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Perspective

Mechanisms of antimicrobial resistance

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DESCRIPTION

Antimicrobial resistance (AMR) occurs when bacteria change their immune system to antimicrobial effects. Antibiotic resistance is a subset of AMR that works directly on antimicrobials. Infections caused by AMR cause millions of deaths each year. Infections caused by resistant germs are very difficult to treat, require high doses of antibiotics, or other drugs that may appear to be very toxic. These methods can also be very expensive. Bacteria with multiple antimicrobials are called multidrug resistant (MDR).

Different mechanisms adapted by microorganisms to show antibiotic resistance

Bacteria: There are five ways through which bacteria shown resistance to antibiotics.

Drug dysfunction or modiication: For example, the enzymatic activation of penicillin G in some penicillin-resistant strains by the production of β-lactamase. Drugs can also be chemically modified by the addition of active groups by transferase enzymes; for example, acetylation, phosphorylation, or adenylation are common ways of resisting aminoglycosides.

Intended- or binding local mutations: For example, PBP (Penicillin-Binding Proteins) mutations — a binding compound targeted to penicillin — in MRSA (Methicillin-Resistant Staphylococcus aureus) and other penicillin-resistant viruses. Another type of protection found among bacterial species is ribosomal protective proteins. These proteins protect the bacterial cell from antibiotics directing the ribosomes of the cell to prevent protein synthesis. This mechanism binds the binding of ribosomal protection proteins to the ribosomes of a bacterial cell, which in turn alters its conformational shape.

Metabolic modiications: For example, some sulfonamide antibodies do not require Para-Amino Benzoic Acid (PABA), an important precursor for the synthesis of folic acid and nucleic acids in sulfonamides-inhibited bacteria.

Reduced drug accumulation: By reducing the potency of the drug or by increasing the outflow (pumping out) of the drug into the cell area. These pumps inside the cell membranes of certain types of bacteria are used to pump antibiotics out of the cell before they can do any damage.

Separation and reconstruction of the ribosome: For example, the ribosome suspension of the drug containing lincomycin and erythromycin released by the heat shock protein found in Listeria monocytogenes, a homologue of HflX from other viruses.

Viruses: Certain antibiotics are used to treat some bacterial infections. These drugs prevent germs from reproducing by preventing important stages of viral replication in infected cells. Antivirals used to treat HIV, hepatitis B, hepatitis C, influenza, herpes viruses include varicella zoster virus, cytomegalovirus and Epstein-Barr virus. With each virus, some strains have become resistant to the drugs offered. Antibiotics usually target the key components of a viral replication; for example, oseltamivir targets influenza neuraminidase, whereas guanosine analogue inhibits viral DNA polymerase. Therefore, resistance to antibiotics is achieved through genetic modification that includes targeted protein.

Fungi: Fungal infections are the cause of high morbidity and mortality in people with disabilities, such as those with HIV/AIDS, tuberculosis or chemotherapy. The fungi candida, *Cryptococcus neoformans* and *Aspergillus fumigatus* cause most of these infections and antifungal resistance occurs in all of them. Resistance to many fungal drugs is increasing due to the widespread use of antimicrobials to treat infections in people who are immune.

Parasites: Protozoan viruses that cause malaria, trypanosomiasis, toxoplasmosis, cryptosporidiosis and leishmaniasis are important human germs. Drug-resistant malaria parasites are now commonplace and have led to an increase in efforts to develop new drugs. Resistance to newly developed drugs such as artemisinin has also been reported. The drug-resistant problem of malaria has fuelled efforts to develop vaccines.

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