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# Drug resistance in tuberculosis: Time for new strategies.

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## Introduction

Tuberculosis (TB), caused by *Mycobacterium tuberculosis*, remains one of the deadliest infectious diseases globally. Despite being preventable and curable, TB claimed over 1.3 million lives in 2022 alone. The emergence of drug-resistant strains—particularly multidrug-resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB)—has complicated treatment and threatens to reverse decades of progress. As conventional therapies falter, it is time to rethink our strategies and embrace innovation, integration, and global collaboration. Drug resistance in TB arises when bacteria evolve mechanisms to evade the effects of anti-TB drugs. Key forms include: Resistant to at least isoniazid and rifampicin, the two most potent first-line drugs. MDR-TB with additional resistance to fluoroquinolones and at least one second-line injectable drug. MDR-TB with resistance to fluoroquinolones but not injectables [1, 2].

Resistance develops due to incomplete treatment, poor adherence, prescription errors, and transmission of resistant strains. According to WHO, nearly half a million people develop drug-resistant TB annually. Treatment success rates for MDR-TB hover around 60%, while XDR-TB outcomes are even poorer1. In many regions, especially low- and middle-income countries, access to diagnostics and second-line drugs remains limited. Genetic mutations in drug target genes (e.g., *katG*, *rpoB*, *gyrA*) [3, 4].

Efflux pumps that expel drugs from bacterial cells. Biofilm formation, which shields bacteria from antibiotics. Drug tolerance, where bacteria survive in a dormant state. Understanding these mechanisms is crucial for designing effective therapies. Traditional TB treatment regimens are lengthy, toxic, and often ineffective against resistant strains. Challenges include: Long duration (up to 20 months for XDR-TB), Severe side effects (e.g., hearing loss, liver toxicity), Low adherence due to pill burden and socioeconomic factors [5, 6]

WHO now recommends 6-month regimens like BPaLM (bedaquiline, pretomanid, linezolid, moxifloxacin) for MDR/RR-TB. These regimens improve adherence and reduce toxicity. Nanoparticles and inhalable formulations enhance drug bioavailability and target delivery to lung tissues. Controlled-release systems reduce dosing frequency and side effects. HDTs modulate the immune response to improve bacterial clearance. Examples include cytokine therapy and autophagy enhancers [7, 8].

Genomic profiling of both host and pathogen enables tailored regimens. AI-driven models predict resistance patterns and optimize therapy. Predictive analytics for resistance detection, Digital adherence tools (e.g., smart pillboxes, mobile apps), AI-assisted diagnostics using chest X-rays and genomic data. These technologies enhance early detection, treatment precision, and patient engagement. Effective TB control requires robust systems: Universal access to rapid molecular diagnostics (e.g., GeneXpert). Integrated care models combining TB, HIV, and diabetes services [9, 10].

## Conclusion

Drug-resistant TB is a formidable challenge but not an insurmountable one. By embracing shorter regimens, advanced drug delivery, host-directed therapies, and AI-powered solutions, we can outpace resistance. The time for new strategies is now. With global commitment, scientific

innovation, and patient-centered care, we can turn the tide and move closer to a TB-free world.

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