Antibiotic resistance

Resistance is the resistance of microorganisms to the action of antibiotics.

Primary resistance

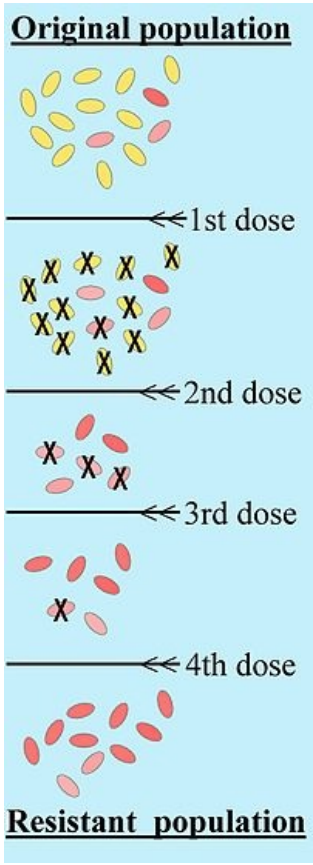
It corresponds to the genetically determined insensitivity of bacteria to a given antibiotic , regardless of previous contact (aminoglycosides in monotherapy do not affect anaerobic infections).

Secondary resistance

It occurs during therapy or **as a result of previous antibiotic administration** . In the presence of an antibiotic, resistant strains are found that are found in every large bacterial population. The rate at which secondary resistance develops depends **on the frequency of mutations** and **the number** of bacteria with a certain degree of resistance.

Secondary resistances also include those that are mediated by **plasmids** . We encounter them more often in **G−bacteria**. Genetic material can be **transferred** either from one microorganism to another by conjugation , bacteriophage transduction , or transformation , where genetic information is transferred by transposomes between plasmids or between plasmid and chromosome.

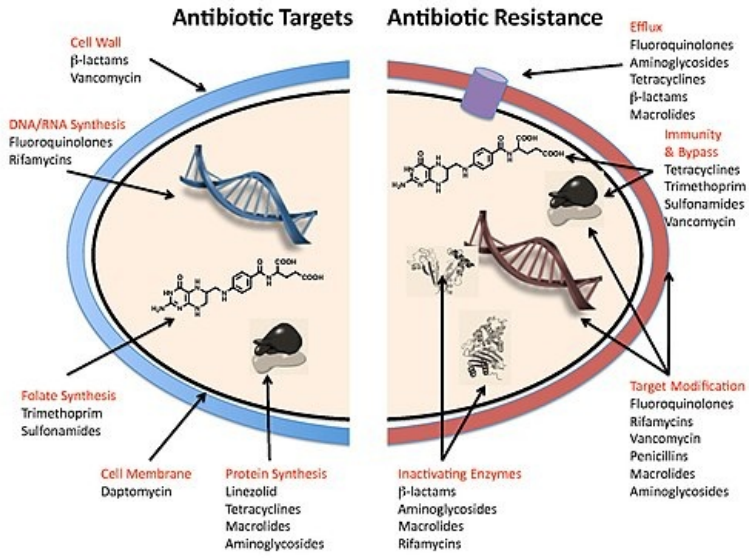
Resistance can then be divided into two basic types. **Penicillin type** *(multiple step mutation)* arises after long-term administration of some antibiotics - eg penicillin , chloramphenicol , bacitracin . **Streptomycin** type *(one step mutation)* with rapid emergence of highly resistant strains is known for streptomycin, erythromycin , lincomycin , rifampicin .



Selection of resistance strains after ATB administration

General mechanisms of resistance

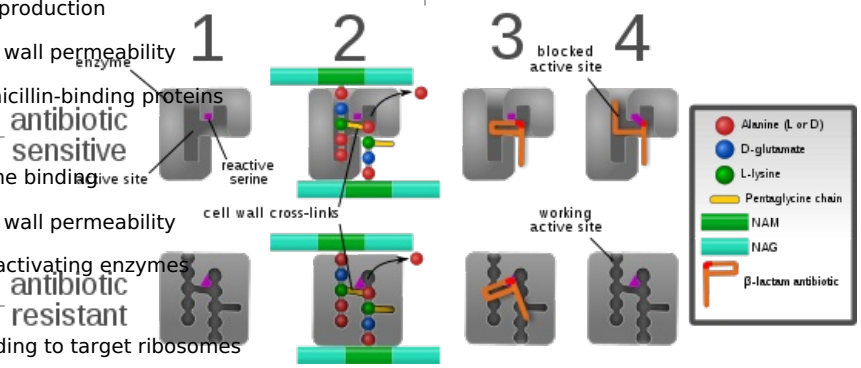
- **Limited penetration** of the antibiotic into the bacterial cell.
- **Alteration of the target structure** - receptor (eg chromosomally mediated resistance of *Haemophilus influenzae* , which conditions the change of PBP protein).
- **Metabolic changes** in the bacterial cell that prevent the antibiotic from acting on the target structures.
- **Enzymatic inhibition or inactivation** of antibiotics such as beta-lactamase .
- **Efflux pumps** - substrate-specific transport mechanisms, arise from increased expression of outer membrane proteins. They actively eliminate xenobiotics from the bacterial cell. Some are responsible for cross-resistance (beta-lactams and fluoroquinolones).



Mechanisms of antibiotic resistance

Tab. No. 3 Overview of the mechanisms of resistance to the most commonly used antibiotics

Antibiotic	Mechanisms of resistance
Beta-lactam	beta-lactamase production reduction of cell wall permeability alteration of penicillin-binding proteins
Aminoglycosides and macrolides	reduced ribosome binding reduction of cell wall permeability production of inactivating enzymes
Chloramphenicol	reduction of binding to target ribosomes reduction of cell wall permeability increase in chloramphenicol acetyltransferase activity
Tetracyclines	reduced transport to ribosomes active cellular efflux (excretion of antibiotic from the cell)
Quinolones	DNA gyrase resistance reduction of cell wall permeability active cell efflux
Sulfonamides, **Trimetoprim**	folic acid synthetase resistance dihydrofolic acid reductase resistance reduction of cell wall permeability



Mechanism of beta-lactam resistance

Cross-resistance

Current insensitivity of microorganisms to antibiotics that have a similar chemical structure and the same mechanism of action.

Bilateral cross-type resistance means that resistance to one antibiotic also means resistance to the other antibiotic (penicillin G and V or tetracyclines to each other). **A one-sided cross-type of resistance** means that susceptibility to one type can be maintained (penicillin G-resistant staphylococci may not be resistant to methycilin, but MRSA are certainly penicillin-G resistant).

Links

related articles

- Antibiotics
- Resistance of clinically important bacteria to ATB of choice
- Antibiotics in neonatology
- Bacterial resistance caused by the production of inactivating enzymes: beta lactam antibiotics, hyperproduction of beta lactamases, chromosomal, plasmid beta lactamases
- Resistance to macrolides and lincosamides (main causes of resistance, efflux)
- Beta-lactamase inhibitors

Source

- MARTÍNKOVÁ, J, S MIČUDA a J CERMANOVÁ. *Antibiotika* [online]. [cit. 2010-02-18]. <https://www.lfhk.cuni.cz/farmakol/predn/bak/kapitoly/atb-bak.doc/>.
-

Reference

- VOTAVA, Miroslav, et al. *Lékařská mikrobiologie obecná.* 2. vydání. Brno : Neptun, 2005. ISBN 80-86850-00-5.
- LINCOVÁ, Dagmar, et al. *Základní a aplikovaná farmakologie.* 1. vydání. GALÉN, 2002. ISBN 80-7262-168-8.

- MARTÍNKOVÁ, Jiřina, et al. *Farmakologie pro studenty zdravotnických oborů.* 2. vydání. Praha : Grada, 2018. ISBN 978-80-271-0929-6.
- ŠVIHOVEC, Jan, et al. *Farmakologie.* 1. vydání. Praha : Grada, 2018. ISBN 978-80-271-2150-2.

Template:Navbox - ATB