

MICROBIOLOGY

LEC. 2 ANTIBIOTICS AND ANTIBIOTICS RESISTANCE

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ANTIBIOTICS

- What are antibiotics?
- Antibiotics are medicines that fight bacterial infections in people and animals. They work by killing the bacteria (bactericidal) or by making it hard for the bacteria to grow and multiply (bacteriostatic).
- Antibiotics can be taken in different ways:
- **Orally (by mouth):** This could be pills, capsules or liquids.
- **Topically:** This might be a cream, spray or ointment that you put on your skin. It could also be eye ointment, eye drops or ear drops.
- **Intramuscular (I.M) injection or intravenously (IV):** This is usually for more serious infections.
- ❖ Range of action of antibiotics

Antibiotics fall into 3 main categories:-

- **a** – action mainly against G+ve organisms like penicillin, erythromycin & lincomycin.
- **b** – action mainly against G-ve organisms like: polymyxin & nalidixic acid.
- **c** – action against both G+ve & G-ve organisms (broad-spectrum activity) like ampicillin & ciprofloxacin.

❖ Mechanisms of action of chemotherapeutic agents

1–Inhibition of cell wall synthesis:-

such as β -lactams that include penicillins & cephalosporins interfere with cell wall synthesis and cause bacteriolysis.

2–Inhibition of cytoplasmic membrane function:- for example polymyxins.

3–Inhibition of protein synthesis:-

such as: tetracyclines, erythromycin, and aminoglycosides e.g. tobramycin & gentamycin.

4–Inhibition of nucleic acid synthesis:-

Such as : quinolone , rifampicin , novobiocin & sulfonamide .

5–Competitive inhibition:-

In which the drugs compete with an essential metabolite for the same enzyme e.g. Such as sulfonamide resemble to p – aminobenzoic acid (PABA) is an essential metabolite for DNA synthesis.

❖ Mechanisms of resistance to antimicrobial agents

1–The organism produced enzymes that destroy the drug e.g. production of β – lactamases that destroy penicillin by

Gram-positive & acetyltransferase which produced by Gram-negative bacilli destroy chloramphenicol.

2–The organism changes its permeability to the drug by modification of protein in the outer cell membranes, thus stopping its active transport into the cell e.g. resistance to polymyxins.

3–The organism develops an altered receptor site for the drug e.g. resistance to aminoglycosides is associated with alteration of a specific protein in the 30s subunit of the bacterial ribosome that serves as a binding site in susceptible organisms.

4–The organism develop an altered metabolic pathway that bypasses the reaction inhibited by the drug e.g. sulfonamide-resistant bacteria acquire the ability to use performed folic acid with no need for extracellular PABA

❖ Origin of resistance to antimicrobial agents

A–Nongenetic drug resistance:- this include

1–Metabolic inactivity:- Most antimicrobial agents act effectively only on replicating cells, non- multiplying organisms are phenotypically resistant to drugs such as M. tuberculosis which survive for years in tissues & their

resistance is due to metabolic inactivity but if they start to multiply after the suppression of cellular immunity of the patient, they become susceptible to the drugs.

2–Loss of target structure:- Protoplast or L-forms of bacteria are penicillin-resistant because they lost their cell wall which is a structural target site of the drug.

3–Bacteria may infect the host in the sites where antimicrobial are excluded or not active e.g. aminoglycosides such as gentamycin are not affected in treating enteric fever because the Salmonellae are intracellular & aminoglycosides do not enter the cells

B–Genetic drug resistance :- this include

1–Plasmid-mediated resistance :- Plasmid is an extrachromosomal genetic element associated with bacterial resistance to antibiotics, plasmids frequently carry many genes that code for the production of enzymes that inactivate or destroy the antibiotics e.g. β -lactamases which destroy the β -lactam ring in penicillin & cephalosporin, plasmids may result in epidemic resistance among bacteria by moving from one to the other by conjugation.

2-Transposon-mediated resistance:- Many transposons carry genes that code for drug resistance, as they move between plasmids & chromosomes they can transfer this property to bacteria . the process is called transposition.

3-Chromosomal drug resistance:- This develops as a result of spontaneous mutation in a gene that controls susceptibility to antimicrobial agents

❖Complications of antibacterial chemotherapy

1-Development of drug resistance

2-Drug toxicity

3-Super-infections

4-Hypersensitivity