EDITORIAL

Overview of multidrug resistant bacteria

Veena Priyadarshini S

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The emergence of pathogenic organisms resistant to antibiotics is one of the greatest concerns facing the health system today. Multidrug-resistant (MDR) bacterium infections are becoming more widespread and pose a severe threat to public health. Originally, these species were only found in hospitals, but now they can be found elsewhere. *Staphylococcus aureus*, *Enterococcus faecium*, *Enterococcus faecalis*, and *Streptococcus pneumoniae* are the most common gram-positive bacteria. *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* have been the most common gram-negative strains. Antimicrobial resistance is genetically determined in these strains and is most typically transmitted through horizontal gene transfer of extra-chromosomal genetic elements. Antibiotic resistance mechanisms include low permeability of the outer membrane in gram-negative bacteria, efflux pumps, the creation of degrading enzymes, and the alteration of targets. The causes primarily responsible for the spread of antibiotic resistance species include globalization, excessive use of antibiotics in animal husbandry and aquaculture, the use of numerous broad-spectrum drugs, and a lack of adequate antimicrobial stewardship.

Antibiotic-resistant diseases are becoming more common, which means there are fewer antimicrobial medicines available to treat illnesses caused by these bacteria. If no new antibiotics are produced or found by 2050, it is estimated that there would be no effective antibiotic available to treat infections. This necessitates the quest for alternative antibiotic-resistant disease control approaches, and numerous research groups around the world are actively exploring for such answers. In bacteria, multidrug resistance is caused by the accumulation of genes, each of which codes for resistance to a specific agent, on resistance (R) plasmids or transposons, and/or the action of multidrug efflux pumps, each of which may pump out more than one drug type. It is because the Target Protein has undergone mutational changes. The enzymatic pathways discussed below are unlikely to inactivate man-made substances like fluoroquinolones. Bacteria can still develop resistance to the chemical through mutations that make the target protein less susceptible to it. Fluoroquinolone resistance is caused by mutations in the target enzymes, DNA topoisomerases, although not solely. The manner of action of the drug determines whether this sort of resistance can be easily spread to other cells via plasmids.

Infections acquired during medical treatment increase morbidity and mortality rates around the world. Antimicrobial resistance is closely linked to an increase in mortality, making antibiotic therapy more restrictive and making it more difficult to treat infections caused by MR bacteria. Infections with carbapenem-resistant gram-negative bacilli, primarily Enterobacteria, were a major public health concern at the turn of the century. MDR gram-negative bacteria, such as *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, ESBL-producing Enterobacteria, and carbapenem-resistant Enterobacteria (CRE), are thought to be the predominant cause of nosocomial infections. The most frequent bacterial infections have recently been reported to be methicillin-resistant *S. aureus* (MRSA) and vancomycin-resistant *Enterococcus* (VRE), and hospitals have also been isolated from foods of animal origin, water, and animals. Clinical isolates of MDR *P. aeruginosa,* Carbapenem-resistant *Enterobacteriaceae*, and *A. baumannii* have been the most common, although certain strains have also been found in foods, animals, and water.

Methicillin-resistant *S. aureus* is a gram-positive bacterium that produces multiple virulence factors that aid disease causation and aid in the rapid development of antimicrobial resistance against antimicrobial medications used to control it, a trait that elevates the pathogen's importance. Penicillin was initially effective in treating *S. aureus* infections. *S. aureus,* on the other hand, gained a plasmid-encoded beta-lactamase that conferred penicillin resistance in the 1940s, shortly after it was introduced for clinical use. For many *S. aureus* infections, vancomycin (VAM) and daptomycin (DAP) are often the medicines of last resort. MRSA strains, on the other hand, have developed an MDR phenotype, and the most concerning issue is the emergence of bacteria that are resistant to or have reduced tolerance to these antibiotics. Vancomycin-intermediate *S. aureus* (VISA), heterogeneous VISA (hVISA), and high-level vancomycin-resistant *S. aureus* are three types of vancomycin-resistant *S. aureus* that differ in vancomycin susceptibilities (VRSA).

Enzymatic drug inactivation is a common resistance mechanism for antibiotics of natural origin, such as aminoglycosides (kanamycin, tobramycin, and amikacin), which are inactivated by enzymatic phosphorylation [by aminoglycoside phosphoryl transferase (APH)], acetylation [by aminoglycoside acetyltransferase (AAC)], or adenylation (by amino as extra genetic components on plasmids, genes coding for these inactivating enzymes can easily produce resistance. Finally, harmful bacteria can persist in an antibiotic-treated patient if they develop a physiologically resistant condition without undergoing genetic alterations.

School of Life Sciences, B.S. Abdur Rahaman Crescent Institute of Science and Technology, Chennai, Tamil Nadu, India

Correspondence: Veena Priyadarshini S, School of Life Sciences, B.S. Abdur Rahaman Crescent Institute of Science and Technology, Chennai, Tamil Nadu, India. Telephone: (+91) 9500084421, E-mail: veenapriya31@gmail.com

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