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ANTIMICROBIAL RESISTANCE: A COMPREHENSIVE REVIEW OF MECHANISMS, BURDEN, AND CONTROL APPROACHES

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ABSTRACT

Antimicrobial resistance (AMR) is a critical and growing global health threat that compromises the effective treatment of infections caused by bacteria, viruses, fungi, and parasites. It arises from the overuse and misuse of antimicrobial agents in human medicine, veterinary practice, and agriculture, as well as from inadequate infection control, poor sanitation, and global travel. The spread of resistance is facilitated by both vertical and horizontal gene transfer, making once-treatable infections increasingly difficult or impossible to cure. Despite the urgent need, the development of new antimicrobial drugs remains slow, and resistance continues to rise, particularly among high-priority pathogens such as multidrug-resistant Klebsiella pneumoniae, Pseudomonas aeruginosa, and Candida auris. A One Health approach addresses human, animal, and environmental health in an integrated way which is essential to slow the spread of AMR. Solutions must include better diagnostics, antimicrobial stewardship, public awareness, and international collaboration to preserve the effectiveness of existing treatments and to support the development of new therapeutic strategies.

KEYWORDS: Antimicrobial resistance, multidrug resistance, One Health, horizontal gene transfer, antibiotic stewardship, drug-resistant pathogens, diagnostic innovation, global health.

INTRODUCTION

Antimicrobial resistance (AMR) has become one of the most serious global health threats of the 21st century.[1] It arises when bacteria, viruses, fungi, or parasites adapt in ways that reduce or eliminate the effectiveness of treatments, making infections increasingly difficult to manage. This alarming trend puts millions of lives at risk, with projections estimating up to 10 million deaths annually by the year 2050 if urgent action is not taken.[2][3]

A major factor contributing to AMR is the widespread misuse and overuse of antibiotics in both human and veterinary medicine.[4] Antibiotics are often taken without proper medical guidance, prescribed unnecessarily, or used to treat illnesses like the common cold, which do not require such treatment. Many people stop taking antibiotics once they feel better, rather than completing the prescribed course, allowing surviving microbes to build resistance.[5] Other practices, such as sharing leftover antibiotics or using substandard or counterfeit medications, also play a significant role in the development of resistance. In addition, poor sanitation and hygiene in many communities lead to the spread of infections, which in turn results in increased antibiotic use and higher chances of resistance developing.[6]

The discovery of new antibiotics has not kept pace with the rapid emergence of resistant microbes. As a result, the world now faces what has been described as a post-antibiotic era, where even minor injuries and common surgical procedures could once again become life-threatening.[7] To effectively combat AMR, global health agencies have endorsed the One Health approach. This strategy recognises that human, animal, and environmental health are closely connected and that coordinated action across these sectors is essential. Resistance genes can be exchanged between bacteria found in humans and animals, highlighting the need for integrated surveillance and control measures.[8]

Although AMR is a global issue, its effects are especially pronounced in low- and middle-income countries. In these regions, infectious diseases remain a leading cause of death, and the healthcare infrastructure often struggles to cope with the rising burden of drug-resistant infections. In India, for example, resistance rates are increasing in hospitals, while data from community settings remain limited. Challenges such as regional disparities, insufficient research capacity, and lack of consistent treatment guidelines make it difficult to contain the spread of resistance.[9]

AMR is most commonly seen in bacteria and fungi, but resistance is also emerging in viruses and parasites. These forms of resistance are referred to as antibacterial resistance, antifungal resistance, antiviral resistance, and anthelmintic resistance, respectively.[10]

Despite growing political attention at international forums, including the G7, public awareness of AMR remains limited. Increasing health literacy, strengthening healthcare systems, and promoting responsible antibiotic use are essential steps toward controlling this crisis. Addressing AMR requires a united global effort, with individuals, communities, and governments working together to preserve the effectiveness of life-saving antimicrobial treatments for the future.

Basic anatomy of bacterial cell

Bacterial cells are structurally simple but functionally efficient. Unlike eukaryotic cells, they lack all membrane-bound organelles such as mitochondria, lysosomes, Golgi apparatus, endoplasmic reticulum, chloroplasts, peroxisomes, glyoxysomes, and true vacuoles. Additionally, they do not possess a true membrane-bound nucleus or nucleolus. Instead, their genetic material is located in a region known as the **nucleoid**, which lacks a nuclear membrane, nucleoplasm, and nucleolus. This nucleoid contains naked DNA that is not associated with histone proteins.[11][12]

The cell wall of bacteria plays a vital role in maintaining the shape and rigidity of the cell. It is primarily composed of peptidoglycan, a complex polymer of sugars and amino acids. The sugar units include N-acetylglucosamine (NAG) and N-acetylmuramic acid (NAM), which are cross-linked by short peptide chains. In some bacterial species, peptidoglycan may constitute up to 90% of the cell wall, while in others, it may account for as little as 10%.[13]

Based on differences in cell wall composition, bacteria are classified into Gram-positive and Gram-negative types.

- **Gram-positive bacteria** possess a thick peptidoglycan layer and contain teichoic acid, which serves as a major surface antigen. These bacteria have only a single plasma membrane beneath the cell wall.[14]
- **Gram-negative bacteria** have a relatively thin layer of peptidoglycan, which is surrounded by an additional outer membrane. This outer membrane contains lipopolysaccharide (LPS), which is composed of Lipid A (a toxic component), a core polysaccharide, and an O-antigen. The presence of this outer membrane provides structural complexity and contributes to the resistance of Gram-negative bacteria to certain antibiotics.[15][12]

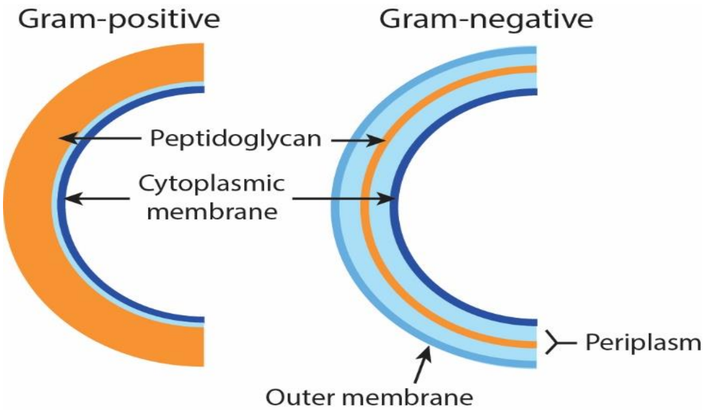

Fig. 1. Schematic illustration of the structure of Gram-positive and Gram-negative bacterial cell walls. (https://commons.wikimedia.org/wiki/File:Bacterial_cell_walls.jpg)

The plasma membrane (also known as the cell membrane) lies beneath the cell wall and is composed of phospholipids and proteins. It plays a crucial role in regulating the selective transport of molecules such as sugars, amino acids, and other nutrients. The nucleoid, considered the bacterial equivalent of a nucleus, functions as the control center of the cell. It governs all cellular activities and stores hereditary information. Unlike eukaryotic nuclei, it is not enclosed by a nuclear envelope and lacks internal structures such as the nucleolus and nucleoplasm.[11]

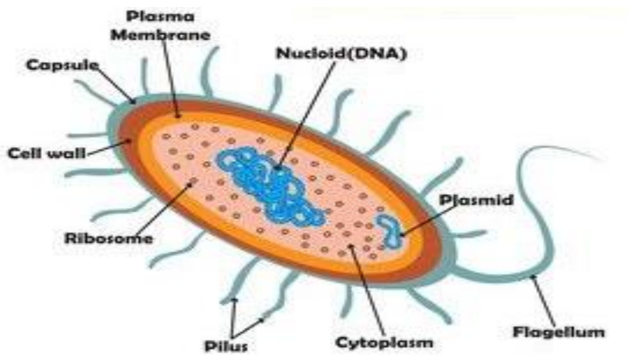

Fig. 2: Bacterial cell structure. (https://i.pinimg.com/originals/e7/a0/a2/e7a0a2de8670feed276f650549000689.jpg)

Ribosomes in bacterial cells are of the 70S type, which are smaller than the 80S ribosomes found in eukaryotic cells. Bacterial ribosomes are universal organelles composed of RNA and proteins, and their primary function is to synthesise proteins essential for cellular structure and metabolic activity.[11] Structurally, they consist of two subunits: the 30S subunit, which decodes messenger RNA (mRNA), and the 50S subunit, which catalyzes peptide bond formation. Together, these form the functional 70S ribosome.[16][12]

Antimicrobial Drugs

Antimicrobial drugs can be grouped according to the microorganisms they act primarily against. For example, antibiotics are used against bacteria, and antifungals are used against fungi. Common antibacterial drugs can be grouped by their mode of action. Examples of modes of action include inhibiting cell wall biosynthesis, inhibiting biosynthesis of proteins, and inhibiting nucleic acid synthesis.

Table 1: Common antibacterial Drugs (Mode of Action).

Mode of Action	Target	Drug Category
Inhibit cell wall synthesis	Penicillin-binding proteins	β-lactams: Penicillin, cephalosporins, Monobactams, Carbapenems
	Peptidoglycan subunits	Glycopeptides
	Peptidoglycan-subunit transport	Bacitracin
Inhibit protein synthesis	30s ribosomal subunit	Aminoglycosides, Tetracyclines
	50s ribosomal subunit	Macrolides, Lacosamide, Chloramphenicol, Oxazolidinones
Interrupt membrane	Lipopolysaccharide, inner and outer membranes	Polymyxin B, Colistin, Daptomycin
Inhibit nucleic acid synthesis	RNA	Rifampicin
	DNA	Fluoroquinolones
Antimetabolites	Folate synthetase	Sulfonamides, Trimethoprim
	Mycolate synthetase	Iso-nicotinic acid hydrazide
Mycobacterial adenosine triphosphate (ATP) synthase inhibitor	Mycobacterial ATP synthetase	Diarylquinoline

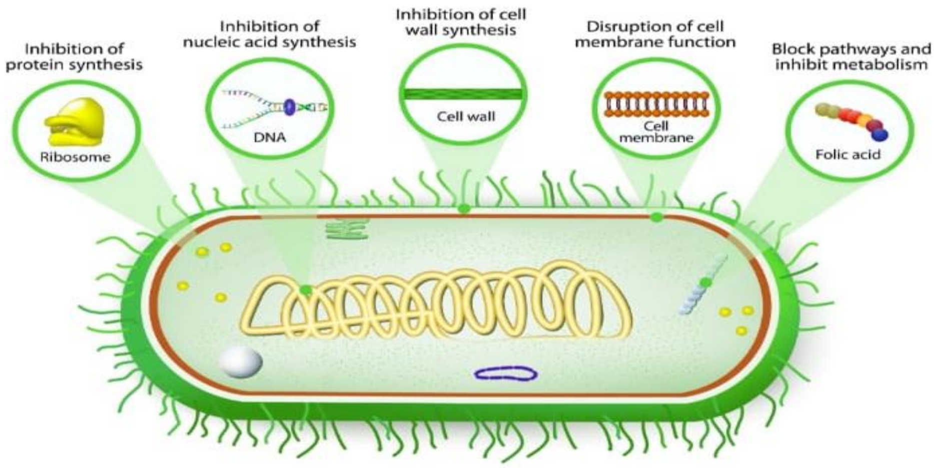

Fig. 3: Mechanism of action of Antibiotic Drugs.

Mechanisms of AMR

The restricted uptake of β-lactam antibiotics is a resistance mechanism that occurs when changes in porin channels within the bacterial cell membrane limit the entry of the drug. In some cases, bacteria also use efflux pumps to actively expel β-lactam antibiotics from the periplasmic space, reducing their effectiveness. This reduced uptake mechanism is frequently seen in Pseudomonas aeruginosa, Acinetobacter baumannii, and members of the Enterobacteriaceae family. For instance, P. aeruginosa can resist antibiotics through mutations or alterations in the OprD2 porin, which impairs drug entry into the cell.[17]

Target mutations and modification are key mechanisms by which bacteria develop resistance to antimicrobial drugs. These resistant strains acquire spontaneous mutations in specific genes, which significantly reduce the drug's ability to bind to or act on its target. In many cases, these genetic changes allow the bacterial cell to maintain internal balance (homeostasis), but only in the continued presence of the antibiotic. Overall, resistance due to mutation often arises through one or more of the following processes: (i) decreased uptake of the drug, (ii) activation of efflux pumps that expel the drug, and (iii) alterations in metabolic pathways that bypass the drug's effects.[17]

Typical resistance mechanisms in bacteria include the enzymatic modification or degradation of antibiotics, which renders them ineffective. Bacteria may also limit the uptake of antibiotics to prevent their accumulation within the cell. Additionally, modifications to antibiotic target sites, such as ribosomes, can reduce drug binding and efficacy. Alterations in metabolic pathways allow bacteria to bypass the antibiotic's effects, while increased activity of efflux pumps helps expel antibiotics from the cell before they reach therapeutic levels. These strategies collectively contribute to reduced antibiotic susceptibility and the persistence of resistant infections.[18]

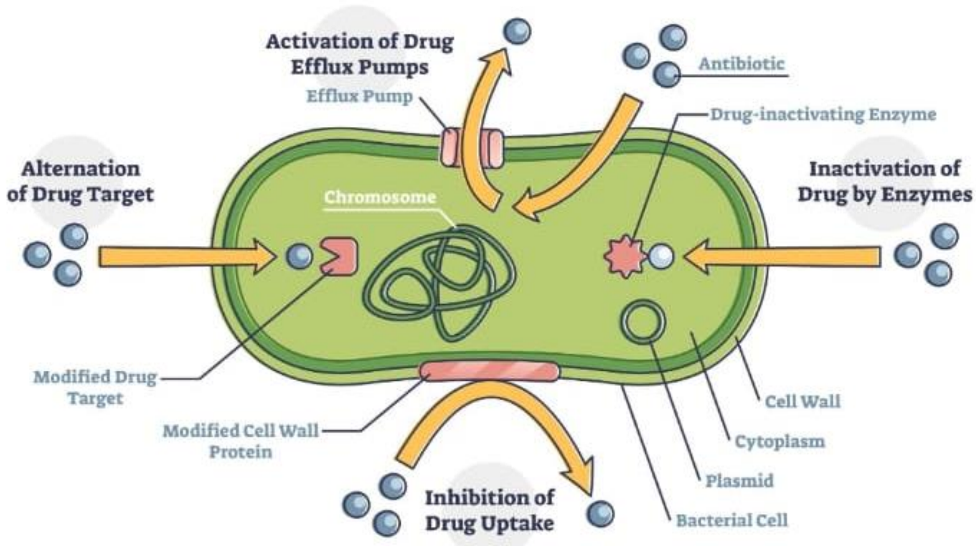

Fig. 4: Mechanism of antimicrobial resistance in bacteria.

Bacteria develop strong resistance to antibiotics when multiple defense strategies work together. This resistance can spread through two key processes. The first is vertical gene transfer, where resistance traits are inherited from parent bacteria by their offspring. The second, more rapid method is horizontal gene transfer, where the bacteria exchange resistance genes with other bacteria, even those from different species. This sharing of genetic material allows resistance to spread quickly, making it increasingly difficult to treat infections effectively.[8]

Transmission of Antibacterial Resistance

AMR spreads through the transfer of genetic material. This transfer can be 'vertical,' when antibiotic-resistant genes are passed down to new generations, or 'horizontal,' when bacteria (and sometimes viruses) share and exchange fragments of genetic material with each other.[19]

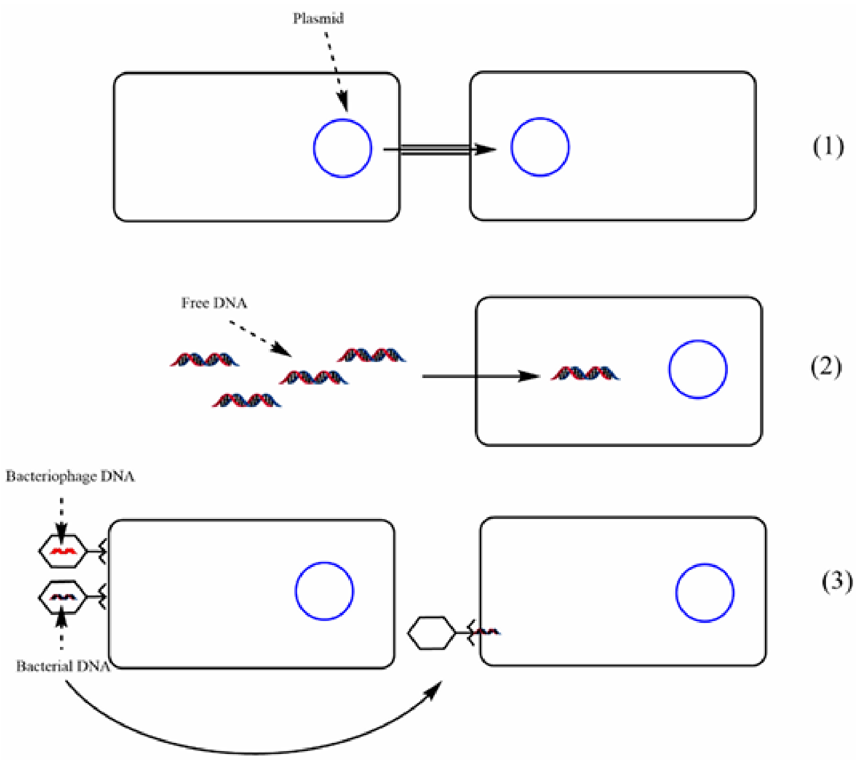

Fig. 5: Genetic transfer in AMR- (1) conjugation, transfer of genes from one bacterial cell to another that requires cell-to-cell contact, (2) transformation-uptake of free DNA from the environment, (3) transfer of plasmid genes from one cell to another by viruses, (4) Gene transfer agents like bacteriophages, which are released upon cell lysis.

AMR in Specific Pathogens

Anthelmintic resistance (AR) in cattle parasites is becoming a serious global issue, with recent cases in northern Germany showing resistance to both macrocyclic lactones and benzimidazoles. The fecal egg count reduction test (FECRT) is commonly used to detect resistance, but results can vary depending on the method and analysis tools like Egg Counts and Bayes Counts. Genetic testing (nemabiome) found Ostertagia ostertagi and Cooperia oncophora to be the most common parasites, but the unexpected variety across farms suggests the problem may be bigger than it seems. Finding resistance to multiple drugs on some farms is a warning sign that it shows the need for smarter, more targeted treatments, combining drugs, and better teamwork between farmers, vets, and researchers to protect cattle health.[20]

The study looked at 208,233 Salmonella genomes from 148 countries between 1900 and 2023 to understand global patterns of antimicrobial resistance (AMR) and what causes it. AMR levels changed depending on the type of Salmonella, the source, and the location. Resistance increased in samples from chickens, food, wild animals, and the environment, but decreased in those from cattle, pigs, and turkeys. A special type of S. Typhimurium was found in the United States earlier than in China. The main factors linked to AMR were antibiotic use, farming, climate, city growth, healthcare, and living conditions. This study also created a global map of Salmonella genes to help track how AMR spreads and changes over time.[21]

Global Epidemiology and Trends

Antibacterial drug development remains slow and insufficient, with only twelve new agents approved between 2017 and 2021, most offering limited innovation and targeting existing resistance mechanisms. The pipeline lacks effective options for critical Gram-negative pathogens like carbapenem-resistant A. baumannii and P. aeruginosa, as well as pediatric and oral treatments. Nontraditional approaches show promise but face regulatory and funding challenges. High attrition in preclinical development and limited investment further hinder progress. COVID-19 has delayed programs and diverted resources. Urgent global action, funding, and innovation are needed to address the growing threat of antimicrobial resistance.[22]

The COVID-19 pandemic led to an increase in antibiotic use in hospitals, even though studies showed that bacterial co-infections at admission were rare. Using antibiotics when they are not needed provides no benefit and can cause harm, including allergic reactions, organ toxicity, and Clostridioides difficile infections. About one in five hospitalized patients who receive antibiotics experience an adverse drug event. These findings highlight the importance of improving how antibiotics are used and ensuring they are only given when truly necessary, in both humans and animals.[23]

Although bacterial coinfections in COVID-19 patients were less than 10 percent, approximately 75 percent received antibiotics, potentially contributing to antimicrobial resistance. This study examined monthly antibiotic sales data for cephalosporins, penicillins, macrolides, and tetracyclines from 71 countries between March 2020 and May 2022. Using panel data regression, the analysis showed that a 10 percent increase in COVID-19 cases was associated with small but consistent rises in antibiotic sales, especially macrolides, with the largest increases observed in Europe, North America, and Africa. No consistent association was found between antibiotic sales and COVID-19 vaccination rates. These findings emphasize the continued importance of antibiotic stewardship throughout the pandemic.[24]

Tackling antimicrobial resistance requires global investment in stewardship, infection prevention, and new treatments. High-risk groups, especially older adults, should be prioritized. Strategies must be tailored to regional needs, with stronger surveillance and global data sharing. Continued research and coordinated international action are essential to reduce the growing AMR burden.[25]

Clinical Implications and Therapeutic Challenges

When bacteria become resistant to commonly used antibiotics, it can delay the start of proper treatment, leading to a higher risk of death, longer hospital stays, and increased healthcare costs. To manage this, doctors often turn to broad-spectrum antibiotics before lab results are ready, but overusing these medicines can worsen resistance. Understanding how multidrug-resistant (MDR) infections spread, identifying high-risk patients, and using the right antibiotic doses are all crucial steps. Tools like rapid diagnostic tests, patient risk assessments, and drug level monitoring can help guide more accurate and targeted treatments, reducing unnecessary antibiotic use and helping to slow the spread of resistance.[26]

AMR also has a big economic cost, with huge losses expected in healthcare and farming. Scientists are exploring how artificial intelligence (AI) can help, like by improving how we diagnose and treat infections. But there are still some issues, like making sure the data used is accurate and fair. To fight AMR properly, we need a One Health approach—this means working together across human, animal, and environmental health. It's important to improve how we track resistant infections, use antibiotics more carefully, and support research to find new treatments. Raising public awareness, educating people, and working together across countries are all key to protecting the power of antibiotics for the future.[4]

Drug Discovery and Development Challenges

Despite the urgent need for new antibiotics, there's a major shortfall due to poor financial incentives. Unlike drugs for cancer or chronic conditions, antibiotics offer low returns on investment. New antibiotics are often held in reserve to prevent resistance, limiting their use and revenue during the patent period. Additionally, pricing pressures keep antibiotic costs low, making it hard for developers to recover high R&D expenses.[27]

Basel has become one of Europe's main centers for life sciences, with over 700 companies working in areas like biotech, health technology, and drug development. Big pharmaceutical companies like Roche, Novartis, and Bayer have offices there, along with smaller groups focused on fighting antimicrobial resistance, such as BioVersys and Polyphor. This mix of big and small companies creates a strong network where people and ideas move easily.[28]

Antimicrobial Resistance Genes and Mobile Elements

Hospital-acquired infections caused by multidrug-resistant (MDR) Klebsiella pneumoniae are a major health concern, necessitating effective disinfection strategies. This study analyzed 50 clinical MDR isolates to evaluate antimicrobial susceptibility and biocide efficacy. All isolates showed high resistance, with multiple antimicrobial resistance (MAR) indices ranging from 0.24 to 1. Benzalkonium chloride MICs increased with resistance (up to 64 µg/mL), while chlorhexidine MICs remained stable. The biocide resistance genes qacEΔ1 and cepA were detected in 62% and 72% of isolates, respectively, with qacEΔ1 significantly linked to cephalosporin resistance. These findings highlight the role of cepA in drug resistance and underscore the importance of optimized biocide use in controlling MDR K. pneumoniae infections.[29]

The emergence of drug-resistant fungal pathogens like Candida auris poses a major global health threat, with mortality rates exceeding 40% and resistance rates over 90%. Due to limited antifungal options, this study explores the role of histone H3 modifications in resistance, focusing on acetylation by Gcn5 and Rtt109 and methylation by Set1, Set2, and Dot1. Mutants lacking these enzymes show altered drug sensitivity. Notably, GCN5 deletion reduces histone H3 acetylation, downregulates genes linked to ergosterol synthesis and drug efflux, and heightens susceptibility to azoles and polyenes. Gcn5 also influences echinocandin resistance and cell wall integrity via calcineurin signaling and Cas5. In infection models, GCN5 deletion lowers virulence, and the Gcn5 inhibitor CPTH2 enhances caspofungin efficacy without toxicity. These results identify Gcn5 as a key regulator of C. auris resistance and pathogenicity, making it a promising antifungal target.[30]

One Health Perspective

Efforts to combat AMR include monitoring antibiotic use and implementing global strategies, such as the UN's Sustainable Development Goals and the One Health approach, which links human, animal, and environmental health. Despite political attention from forums like the G7, public awareness and health literacy remain insufficient.(2) AMR is linked to each of these three components due to the irresponsible and excessive use of antimicrobials in various sectors (agriculture, cattle raising, and human medicine)(31) AMR is a major global threat impacting human, animal, and environmental health (Figure.5). Due to its complexity, addressing AMR requires a multidisciplinary perspective within the One Health approach.[32]

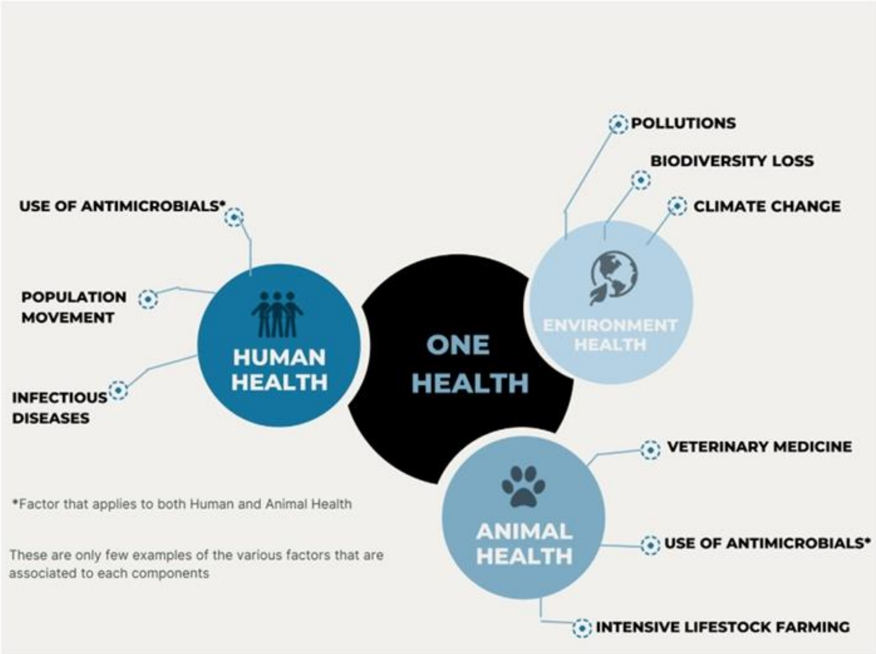

Fig. 6: Illustration representing the concept of One Health Approach (2)

The One Health concept is a multisectoral approach that requires joint efforts from all stakeholders to be effective. The World Health Organization (WHO) works closely with the Food and Agriculture Organization (FAO) and the World Organisation for Animal Health to ensure coordinated actions across sectors to reduce the risks of antimicrobial resistance.[33]

Novel Therapeutic Approaches, Public Health and International Strategies

Antimicrobials are agents designed to kill or inhibit harmful microbes. The global rise in antimicrobial resistance (AMR) is a growing public health concern. While developing new drugs is a key strategy, it is a slow and resource-intensive process. Meanwhile, multi-drug resistant (MDR) bacteria continue to evolve rapidly. To keep pace, research is increasingly focusing on boosting the effectiveness of existing antimicrobials through combination therapies, bacteriophage therapy, adjuvant use, and nanotechnology.[34]

Public awareness and education are vital to combat AMR. People must understand proper antibiotic use, the dangers of misuse, and the importance of completing prescribed treatments.[35]

Healthcare professionals must remain informed about antimicrobial stewardship, infection prevention, and control practices. In hospital settings, stewardship includes establishing guidelines, staff education, and implementing protocols to ensure prudent antibiotic use. In outpatient care, it emphasizes patient education, accurate diagnostics, and prescribing antibiotics only when necessary. Cultivating a culture of responsible antimicrobial use reduces microbial selection pressure and slows the emergence of resistance.

Alternative strategies to AMR include

- Developing new antibiotics (addresses resistance to existing drugs).
- Combination therapy (synergy can enhance effectiveness, reduce resistance).
- Phage therapy (using viruses that infect bacteria to target specific bacterial strains).
- Prebiotics and probiotics (support healthy microbiota, reduces space for pathogens).
- Immunotherapy (enhancing the body’s immune response to fight infections).
- Drug repurposing.
- Non-antibiotic alternatives.
- Public education (rational antibiotic use involves prescribing only when necessary and ensuring the appropriate dosage and duration).
- Surveillance systems.

CONCLUSION

Antimicrobial resistance (AMR) is a rapidly growing global health challenge that threatens to reverse many advances in modern medicine. It stems from the improper use of antimicrobials in humans, animals, and the environment, along with insufficient progress in developing new treatments. The impact of AMR is farreaching, leading to delayed treatments, higher mortality rates, increased healthcare costs, and limited therapeutic options. Combating this threat requires a comprehensive and coordinated approach that includes improving public awareness, promoting responsible antibiotic use, strengthening healthcare systems, and investing in research and innovation. The One Health approach, which integrates efforts across human, animal, and environmental health, is crucial to addressing the complex nature of AMR. With global collaboration and sustained commitment, it is possible to slow the spread of resistance and preserve the effectiveness of life-saving antimicrobial treatments for future generations.

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