Antimicrobial Resistance Mechanisms

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Antimicrobial Resistance Mechanisms

According to Mutuku et al. (2022), antimicrobial resistance is a critical health challenge that can cause the ineffectiveness of standard treatments. Antimicrobial resistance has increasingly become a concern worldwide, as it is one of the main causes of mortality and morbidity. Antimicrobial resistance leads to increased infections, which quickly spreading to other victims. The four main mechanisms of microbial resistance are discussed below.

Enzymatic Degradation or Modification of Antimicrobial Drugs

This is one of the mechanisms bacteria use to resist antimicrobial drugs. In this case, Abushaheen et al. (2020) reported that bacterial enzymes may chemically modify the antibiotics or degrade them. This eventually makes the antibiotics ineffective in fighting the bacteria. A good example of antibacterial enzymes produced for this purpose are beta-lactamases, which degrade cephalosporins, and penicillin beta-lactam rings. Because of this mechanism, the enzymes eventually neutralize the antibiotics, making them ineffective in the body. Aminoglycoside-modifying enzymes (AMEs) are also crucial examples of enzymes that work by adding several chemical groups to the aminoglycoside class of antibiotics. This process impedes the drugs' ability to stop protein synthesis by binding to the bacterial ribosomes.

Altering the Targets Site

In this mechanism, microbes modify the structures to which antibiotics bind to enhance resistance. According to Moo et al. (2020), the work of antibiotics is to target various bacterial elements, such as ribosomal subunits and enzymes that enhance the production of proteins. If microbes such as bacteria change these targets, the result is that the antibiotics would not bind to them effectively, making them ineffective. For example, methicillin-resistant *Staphylococcus aureus* (MRSA) is known to alter penicillin-binding proteins (PBPs), thereby preventing cell wall synthesis inhabitation. On the same note, some

microbes are known to enhance 235 ribosomal RNA methylation, which negatively impacts antibiotics' efficacy.

Efflux Pumps

According to Moo et al. (2020), bacteria use this mechanism to expel various toxic elements from their cells, thereby reducing their intracellular concentration with antibiotics. The bacteria's cell membranes contain these pumps, which are also protein complexes. Their function is to transport various unwanted substances, such as antibiotics. Moo et al. (2020) further explained that bacteria can have specific efflux pumps for certain drugs or handle different classes and types. Escherichia coli's AcrAB-TolC efflux pump, for instance, can eject several drug substances, such as chloramphenicol, from the cell. *Pseudomonas aeruginosa's* MexAB-OprM pump can also pump out antibiotics such as quinolones from the cell. Mutuku et al. (2022) outlined that the location of the genes that encode the pumps facilitates horizontal gene transfer, thereby increasing their resistance rate.

Reduced Permeability

Sometimes, bacteria membranes and walls may prevent antimicrobial agents' entry. The permeability changes of the barriers preventing the agents' entry can lead to antimicrobial resistance. For instance, Gram-negative bacteria's outer membrane prevents the entry of several antibiotics. Abushaheen et al. (2022) further explained that mutations in porin channels can reduce the permeability of aminoglycosides and other antibiotics. Moreover, studies have found that *Pseudomonas aeruginosa* exhibits porin expression changes, which reduce antibiotics' permeability.

Conclusion

As this work outlines, microbes use diverse mechanisms to resist antibiotics. The four mechanisms described in this work are target site alteration, enzymatic degradation, reduced permeability, and efflux pumps. As Abushaheen et al. (2022) noted, addressing antimicrobial

resistance is critical in developing strategies to prevent the impacts and spread of antimicrobial resistance, consequently reducing morbidity and mortality.

References

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