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Cracking the code: Mechanisms behind antimicrobial resistance.

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Introduction

Antibiotics revolutionized healthcare in the 20th century, drastically reducing mortality from bacterial infections. However, their widespread and often indiscriminate use in human medicine, agriculture, and veterinary care has accelerated the natural evolutionary process of resistance. Bacteria, through genetic plasticity and rapid reproduction, adapt swiftly to environmental pressures, including antibiotic exposure. Antimicrobial resistance (AMR) has emerged as one of the most formidable challenges in modern medicine. Once hailed as miracle drugs, antibiotics are now facing a crisis of efficacy as bacteria evolve to evade their effects. Understanding the molecular and biochemical mechanisms behind AMR is essential for developing effective countermeasures and safeguarding global health [1, 2].

AMR is responsible for over one million deaths annually and is projected to cause 39 million deaths between 2025 and 2050. In India, resistance to ciprofloxacin and meropenem has surged, compromising treatment for common infections like urinary tract infections and pneumonia. The World Health Organization has identified AMR as one of the top ten global public health threats. Bacteria employ several sophisticated strategies to resist antimicrobial agents. These mechanisms can be intrinsic (naturally occurring) or acquired (through mutation or gene transfer). Bacteria can alter the molecular structure of antibiotic targets, rendering the drug ineffective. For example, mutations in ribosomal RNA prevent macrolides from binding, while changes in penicillin-binding proteins (PBPs) reduce β-lactam efficacy [3, 4].

AMR is not just a medical issue it's a societal and economic crisis. It threatens the efficacy of surgeries, cancer therapies, and intensive care. Without urgent action, we risk entering a post-antibiotic era where minor infections become deadly. The battle against AMR requires global collaboration, innovation, and sustained commitment. By cracking the molecular code of resistance, we can develop smarter therapies and preserve the miracle of antibiotics for future generations. Some bacteria produce enzymes that degrade or modify antibiotics. β-lactamases, for instance, hydrolyze the β-lactam ring in penicillins and cephalosporins, neutralizing their antibacterial activity.

Efflux systems actively expel antibiotics from bacterial cells before they reach their targets. These pumps can confer multidrug resistance and are particularly prevalent in Gram-negative bacteria [5, 6].

Traditional susceptibility testing may fail to detect dynamic resistance mechanisms like gene amplification or biofilm-associated resistance9. Moreover, the antibiotic pipeline is dwindling, with few new drugs reaching the market. Innovative approaches such as antimicrobial peptides, CRISPR-Cas systems, and nanoparticle-based delivery are being explored. Alterations in membrane porins or lipid composition can limit antibiotic entry. This mechanism is common in Pseudomonas aeruginosa and other pathogens with robust outer membranes. Certain enzymes chemically modify antibiotics, such as acetyltransferases that inactivate aminoglycosides. These modifications prevent the drug from binding to its target [7, 8].

Recent studies show that bacteria can amplify existing resistance genes under antibiotic pressure. For example, E. coli exposed to piperacillin/tazobactam increased copies of the blaTEM-1 gene, overwhelming the drug's inhibitory effect. Bacteria in biofilms are up to 1,000 times more resistant to antibiotics than planktonic cells. Biofilms create a protective matrix that impedes drug penetration and fosters horizontal gene transfer. Resistance genes can spread rapidly through conjugation, transformation, or transduction. Plasmids carrying multiple resistance genes are a major driver of multidrug-resistant organisms (MDROs) [9, 10].

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