International Scholars Journals

Advanced Journal of Microbiology Research ISSN 2736-1756, Vol. 17 (1), pp. 001-002, January, 2023. Available online at www.internationalscholarsjournals.org © International Scholars Journals

Commentary

Transformation of resistant genes from bacteria by antimicrobial agents

Stefan Roberts*

Department of Microbiology, Freie Universität Berlin, Berlin, Germany.

Received: 02-Mar-2023, Manuscript No. AJMR-23-89722; Editor assigned: 06-Mar-2023, PreQC No. AJMR-23-89722 (PQ); Reviewed: 21-Mar-2023, QC No. AJMR-23-89722; Revised: 28-Mar-2023, Manuscript No. AJMR-23-89722 (R); Published: 04-Apr-2023

DESCRIPTION

Bacteria of animal origin have been found to be resistant to almost all antimicrobial treatments in recent years. The mechanisms that have been discovered thus far for the major kinds of antimicrobial drugs are described in depth in microbiology (Schwarz S et al., 2001). The primary processes include alterations at the cellular target sites, decreased intracellular accumulation by either decreased influx or greater efflux of antimicrobial drugs, and enzymatic inactivation by disintegration or chemical modification of antimicrobial agents (i.e., mutational changes, chemical modification, protection, or even replacement of the target sites). To increase bacterial resistance to antimicrobial drugs, many processes frequently interact.

Since there were few resistant strains and several novel, highly powerful antimicrobial drugs of various classes had been discovered, antimicrobial resistance was not viewed as a serious issue in the early days of antimicrobial chemotherapy (Datta N et al., 1972). These early antimicrobial substances were by-products of soil bacteria's metabolic processes and gave their producers a competitive edge while competing for resources and ecological niches. As a result, it is safe to believe that bacterial resistance to antimicrobial medicines originated long before these drugs were used in clinical settings (Livermore DM., 1995).

Since the 1950s, the widespread use of antimicrobial medicines has significantly increased the selective pressure on bacteria, which has hastened the emergence and dissemination of bacterial resistance to these drugs. The majority of the time, the first resistant target microorganisms appeared three to five years after an antimicrobial treatment was first used in a clinical setting. This is especially true for broad-spectrum antibiotics that have many uses in human and veterinary medicine, horticulture, and/or aquaculture, such as tetracyclines, aminoglycosides, macrolides, and –lactams (Paulsen IT et al., 1996).

Resistance to antimicrobial agents

There are two primary categories of resistance to antimicrobial agents: acquired resistance and innate resistance. Primary or innate resistance, another name for intrinsic resistance, refers to a condition in which bacteria are generally resistant to a particular antimicrobial agent or set of agents (Ambler RP., 1980). This is frequently caused by the absence or accessibility of certain antimicrobial agents' target structures, such as resistance to -lactam antibiotics and glycopeptides in bacteria that lack cell walls, such as Mycoplasma spp., or vancomycin resistance in Gram-negative bacteria because vancomycin cannot pass through the outer membrane. There are three basic types of resistance mechanisms that can be distinguished: (i) enzymatic inactivation through chemical or physical modification of the antimicrobials, (ii) reduced intracellular accumulation through decreased influx and/or increased efflux of antimicrobials, and (iii) modification of the cellular target sites through mutation, chemical modification, or target site protection, but also overexpression of sensitive targets or the replacement of sensitised targets (Ehlert K., 1999).

Antimicrobial resistance is developed by a variety of methods, including mutations, the creation of novel resistance genes, and the transfer of resistance genes from other bacteria. Different bacteria have been shown to contain a large number of resistance genes that define distinct resistance strategies. Regarding the specific bacteria involved, the selective pressure caused by the use of antimicrobial drugs, and the availability and transferability of resistance genes in the gene pools available to the bacteria, resistance development progresses at varying rates. These fundamental truths apply to both human- and animal-derived germs developing resistance.

*Corresponding author. Stefan Roberts, E-mail: Roberts@fuberline.de

In order to maintain the effectiveness of antimicrobial agents for the control of bacterial infections in animals, prudent use of antimicrobial agents is strongly advised in both human and veterinary medicine, but particularly in food animal production. This is because of every antimicrobial substance select for bacteria that are resistant to it.

REFERENCES

1. Schwarz S, Chaslus-Dancla E (2001). Use of antimicrobials in veterinary medicine and mechanisms of resistance. Vet Res. 32: 201–225.
2. Datta N, Hedges RW (1972). Trimethoprim resistance conferred by W plasmids in Enterobacteriaceae. J Gen Microbiol. 72: 349–355.
3. Livermore DM (1995). Beta-Lactamases in laboratory and clinical resistance. Clin Microbiol Rev. 8: 557–584.
4. Paulsen IT, Brown MH, Skurray RA (1996). Proton-dependent multidrug efflux systems. Microbiol Rev. 60: 575–608.
5. Ambler RP (1980). The structure of β-lactamases. Philos Trans R Soc Lond B Biol Sci. 289: 321–331.
6. Ehlert K (1999). Methicillin-resistance in Staphylococcus aureus: Molecular basis, novel targets and antibiotic therapy. Curr Pharm Des. 5: 45–55.