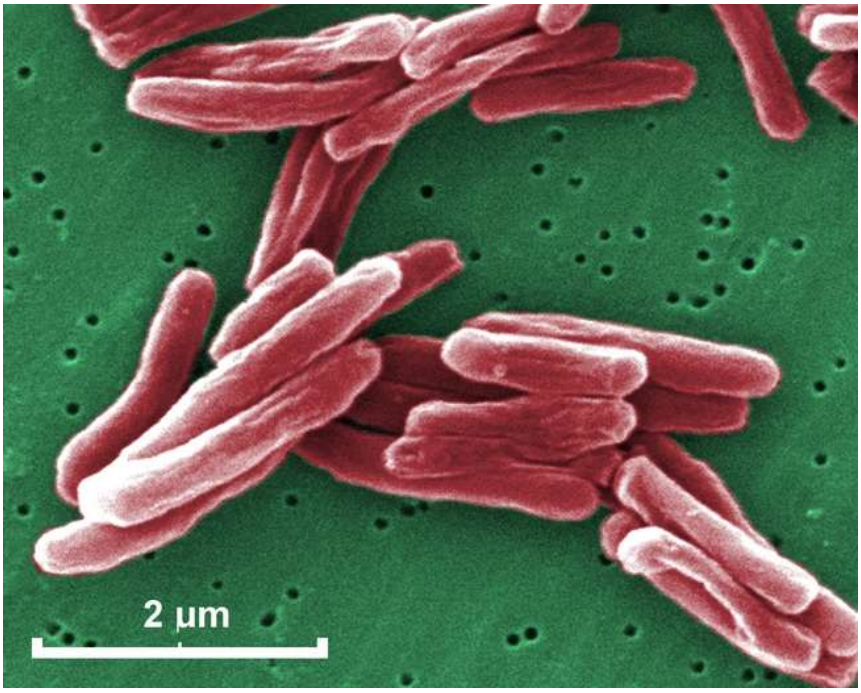

Figure 2: Scanning electron micrograph of M. tuberculosis bacilli (false-colored). These acid-fast rod-shaped bacteria are the etiologic agent of CTB.

Classification and Clinical Forms

CTB lesions are classified by infection route and host immunity. True CTB (bacteria present) includes exogenous forms (direct inoculation) and endogenous forms (contiguous or hematogenous spread). Exogenous CTB occurs in individuals with or without prior TB immunity: Tuberculous chancre (primary inoculation) arises in a naïve host; TB verrucosa cutis (warty TB) occurs with prior immunity. Endogenous CTB results from spread from internal foci: scrofuloderma (contiguous spread from lymph nodes, bones, etc.); lupus vulgaris (hematogenous or lymphatic spread in moderately immune hosts); metastatic tuberculous abscess (gumma) in immunocompromised individuals; and acute miliary CTB (disseminated form). The tuberculids are paucibacillary hypersensitivity reactions (papulonecrotic tuberculid, erythema induratum of Bazin, lichen scrofulosorum) occurring in individuals with moderate to high TB immunity. Table 1 summarizes key forms of CTB, their modes of infection, and clinical features [7][8].

Table 1: Clinical forms of cutaneous tuberculosis. The classification is based on infection route and host immunity.

Form	Route/Source	Immune status	Key Features
Tuberculous chancre (primary TB)	Skin inoculation (direct)	No prior TB immunity	Painless papule ulcerating into chancre; regional lymphadenopathy
TB verrucosa cutis (warty TB)	Skin inoculation (direct)	High TB immunity	Warty plaque (often on hands/feet) with verrucous surface; lesion may heal spontaneously
Lupus vulgaris	Hematogenous or contiguous spread	Moderate/high TB immunity	Reddish-brown plaque with “apple-jelly” nodules on diascopy; slow progressive course

Scrofuloderma	Contiguous spread (from lymph node/bone)	Variable	Cold abscesses and ulcerations overlying infected node; common on neck/jaw
Metastatic TB abscess (gumma)	Hematogenous dissemination	Low TB immunity	Deep subcutaneous abscesses that form draining sinus tracts; often in children
Acute miliary CTB	Hematogenous dissemination (widespread)	Low TB immunity	Numerous millet-sized papules/ulcers; indicates severe dissemination
Tuberculids (e.g. erythema induratum, papulonecrotic)	Hypersensitivity to TB antigens	Moderate/high immunity	Necrotic papules/nodules on extremities; no viable bacilli; suggests internal TB focus

Figure 5 shows a classic lupus vulgaris lesion (plaque on the cheek). Chronic lesions like this reflect high host immunity controlling bacillary burden.

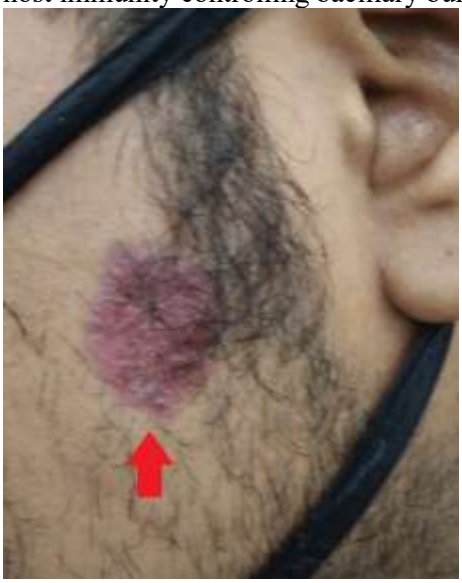

Figure 5: Lupus vulgaris on the face (arrow), a chronic paucibacillary form of CTB. Patients often do not recall a specific exposure; diagnosis relies on biopsy/histology.

Diagnosis of CTB

Diagnosing CTB requires a high index of suspicion. Histopathology is cornerstone: skin biopsy typically shows tuberculoid granulomas with caseation, epithelioid histiocytes, and Langhans giant cells. Ziehl–Neelsen or Auramine stains may reveal rare acid-fast bacilli, but bacilli are often not seen in paucibacillary forms [9]. Culture of tissue for M. tuberculosis (on Lowenstein-Jensen or MGIT media) is the gold standard but slow. PCR amplification of mycobacterial DNA from skin tissue can be rapid and sensitive, although specialized lab support is needed. Immunologic tests support the diagnosis: a strong tuberculin skin test (e.g. ≥10 mm induration) or a positive interferon-gamma release assay (IGRA) suggests TB exposure. Radiologic evaluation (e.g. chest X-ray, CT) is performed to seek pulmonary or other extrapulmonary foci in suspected CTB. **Table 2** summarizes diagnostic modalities for CTB [10][11].

Table 2: Diagnostic methods for cutaneous tuberculosis.

Test	Method	Comments
Skin biopsy & histopathology	Histologic exam (H&E); special stains	Granulomatous inflammation; AFB often scarce; caseation may be seen. Requires experienced dermatopathologist.
Culture of biopsy/tissue	Mycobacterial culture (solid/liquid)	Confirms infection; slow (weeks). Often low yield in paucibacillary lesions.
PCR / Nucleic acid tests	DNA amplification (e.g. GeneXpert®)	Rapid (<24–48 h) detection; higher sensitivity than smear in CTB. Cannot distinguish live/dead bacilli.
Tuberculin skin test (PPD)	Mantoux intradermal injection	Positive test indicates TB exposure, not specific for active disease. Useful supportive evidence.
Interferon-gamma release assay (IGRA)	Blood assay (e.g. QuantiFERON-TB)	Measures immune response to TB antigens; not affected by BCG. Same caveat as PPD.
Imaging (X-ray/CT/MRI)	Radiology of chest or lesion site	Can identify hidden TB foci (e.g. pulmonary TB or bone TB).
Dermoscopy	Polarized skin microscopy	Non-invasive; lupovus vulgaris shows apple-jelly spots. (Emerging technique)
Skin test with auramine-rhodamine	Fluorescence microscopy	More sensitive staining for AFB in tissue specimens than traditional Ziehl-Neelsen.

Standard Treatment and Its Limitations

In CTB, chemotherapy is essentially the same as for pulmonary TB. The first-line regimen is a 6-month course: 2 months of HRZE (isoniazid, rifampin, pyrazinamide, ethambutol) followed by 4 months of HR. Fixed-dose combination pills are commonly used. In tuberculids (hypersensitivity lesions), therapy is identical because an active TB focus is presumed. Treatment adherence is critical. Standard therapy cures over 95% of drug-susceptible TB cases if fully completed [12][13].

However, several issues complicate treatment: prolonged duration (6–9 months) often leads to incomplete adherence and default. Hepatotoxicity is a concern, especially with isoniazid and pyrazinamide (monitoring liver enzymes is required). Moreover, rising drug resistance (MDR/XDR-TB) limits effectiveness: MDR-TB (resistant to at least H and R) currently requires specialized regimens. New anti-TB drugs have been developed recently for resistant cases: bedaquiline (2012), delamanid (2014), and pretomanid (2019) target different pathways and shorten therapy for MDR/XDR-TB. Current WHO guidelines now include the BPaL/M regimen (bedaquiline + pretomanid + linezolid, with or without moxifloxacin) for XDR-TB. While these are systemic therapies, their skin penetration and role in CTB specifically are still under study [14][15].

Because of these challenges, novel delivery approaches are being studied to improve drug targeting in CTB. For example, local intralesional injections of drugs like streptomycin have historical precedent. Modern strategies aim to deliver drugs directly into skin lesions or via skin-friendly carriers to achieve high local concentrations while reducing systemic side effects [16].

Nanotechnology and Novel Drug Delivery

Nanotechnology offers innovative drug-delivery platforms for TB (including CTB). Nanocarriers (liposomes, nanoparticles, dendrimers, nanotubes, etc.) can encapsulate anti-TB drugs, improving solubility and targeting. Their small size and surface properties promote uptake by macrophages – the very cells that harbor M. tuberculosis – enabling intracellular delivery. Ligand-functionalization (e.g. mannose decoration) can further direct nanoparticles to macrophage receptors. Figure 1 illustrates a strategy: nanoparticles coated with macrophage membrane proteins serve as “stealth” carriers that home to infection sites. Such bioinspired carriers combine the targeting of immune cells with drug payload [17][18].

Nanocarrier advantages include reduced toxicity, controlled release, and potential co-delivery of multiple drugs or adjuvants. Examples under investigation include rifampicin-loaded dendrimers and polymeric

nanoparticles, isoniazid-conjugated nanotubes, and lipid nanocarriers. Ahmed et al. developed PEGylated PAMAM dendrimers encapsulating rifampicin, achieving high drug loading and slow release with minimal toxicity. Chen et al. formulated isoniazid-chitosan carbon nanotubes; these promoted wound healing in a TB-ulcer animal model and showed superior ulcer healing compared to free drug. Van Zyl and Viljoen prepared elastic lipid vesicles (niosomes) loaded with clofazimine and other TB drugs for **topical** CTB therapy; clofazimine-loaded niosomes showed the highest antimycobacterial activity in vitro. These nanocarriers are summarized in Table 3 [19][20].

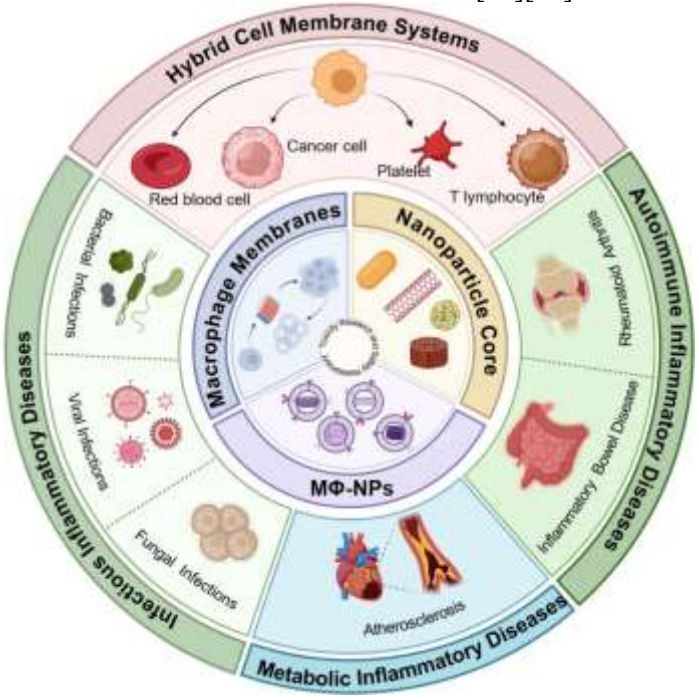

Figure 1: Schematic of macrophage-membrane-coated nanoparticles (MΦ-NPs) for targeted TB therapy. Coating nanoparticles with immune cell membranes enhances immune evasion and directs them to infected tissues. Such carriers can deliver drug payloads to macrophages harboring M. tuberculosis. (Adapted from Biomaterials Research)

Table 3: Selected nanocarrier-based therapies for TB/CTB. These platforms enhance drug delivery to infected macrophages.

Nanocarrier & Modifications	Payload / Function	Application / Results	References
Mannose-functionalized mesoporous polydopamine NPs	Rifampicin + photothermal agent (NIR absorber)	Selectively targets macrophages via mannose receptor; NIR laser triggers local heating and rapid rifampicin release, killing M.tb in CTB lesions. Demonstrated efficacy in mouse CTB model without systemic toxicity.	[11] (2023)
Cationic lipid nanoemulsion gel	Rifampicin	Enhances transdermal drug flux and skin deposition. In vivo, transdermal delivery achieved ~4-fold higher plasma levels than oral rifampicin, suggesting efficacy for cutaneous and systemic TB.	[69] (2020)
PEGylated PAMAM dendrimers	Rifampicin	High encapsulation (>60%), extended release and low toxicity. Potential for sustained drug levels in tissues.	[64] (2021)

Isoniazid-chitosan carbon nanotubes	Isoniazid	Prolonged drug release, reduced cytotoxicity. In animal TB ulcers, markedly reduced CD4+ T-cell infiltration (less inflammation) and accelerated healing compared to free isoniazid.	[64] (2019)
Lipid nanovesicles (niosomes)	Clofazimine, artemisone, decoquinate	Topical delivery to CTB lesions. In vitro, 1% clofazimine-niosomes achieved strongest M.tb inhibition. Demonstrates feasibility of topical nanotherapy for CTB.	[30] (2019)
Liposomal antigen (vaccine)	Tuberculosis fusion protein (ID93 + GLA adjuvant)	Post-BCG booster. Liposomal vaccine induced high IFN-γ and IL-12 levels, enhancing immune response. (TB vaccine development context.)	[64] (2019)

Photothermal and Photodynamic Therapies

An emerging strategy combines antimicrobial drugs with photothermal therapy (PTT) or photodynamic therapy (PDT) for CTB. Photothermal systems use nanoparticles that absorb near-infrared (NIR) light to generate localized heat, killing bacteria and triggering drug release. Fan et al. engineered mannose-modified polydopamine nanoparticles loaded with rifampicin. Upon NIR laser irradiation of the CTB lesion, the nanoparticles heat up (PTT) and simultaneously release rifampicin inside macrophages. This dual action resulted in significant M.tb reduction in a murine cutaneous TB model without damaging surrounding tissue [21]. The polydopamine NPs also upregulated host antioxidant defenses (Nrf2/HO-1) to inhibit ferroptosis, while promoting autophagy of infected macrophages. These novel "theranostic" nanoparticles illustrate how PTT can synergize with conventional drugs to overcome drug-resistant TB [22].

Photodynamic therapy – using light-activated photosensitizers to generate reactive oxygen species – has also been tested in TB (e.g. methylene blue or porphyrin formulations against M.tb). While primarily studied in vitro or for lung TB, PDT could be applied topically to CTB ulcers. However, concerns about skin penetration and potential toxicity remain. Combinations of laser/UV light with antibiotics for CTB are under preliminary investigation [23].

Topical Formulations and Local Therapies

Localized treatment offers another novel angle. Since CTB lesions are often accessible on the skin, topical or intralesional drug delivery can achieve high local concentrations. Historically, intralesional injections of streptomycin into lupus vulgaris were attempted (circa 1951), but systemic therapy is now standard. Modern formulations include antibiotic creams, gels, or patches containing TB drugs. For example, a rifampicin-laden nanoemulsion gel showed enhanced skin penetration (Figure 4). In that study, the optimized gel increased rifampicin flux and deposition in skin by >7-fold, suggesting potential to clear local infection with lower doses and fewer systemic effects. Similarly, hydrogels or polymer films impregnated with isoniazid or clarithromycin have been explored for ulcerative TB lesions (not yet in clinical use). Table 4 compares conventional vs novel pharmacologic approaches for CTB [24]]25].

Table 4: Comparison of conventional versus novel CTB therapies. Conventional ATT requires prolonged systemic therapy, while new approaches aim for targeted delivery and reduced toxicity.

Therapy Type	Examples	Administration & Notes
Conventional ATT	Isoniazid, Rifampin, Ethambutol, Pyrazinamide	Oral or IV, multi-drug regimen for 6+ months. Highly effective for drug-susceptible CTB; systemic toxicity and long duration.

BCG Vaccination	BCG (live-attenuated M. bovis)	Provides some protection; used in infants. Not a treatment, but part of TB control. (BCG scar can rarely show lupus vulgaris.)
Local injection therapy	Streptomycin (historical)	Direct injection into lesion was tried historically; not standard now due to pain/risk.
Topical antibiotic gel	Rifampicin nanoemulsion gel	Applied to lesion; enhances skin uptake and local kill. Can reduce required dose.
Nanoparticle-mediated	Mannose-PDA NP, Lipid nanovesicles (Table 3)	Targeted delivery to macrophages; NIR-triggered release. Reduces systemic exposure.
Photothermal/Photodynamic	NIR lasers with drug carriers	Local heating or ROS kill bacilli in lesions; synergizes with antibiotics.
Host-directed therapies	E.g. Metformin, statins (investigational)	Aim to boost immune clearance of M.tb; still experimental for TB.
Surgical/Physical	Debridement of abscesses	Adjunct for cold abscesses in scrofuloderma; heals wound.

Future Directions and Conclusion

Advances in biotechnology are poised to transform CTB management. Nanotechnology offers multifunctional platforms for simultaneous diagnosis and therapy (theranostics), while potent new drugs and vaccines (e.g. recombinant BCG, subunit vaccines) may eventually reduce TB burden. Improved diagnostics like point-of-care PCR or imaging agents will aid earlier detection. Immunomodulators (e.g. TNF inhibitors' effects on TB reactivation) highlight the need to understand host factors. Pharmacists and drug developers should focus on formulating stable, skin-permeable carriers (e.g. nanogels, transdermal systems) and on ensuring safety (e.g. phototherapy skin tolerance) in clinical trials [26].

In conclusion, while standard anti-TB drugs remain the backbone of CTB therapy, novel pharmaceutical approaches are emerging to address CTB's challenges. These include targeted nanocarriers (Figure 1), advanced topical formulations (Figure 4), and combination modalities like photothermal therapy (Figure 3). They aim to achieve higher local drug levels, overcome resistant bacilli, and shorten treatment duration. Ongoing research, along with classic vigilance in diagnosis and adherence, will improve outcomes for this uncommon but debilitating disease [27][28].

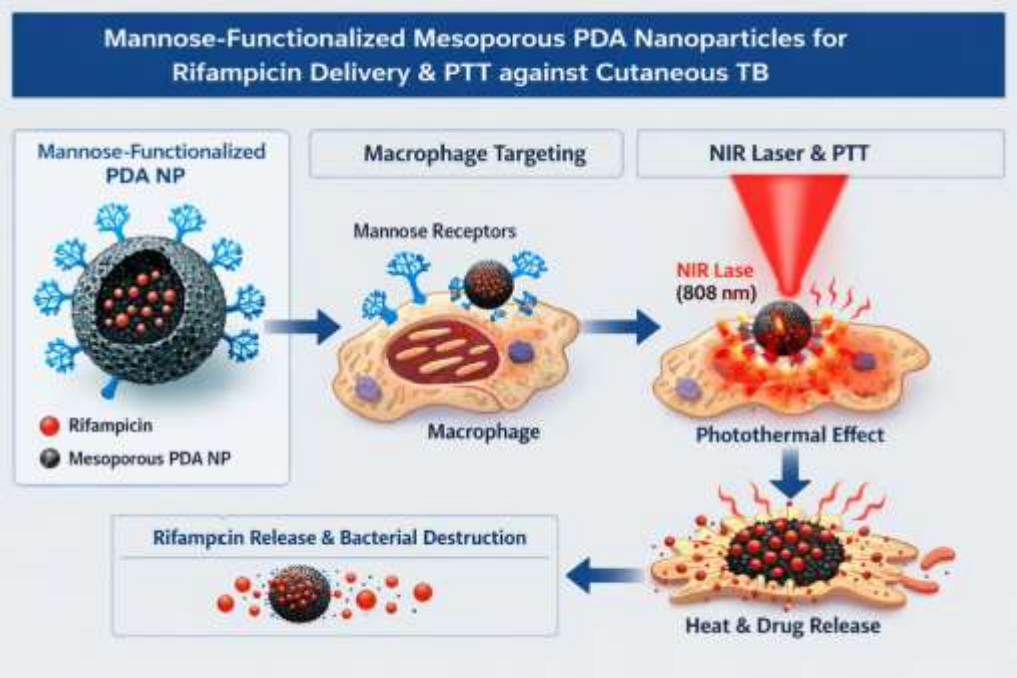

Figure 3: Graphical scheme of mannose-functionalized mesoporous polydopamine nanoparticles delivering rifampicin into macrophages for PTT against CTB. Mannose (blue trees) targets macrophage receptors; NIR laser causes heat and rapid drug release, killing intracellular M.tb [29].

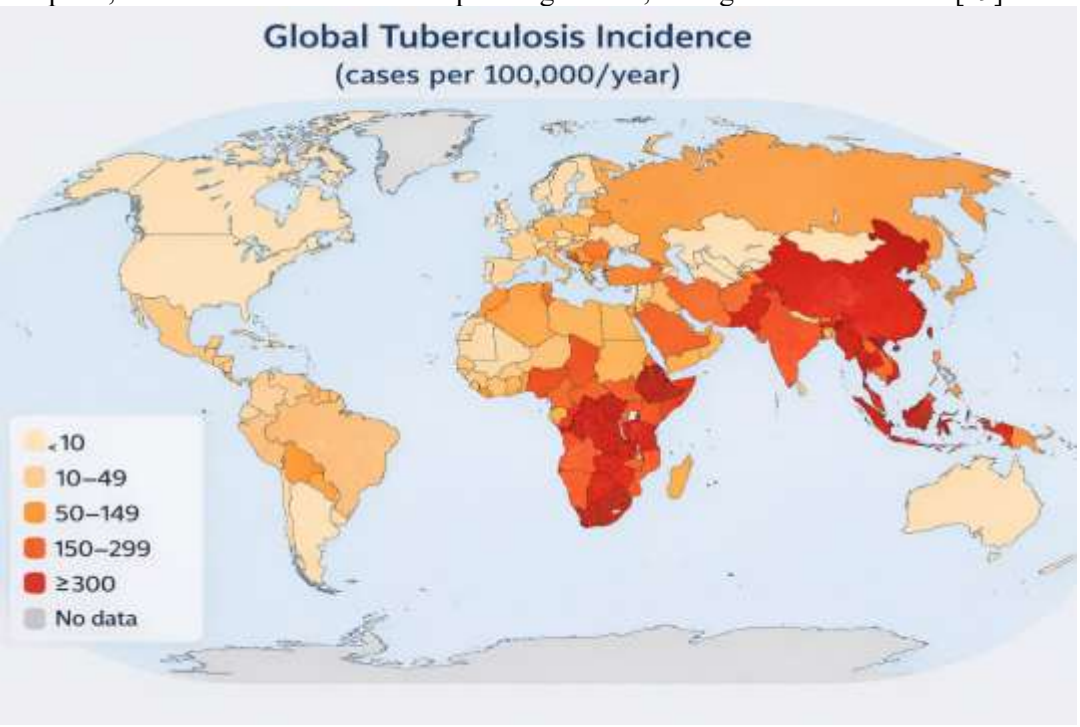

Figure 4: World map of TB incidence (cases per 100,000). CTB incidence parallels TB endemicity: highest in sub-Saharan Africa and Southeast Asia (dark red), lowest in North America and Europe (peach). (CTB rates are ~1% of overall TB in high-burden areas.) [30].

Conclusion

Cutaneous tuberculosis (CTB) remains a clinically significant but often underdiagnosed manifestation of Mycobacterium tuberculosis infection, particularly in TB-endemic regions. Although conventional anti-tubercular therapy (ATT) continues to be the gold standard and achieves high cure rates in drug-susceptible cases, challenges such as delayed diagnosis, prolonged treatment duration, systemic toxicity, and emerging multidrug resistance necessitate innovative pharmaceutical strategies.

Recent advances in nanotechnology, targeted drug delivery systems, photothermal-assisted therapy, and topical nanoformulations represent promising developments in CTB management. Macrophage-targeted nanoparticles, lipid-based nanovesicles, dendrimer systems, and nanoemulsion gels have demonstrated enhanced intracellular drug delivery, improved skin penetration, and reduced systemic exposure. These approaches not only optimize pharmacokinetic profiles but also enhance therapeutic efficacy against intracellular bacilli while minimizing adverse effects. Additionally, integration of host-directed therapies and advanced diagnostic tools strengthens the overall treatment paradigm.

Future research should prioritize translational studies, large-scale clinical trials, and cost-effective formulation development to ensure accessibility in resource-limited settings. A multidisciplinary pharmaceutical approach combining conventional chemotherapy with advanced delivery platforms holds significant potential to revolutionize CTB management, improve patient compliance, and contribute to global tuberculosis control efforts.

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