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Opinion Article

Enzymatic modification and breakdown of antibiotic resistance in bacteria

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DESCRIPTION

Three broad mechanisms—preventing drug interaction with the target, antibiotic efflux from the cell, and direct amplification or alteration of the compound—can result in antibiotic resistance. This review examines the latter processes, which include hydrolysis, group transfer, and redox reactions, with an emphasis on the chemical method of antibiotic inactivation. While group transfer techniques like as acyltransfer, phosphorylation, glycosylation, nucleotidylation, ribosylation, and thiol transfer are among the most varied, hydrolysis is still crucial therapeutically, especially when used with -lactam antibiotics. Since these methods alone actively lower the concentration of medicines in the local environment, enzymes that physically change antibiotics have a distinctive characteristic and provide a distinctive challenge to researchers and clinicians thinking about novel ways to anti-infective therapy.

Antibiotic resistance has been noted ever since its discovery and subsequent clinical usage, which has had a detrimental effect on the management of infectious diseases. This warning letter foretold the cycle of antibiotic discovery and eventual resistance that inevitably follows the use of these compounds. In hospitals and, increasingly and alarmingly often, in the community, antibiotic resistance is now widely acknowledged to be a significant concern in the treatment of illnesses. Resistance can be either passive or active. Three main processes by which bacteria develop active drug resistance are the efflux of the antibiotic from the cell, alteration of the antibiotic target, and production of modifying enzymes that specifically target and eliminate the action of antibiotics. In order for the cell to respond to the presence of antibiotics through any of these pathways, fresh genetic programming is necessary. In numerous instances, antibiotics or their effects in fact genetically control how resistance genes are expressed.

Hydrolysis Chemical bonds in many antibiotics are hydrolytically sensitive, and biological action depends critically on their integrity. The existence of several examples of enzymes that have evolved to target and break these weak links and, as a result, provide a method of eliminating antibiotic action is not surprising. The amides that cleave the -lactam ring of the penicillin and cephalosporin medication families are among the most important of these. Although group transfer and hydrolysis make up the majority of documented enzymatic resistance mechanisms, bacteria have developed an increasing number of alternative detoxification methods for antibiotics.

Several well-studied enzymatic processes and antibiotic inactivation mechanisms have commonalities. Enzymes that are involved in primary and intermediate microbial metabolism, such as those that catalyse group transfer, hydrolysis, and redox reactions, are probably where resistance first emerged. The primary sequencing investigation of resistance proteins, which focuses on determining their molecular processes and three-dimensional structures, has, as mentioned multiple times above, discovered similarities to well-known metabolic. Strategies to overcome resistance To create strategies to finally defeat resistance, one must have a thorough understanding of the molecular structure and function of the enzymes that break down antibiotics. For instance, the discovery of resistance kinases in the 1960s allowed for the development of aminoglycosides like tobramycin and gentamicin that lacked sites of inactivation. This was made possible by an understanding of the 3′-regiospecificity of aminoglycoside resistance.

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