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Scope and Action of Aminoglycosides as Antibacterial Drugs

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Commentary

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DESCRIPTION

Aminoglycosides are a type of classic Gram-negative antibacterial drug that inhibits protein synthesis and contains an amino-modified glycoside as part of the molecule. The word can also refer to any chemical compound with amino sugar substructures. Aminoglycoside antibiotics have bactericidal efficacy against Gram-negative aerobes and some anaerobic *bacilli* when resistance has not yet developed, but not against Gram-positive or anaerobic Gram-negative bacteria. Streptomycin is the first aminoglycoside antibiotic. It is developed from Streptomyces griseus and was the first modern agent used to treat tuberculosis. Streptomycin lacks the common 2-deoxystreptamine seen in the majority of other members of this class. Originally, aminoglycosides were thought to have antibacterial activities because they inhibited bacterial protein synthesis *via* irreversible binding to the 30S bacterial ribosome. This argument, however, does not explain for these drugs' powerful bactericidal characteristics, because other antibiotics that impede protein synthesis, such as tetracycline, are not bactericidal. Recent experimental findings suggest that the outer bacterial membrane is the first site of action.

Cationic antibiotic molecules cause fissures in the outer cell membrane, resulting in intracellular leakage and increased antibiotic uptake. The fast action at the outer membrane is most likely responsible for the majority of the bactericidal activity. Aminoglycoside absorption into the bacterial cell requires energy. Anaerobes have less accessible energy for this absorption, so aminoglycosides are less active against anaerobes.

Aminoglycosides are antibiotics that work by suppressing protein synthesis. Since the isolation of streptomycin from Streptomyces griseus, the class has been a cornerstone of antibacterial chemotherapy.

Scope of aminoglycosides

Aminoglycosides are very effective against Enterobacteriaceae members such as *E. coli, Klebsiella pneumoniae* and *K. oxytoca, Enterobacter cloacae* and *E. aerogenes, Providencia spp*., *Proteus spp., Morganella spp.,* and *Serratia species*.

As with other groups of antibiotics, the spectrum of antibacterial action of aminoglycosides varies significantly. Numerous aminoglycoside-modifying enzymes that inactivate diverse antibiotics in this class play a substantial role in the variances in their in vitro activity. In general, novel aminoglycosides with broader activity spectra than older drugs like streptomycin and kanamycin include gentamicin, tobramycin, amikacin, netilmicin, isepamicin, dibekacin, and arbekacin. Gentamicin is more effective than tobramycin against *Serratia* spp., although tobramycin is more effective against *Pseudomonas aeruginosa*. According to a recent study, isepamicin has two to four times the in vitro activity of amikacin against members of the *Enterobacteriaceae* family, and both antibiotics have comparable activities against *Pseudomonas aeruginosa*.

Mechanism of action

Aminoglycosides suppress protein synthesis by binding with high affinity to the A-site on the 30S ribosomal RNA's 16S ribosomal RNA. Although members of the aminoglycoside class have varying specificities for distinct areas of the A-site, they all change its conformation. As a result of this interaction, the antibiotic promotes mistranslation by activating codon misreading during the delivery of the aminoacyl transfer RNA. This leads to error-prone protein synthesis, allowing erroneous amino acids to assemble into a polypeptide that is then released to cause harm to the cell membrane and elsewhere. Some aminoglycosides can also interfere with protein synthesis by inhibiting elongation or directly inhibiting initiation. The precise mechanism of binding and the ensuing downstream effects vary depending on the chemical structure, but all aminoglycosides are immediately bactericidal and typically have a lengthy postantibiotic impact (PAE). The PAE has been found to be closely connected to the time it takes bacteria to recover from protein synthesis inhibition. This is thought to be due to the antibiotic eventually dissociating from its target and exiting the cell.

Aminoglycoside entrance into bacterial cells is divided into three stages, the first of which enhances bacterial membrane permeability, while the second and third are energy-dependent. The polycationic aminoglycoside is electrostatically bound to the negatively charged components of the bacterial membrane, such as phospholipids and teichoic acids in Gram-positive organisms and phospholipids and lipopolysaccharide in Gram-negative organisms, and then magnesium ions are displaced. These cations are crucial for bridging and stabilising the lipid components of the bacterial membrane, and their removal results in disruption of the outer membrane, increased permeability, and the commencement of aminoglycoside uptake. This phenomenon promotes cytoplasmic entrance *via* a gradual, energy-dependent, electron-transport-mediated mechanism. When aminoglycoside molecules enter the cytoplasm, they inhibit protein synthesis and cause protein mistranslation. These mistranslated proteins insert into the cytoplasmic membrane, causing damage and facilitating future aminoglycoside entrance. This causes rapid uptake of more aminoglycoside molecules into the cytoplasm, increasing inhibition of protein synthesis, mistranslation, and cell death.