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Perspective

Drugs Involved in Inhibition of Protein Synthesis

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DESCRIPTION

Protein synthesis inhibitors are chemical compounds that stop or slow cell growth or proliferation by interrupting processes that directly lead to the formation of new proteins. Ribosomes use nanoscale protein dynamics to synthesize RNA. It is a biological machine that converts into protein. A broad interpretation of this definition can be used to describe almost any compound in terms of concentration, but in practice it usually refers to compounds that act on the translational machinery at the molecular level (either the ribosome itself or the translation factor taking advantage of key differences between prokaryotic and eukaryotic ribosome structures.

Protein synthesis inhibitors represent another large group of clinically useful antibiotics, including erythromycin, tetracyclines, chloramphenicol and aminoglycosides. They selectively interact with 70S bacterial ribosomes and spare 80S eukaryotic ribosomal particles. Protein synthesis is a complex multistep process involving many enzymes and conformational orientations. However, most of the antibiotics that block bacterial protein synthesis interfere with the processes of the 30S or 50S subunits of the 70S bacterial ribosome. Aminoacyl-tRNA synthetases, which activate each amino acid required for peptide synthesis, are not targets of antibiotics. Instead, the key steps in this targeted process are formation of the 30S initiation complex (composed of mRNA, 30S ribosomal subunits, and formyl-methionyl transfer RNA), activation of the 70S ribosome by is the formation of The 30S initiation complex and the 50S ribosome, and the elongation process that assembles amino acids into a polypeptide.

Tetracyclines, including doxycycline, prevent aminoacyl-tRNA binding by blocking the A (aminoacyl) site of the 30S ribosome. They can inhibit protein synthesis in both 70S and 80S (eukaryotic) ribosomes, but preferentially bind to bacterial ribosomes due to structural differences in their RNA subunits. In addition, tetracyclines are effective against bacteria by exploiting bacterial transport systems and increasing the concentration of antibiotics inside cells to levels significantly higher than those in the surroundings.

Aminoglycoside antibiotics have affinity for the 30S ribosomal subunit. One of the most commonly used aminoglycosides, streptomycin which interferes with the formation of the 30S initiation complex. Kanamycin and tobramycin also bind to 30S ribosomes and block formation of the larger 70S initiation complex. The macrolide erythromycin binds to the 23S rRNA component of the 50S ribosome and interferes with assembly of the 50S subunit. Erythromycin, roxithromycin, and clarithromycin all prevent elongation during the transpeptidation step of synthesis by blocking the 50S polypeptide. After a small peptide is formed, elongation is prematurely terminated, but it cannot move across the macrolide obstacle.

Peptidyltransferases are key enzymes involved in translocation, the final step in the peptide elongation cycle. Lincomycin and clindamycin are specific inhibitors of peptidyltransferases, whereas macrolides do not directly inhibit the enzyme. Hygromycin B is an aminoglycoside that specifically binds to a single site within the 30S subunit in a region that contains the A, P, and E sites of tRNA. It has been theorized that this binding distorts the ribosomal A site and may be the cause of the ability of hygromycin to induce misreading of aminoacyl-tRNAs as well as prevent the translocation of peptide elongation. In general, protein synthesis inhibitors work at different stages of prokaryotic mRNA translation into proteins like initiation, elongation (including aminoacyl tRNA entry, proofreading, peptidyl transfer, and ribosomal translocation), and termination. Linezolid acts at the initiation stage, possibly by interfering with the formation of the initiation complex, although the mechanism is not fully understood. Tetracycline and tigecycline (a glycylcycline related to tetracycline) block A-site of the ribosome, preventing binding of aminoacyl-tRNAs. The aminoglycoside interferes with the proofreading process and increases the error rate of synthesis through premature termination, among other possible mechanisms of action. Chloramphenicol blocks the peptidyl transfer step of her 50S subunit elongation of the ribosome in both bacteria and mitochondria. By targeting different stages of mRNA translation, antibiotics can be changed in the event of resistance to one or more drugs.

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