

Bacterial resistance, the global threat

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Abstract

Through years and decades the people of the world were suffering tough infections which is considered dangerous in most of times or it could be lethal in numerous times Until Alexander Fleming (The Scottish physician) came in 1922 and discovered the first Antibiotic ever which is benzyl penicillin or Penicillin G - that miracle drug originated from a culture of *Staphylococcus Aurius* - that changed the concept of treatment of bacterial infections in the scientific society forever. Since that time, scientists all over the world to develop new chemicals and Antibiotics to overcome infections whether Bacterial, viral or fungal infections. Antibiotic Resistance (AMR) is considered now the most serious obstacle facing the healthcare section and threatens the communities lives. We can say that AMR is causing more than 7,00,000 deaths worldwide annually, which is a massive number that can be avoided easily by simple steps. Antibiotic resistance are all occurs due to the misuse of drugs, missed doses, using inaccurate concentrations of drug that could be insufficient to eradicate the microorganism, using antibiotic without consultation from a physician or healthcare specialist, using concomitant medications that may interact with the antibiotic or alter its effect and pharmaceutical characteristics, giving an antibiotic in infections where the microorganism is resistant to its effect and termination of course earlier days before full control of the infection

Plasmids are stable, extra chromosomal, circular fragments of DNA that can include active genes capable of conferring resistance to the carrier bacteria. They are transmitted between different bacterial cells by conjugation and in some occasions, this can occur only between distinct species. Intrinsic resist. Is responsible for the natural lack of response of certain bacterial strains to specific antibiotics, EX: The genus *Pseudomonas* is naturally impermeable to many antibiotics. Mutations also may affect the structure of the target proteins, causing a reduction in their capacity to interact with the antibiotics due to a loss of affinity. The enzyme-mediated inactivation of drugs is another method of resistance that we face though our war against bacteria, it can be due to production of lytic enzymes or by substrate modification (acetyltransferase, phosphorylase, nucleotidase) before or after their entry into the cell. Expression may be inducible, constitutive or plasmid dependant. Beta-lactamases are enzymes that inactivate the penicillins by hydrolysis of the beta-lactam ring, which is the active moiety in their structure. This resistance is a population phenomenon and is more effective as larger numbers of individual organisms present. The beta-lactamase are constitutive in gram-negative organisms and remain in the periplasmic space, increasing individual efficacy.

Changing in the permeability to the antibiotics maybe due to a reduction in the capacity to transport the molecules through the porins of the external membrane (mutations providing resistance to

multiple substances). Porins can provide a path through the organism membrane to hydrophilic antibiotics (Due to the lipophilic nature of membranes), such as some beta-lactams, tetracyclines and fluoroquinolones. Any decrease in the ability or rate of entry of these compounds can lead to resistance. There is an abundance of reports of antibiotic resistance acquired through loss or functional changes of porins in a large number of organisms such as, *E. coli*, *P. aeruginosa*, *Neisseria gonorrhoeae*, *Enterobacter aerogenes* and *Klebsiella pneumonia*

Change in the permeability to the antibiotics may be due to a reduction in the capacity to transport the molecules through the porins of the external membrane (mutations providing resistance to multiple substances) TO the quantity of LPS reducing the absorption of hydrophobic molecules, to changes in the membrane transport protein (mutations reducing the affinity of the transporters) which may be a channel, electrochemical potential-driven transporter, primary active transporter or electron carrier. One more important way of resistance in some strains of bacteria (ex: *S. aureus*) is the expression of pumps which is responsible for the transport of such toxic compounds as drugs, toxins throughout the cell membrane and actively pump the drug out of the cell. Bacteria pumps out the antibiotics from their cellular interior to the external environment using these special transporter proteins called efflux pumps. Inhibiting these pumps seems to be an attractive strategy at a time when novel antibiotic supplies are dwindling. Examples of antibiotics susceptible to the efflux are Quinolones and Tetracyclines. Changes in the molecular targets are usually caused by point mutations that alter the

affinity between the two molecules. Although there is no increase in the rate of mutations at particular sites, there is a very large selective pressure for clonal selection in the presence of antibiotics. The target molecules may be on the plasma membrane, such as penicillin-binding protein (methicillin-resistant staphylococci produce an additional chromosomal PBP with a heterogeneous expression and a lower affinity for methicillin), or intracellular, such as DNA-gyrase (quinolones), metabolic enzymes such as Dihydropteroate reductase (sulfonamides), and changes in the proteins of the 30s ribosomal subunit (Aminoglycosides) or 50s subunit (macrolides).

Biography

Mohamed Sabri Youssef is a clinical experienced pharmacist with over 15 years of experience in the medical field, with interest in microbiology and its impact in our lives, he served as a clinical pharmacist in many hospitals and pharmacies in Egypt, Saudi Arabia and Kuwait.



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