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A Review of Antibiotic Resistance: Global Reports, Sources, Incidents, Resistance Strategies, and Control Plans

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ABSTRACT

Antimicrobial resistance is a major global issue that is only getting worse. Acquired resistance is defined by the emergence of coding genes for strategies of drug evasion from antimicrobial agents. The *Enterobacteriaceae* family has been linked to this behavior. Antibiotics like beta-lactams and carbapenems, which are the most used types, are used to treat bacterial infections. From a clinical perspective, research on antibiotic resistance is very important because of the effects it has on human health. Furthermore, one of the rare instances of evolution that can be researched in real time is antibiotic resistance. Therefore, doctors, evolutionary biologists, and ecologists are interested in understanding the general processes involved in the acquisition of antibiotic resistance. Environmental microbes are the source of antibiotic resistance genes that are currently found in human diseases. Therefore, research on both natural and medical environments is necessary to fully comprehend the emergence of antibiotic resistance. Recent findings about the evolutionary processes underlying resistance suggest that viability costs, the founder effect, and ecological connectivity are significant barriers that control the spread of resistance from environmental bacteria to diseases.

Keywords: Antimicrobial Resistance (AMR), *Enterobacteriaceae,* β-lactamases, mechanisms, and control plans.

INTRODUCTION:

A wide and diverse group of rod-shaped, Gram-negative bacteria that are naturally prevalent in the mammalian gut but can also be found in other settings make up the family *Enterobacteriaceae* (Lazm *et al.,* 2019). Frequently found in clinical laboratories, they cause a wide range of diseases, including gastrointestinal illnesses, urinary tract infections, burn and wound infections, bloodstream infections, pneumonia, & infections of the eyes, ears, and sinuses. Many *E. coli* serotypes, or strains from *Citrobacter, Enterobacter, Hafnia, Klebsiella, Proteus, Providencia, Salmonella, Yersinia*, and other genera, have long been linked to a significant portion of the nosocomial infections, including intestinal infections like endocarditis and diarrhea, as well as infections of the skin, joints, bones, eyes, respiratory tract, and urinary tract (Brenner *et al.,* 2005; Lazm *et al.,* 2019). Antibiotics are among the most effective medications ever created.

Because they are the natural products, their chemical makeup, biosynthetic processes, evolutionary history, and biochemical mechanism of the action all present stimulating intellectual challenges and the surprises

(Brötz-Oesterhelt & Brunner, 2008; Strohl, 1997). The comp-lete synthesis of these natural compounds in the lab is challenging since the functionality and chirality of these tiny molecules are frequently incredibly complex (Beutler, 2009). It was clear as soon as they discovered that bacteria may develop resistance to them. For a few decades, the continuous development of novel anti-biotics served to mitigate this issue. The upshot of many years of constant selection pressure from human uses of antibiotics, via underuse, overuse, and misuse, is the production of the generations of antibiotic-resistant bacteria and their dispersion in microbial communities through-out the biosphere. This is a man-made circumstance that has been forced on nature not a natural process (Serwecińska, 2020). But in recent years, this has significantly slowed down, which has increased the prevalence of the bacterial infections that are resistant to antibiotics (ABRs) (Levy & Marshall, 2004). It is a global issue because antibiotic resistance in the pathogenic bacteria might evolve primarily as a result of misuse (Ahmed *et al.,* 2019). It is expected to cause a sharp rise in mortality as a worldwide health hazard, accounting for thous-ands of deaths per year (Momtaz *et al.,* 2012). The identification of these pathogenic organisms in the late 1800s sparked the hunt for suitable prophylactic and therapeutic protocols; yet effective treatment was not achieved until the discovery and commercialization of antibiotics in the 1950s.

Millions of lives have been saved and medicine has altered since the advent of the bacteria (Spellberg & Gilbert, 2014). Since the discovery, widespread use, and appli-cation of antimicrobial resistance (AMR), the treatment of infectious diseases and its relationship to unfavorable health outcomes, such as treatment failure, the prolonged illness, & mortality, has been transformed (Organization, 2014). According to the most recent annual report of the European Anti-microbial Resistance Surveillance Network (EARS-Net), Romania has one of the highest incidences of multidrug resistant (MDR) Enterobacteriaceae. Resis-tance to aminoglycosides and third-generation cepha-losporins, as well as resistance to the fluoroquinolones, aminoglycosides, and third-generation cephalosporins combined, have both significantly increased recently (Mader *et al.,* 2021). Different bacteria can produce and spread antibiotic resistance genes (ARGs) quickly. In this sense, AMR makes harmful organisms more accessible than ever before, limiting clinical medicine's options for medication regimens intended to treat bacterial infections. ARG types or AMR strains can spread widely from patient to patient in hospitals, between individuals in the com-munity, or between clinical settings and the surrounding environment (Szekeres *et al.,* 2017). This review looks at the broad ideas surrounding the emergence, dissemination, & evolution of the antibiotic resistance in bacterial pathogens.

Global reports on antibiotic resistance

Since they combat germs, antibiotics are referred to as the "wonder medicines" (Zaman *et al.,* 2017). Their discovery and eventual therapeutic use represent a medical miracle (Watkins & Bonomo, 2016). The emergence of resistance mechanisms has hampered the therapeutic use of sulfonamides, the first class of antimicrobials to be proven effective, since its debut in 1937. Around the 70 years have passed since the late 1930s, when sulfonamide resistance was the first documented (Abraham & Chain, 1940). The evolution and resistance acquisition of different antibiotics is shown in **Fig. 1**. Following the antibiotic's widespread use, resistant strains that could neutralize the medication started to proliferate. As a result, research was done to chemically alter penicillin to stop it from being broken down by penicillinase (β lactamases). It's interesting to note that, in light of the subsequent discoveries showing that the many antibiotic-resistant genes are present in naturally occur-ring microbial populations, the discovery of a bacterial penicillinase prior to the application of the antibiotic can now be appreciated (D'Costa *et al.,* 2006). Because antibiotics can be used to treat the diseases they cause, antibiotic resistance has evolved. First, resistance was seen in *Staphylococci*, *Streptococci*, and *Gonococci*. In 1941, the first commercial antibiotic, penicillin, was released into the market. A year later, in the 1942, penicillin-resistant S. aureus was discovered (Dodds, 2017). It was discovered that during patient treatment, mutant strains of *Mycobacterium tuberculosis* resistant to therapeutic amounts of the antibiotic emerged. Strepto-mycin was first developed in 1944 to treat tuberculosis (the TB; "The Great White Plague"). When genetically

transferable antibiotic resistance was unexpectedly discovered in Japan in the middle of the 1950s (and was first met with the skepticism in the West), it completely altered the landscape by introducing the heretical genetic concept that antibiotic resistance gene collections could spread by the bacterial conjugation throughout a population of the bacterial pathogens (Davies, 1995; Helinski, 2004; Shahen *et al*., 2019).

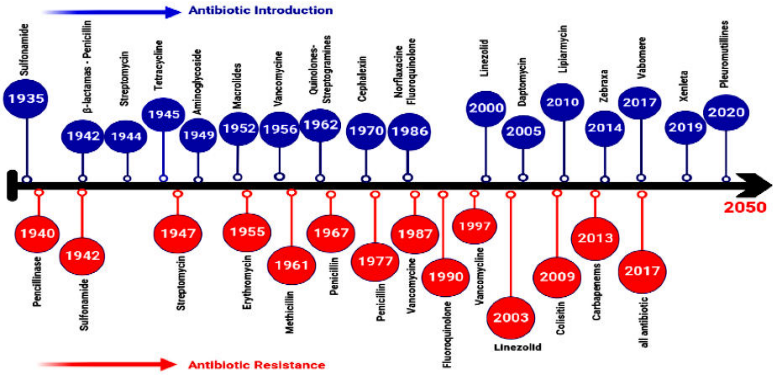

Fig. 1: The history of antibiotics evolution.

Methicillin, a semi-synthetic antibiotic related to penicillin that was first brought to the market in 1960 to treat *S. aureus* that was resistant to the penicillin, developed resistance to methicillin that same year (Durand, Raoult, & Dubourg, 2019). During that same decade, the first cases of the methicillin-resistant *Staphylococcus aureus* (MRSA) were discovered in the US in 1968 and in the UK in 1962 (Ventola, 2015). Methicillin resistance *in S. aureus* and coagulase-negative staphylococci was treated with vancomycin in 1972. However, it was thought that the injected vancomycin resistance would not materialize in a clinical context. On the other hand, the vancomycin-resistant coagulase-negative s*taphylococci* were the documented in 1979 and 1983 (Sengupta *et al.,* 2013).

Another source of the potentially fatal infections is multidrug-resistant Pseudomonas aeruginosa; isolates resistant to the carbapenem have been classified as "critical." on the WHO's list of priority pathogens. Methicillin-resistant *Staphylococcus aureus* (MRSA), carbapenem-resistant *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* are only a few of the pathogens on the World Health Organization's (WHO) priority list of infections that have been documented in the nation (Pitout *et al.,* 2008; Tacconelli, 2017). The public health community has identified *Enterobacteriaceae* resistant to carbapenems and cephalosporins of the third as critically essential because there are the limited treatment options for these illnesses (Tacconelli, 2017). In recent years, it has become clear that gene exchange is a characteristic shared by all bacteria and has happened during the entire history of microbial evolution (Tacconelli, 2017). Since more than 70% of harmful bacteria are resistant to at least one antibiotic, resistance to the antibiotics can develop quickly. It is currently one of the biggest threats to food safety, public health, and sustainable healthcare *(Rahman et al*., 2019; Uddin *et al.,* 2021).

Sources of antibiotic resistance

The bacterial species *streptococc*i and *gonococci* were the first to be found to be resistant. When using antibiotics to treat *tuberculosis* (TB), resistance initially posed a significant problem (Gillespie, 2002). The three primary sources of the antibiotic resistance, according to the European Centre for Disease Prevention and Control, were recently described. According to their research, there are three instances of antibiotic overuse that are the known to increase the risk of antibiotic resistance (Prestinaci *et al.,* 2015). The first kind of overuse involves giving antibiotics for viral illnesses when they are not needed. Antibiotics do not affect viral infections; they are only effective against bacterial infections (Llor & Bjerrum, 2014). Second, the growth and spread of antibiotic-resistant bacteria are accelerated by the overuse of antibiotics. The improper use of antibiotics that hastens the emergence and spread of micro-organisms resistant to antibiotics is identified as the third misuse scenario. Thus, when

an antibiotic is the introduced into the market, micro-organisms are the susceptible to it (from a clinical perspective); resistance develops later. Resistance has emerged through two different mechanisms: horizontal gene transfer (HGT) and mutation (Tao *et al.,* 2022; Ventola, 2015). Since the etiology of the illness is uncertain due to the development of resistant bacteria, physicians prescribe a broad-spectrum antibiotic that kills off germs rather than only the specific bacteria linked to the ailment. The ECDC concludes by the highlighting improper antibiotic use as the third misuse scenario that contributes to the antibiotic resistance (Davies & Davies, 2010; Llor & Bjerrum, 2014).

Incidences of antibiotic resistance

Antibiotic resistance in human isolates

A summary of the Africa, the Americas, the eastern Mediterranean, Europe, Southeast Asia, and the western Pacific was given in the World Health Organization's 2014 report on worldwide resistance (Organization, 2020). After the usage of antibiotics, bacterial pathogens linked to the human illness outbreaks have changed into multidrug-resistant (MDR) strains. As the 20th-century equivalent of an ancient pathogen, MDR *M. tuberculosis*, for instance, is a significant pathogen present in both industrialized and underdeveloped countries (Davies & Davies, 2010). Numerous penicillin's and cephalosporins (**Table 1**) can be resistant to the carriage of the extended spectrum β-lactamases (ESBLs; see Glossary), and these mechanisms frequently co-occur with those that impart resistance to other antibiotic classes (Iredell *et al.,* 2016).

Table 1: Selected important β lactamases in *Enterobacteriaceae* (Iredell *et al.,* 2016; Paterson & Bonomo, 2005).

Commonly affected ß lactams	Common terminology	Examples	Clinical inhibitors	Laboratory inhibitors	Effective ß lactams	Commonly implicated species
PEN, 1GC	Penicillinases	TEM, SHV	Clavulanate, tazobactam, sulbactam avibactam	Clavulanate, tazobactam, sulbactam	TZP, 3GC, 4GC, MEM	*E. coli* (P), *Klebsiella* spp (C) (P)
PEN, 1GC 3GC, ATZ	ESBLs	CTX-M			MEM, (TZP, 4GC) *	ESBL: most medically important Enterobacteriaceae (P)
PEN, 1GC, 2GC† 3GC	OXA-48-type‡ β lactamases	OXA-48/181‡	Avibactam	None	(MEM, ATZ) *	OXA, MBL, KPC: most medically important Enterobacteriaceae (P)
PEN, 1GC, 2GC†	K pneumoniae carbapenemases	KPC	Avibactam	Boronic acids	MEM	*K. pneumoniae* carbapenemases (Ambler)

Abbreviations: 1/2/3/4GC=1st/2nd/3rd/4th generation cephalosporins; AmpC=ampicillin hydrolyzing enzymes; ATZ = monobactam antibiotics (such as aztreonam); CMY-2=cephamycinase; CTXM=cefotaximase, first identified in Munich; ESBLs=extended spectrum β lactamases; IMP=imipenemase; KPC=Klebsiella pneumoniae carbapenemase; MBLs = metallo β lactamases; MEM=carbapenems (such as meropenem); NDM=New Delhi metallo β lactamase; PEN = penicillins; TEM, SHV, and OXA=common and diverse groups of β lactam hydrolyzing enzymes; TZP=penicillin β lactamase inhibitor combinations (such as piperacillin-tazobactam); VIM=Verona integron encoded metallo β lactamase; (P)/(C), commonly plasmid/chromosomally encoded.

*May vary with enzyme or drug combination or coexisting mechanisms that contribute to the phenotype.

Subgroups with a high prevalence of resistance

Travelers returning to nations with modern healthcare systems are frequently the first to the identify "new" antibiotic resistance problems, and screening and the laboratory testing are the strongly advised for people returning after receiving healthcare (Adler *et al.,* 2011; Yong *et al.,* 2009). From very low baselines in those countries, returning Dutch and Swedish international travelers were the reported to have 24% ESBL colonization rates. Asymptomatic military personnel stationed in Afghanistan were reported to have 10-fold higher colonization rates of the *E. coli* that contained ESBL than those stationed in the US (Hasan *et al.,* 2014; Musyok *et al.,* 2019). Antibiotic resistance is also sometimes highly prevalent in the long-term inhabitants of the elderly care institutions, who are frequently exposed to medical care, antibiotics, and common cross-transmission of microorganisms. Hospital-linked infections with *Staphylococcus aureus,* the *Staphylococcus epidermis, Stenotrophomonas malto-*

philia, Streptococcus pneumoniae, Acinetobacter baumannii, Burkholderia cepacia, Campylobacter jejuni, Citrobacter freundii, Clostridium difficile, Enterobacter spp., Enterococcus faecium, Enterococcus faecalis, E. coli, Haemophilus influenzae, Klebsiella pneumoniae, Proteus mirabilis, Pseudomonas aeruginosa, Salmonella spp., and *Serratia spp.,* are the available antibiotic resistance data, in which antibiotic exposure and cross-transmission are expected to be increased (Davies & Davies, 2010; Musyok *et al.,* 2019).

Antibiotic resistance in isolates from environmental and agricultural sources

Resistance is likely influenced by antimicrobials used in animal husbandry as well as trash and effluent from manufacturers and hospitals (Diwan *et al.,* 2010). Human and animal waste contaminates the environment, and in both resource-poor and resource-rich nations, highly resistant *E. coli* may be present in the drinking and environmental water supplies. Similar multi-resistant isolations to humans may be found in domestic pets. Antibiotic-resistant human infections are also frequently found in food and in animals that are part of the food chain (Schmiedel *et al.,* 2014; Timofte *et al.,* 2011). Animals in the wild are the frequently impacted, especially scavengers like seagulls, who are significant carriers of the antibiotic resistance in the infections known to affect humans (Iredell *et al.,* 2016).

Bacterial resistance strategies against antibiotics

Antibiotic-resistant bacteria are a serious and the expanding global issue. Antibiotic-resistant bacteria have evolved a variety of defense mechanisms, making illness treatment difficult. These resistance mechanisms might be acquired (formed over time because of selective pressure from antibiotic use) or intrinsic (occurring naturally). A list of antibiotics and their modes of action are given in **Table 2**.

Table 2: Names of drugs and their resistance mechanisms (Davies & Davies, 2010; Morar & Wright, 2010).

Drug Class	Examples	Target	Modes of Action
Aminoglycosides	Gentamicin, streptomycin, spectinomycin	Translation	Phosphorylation, acetylation, efflux pump, altered target
Beta-lactams	Penicillins (ampicillin), cephalos-porins (cephamycin), carbapenems (mero-penem), monobactams (aztreonam)	Peptidoglycan biosynthesis	Hydrolysis, efflux pump, altered target gene
Tetracyclines	Minocycline, tigecycline	Translation	Monooxygenation, efflux pump, altered target gene
Glycopeptides	Vancomycin	Peptidoglycan biosynthesis	Reprogramming peptidoglycan biosynthesis
Lincosamides	Clindamycin	Translation	Nucleotidylation, efflux pump, altered target gene
Macrolides	Erythromycin, azithromicin	Translation	Hydrolysis, glycosylation, phosphor-rylation, efflux pump, altered target gene
Oxazolidinones	Linezolid	Translation	Efflux pump, altered target gene
Quinolones	Ciprofloxacin	DNA replication	Acetylation, efflux, altered target
Phenicols	Chloramphenicol	Translation	Acetylation, efflux, altered target
Sulfonamides	Sulfamethoxazole	C1 metabolism	Efflux pump, altered target
Pyrimidines	Trimethoprim	C1 metabolism	Efflux pump, altered target
Lipopeptides	Daptomycin	Cell membrane	Altered target gene
Polymyxin b	Colistin	Cell membrane	Efflux pump, altered target gene

Antibiotic management plans

Antibiotic stewardship programs, also known as antibiotic management plans, are comprehensive initiatives used in hospital settings to guarantee the prudent use of antibiotics. These strategies seek to minimize the emergence of antibiotic resistance, enhance patient outcomes, and maximize the use of antibiotics. Global antibiotic resistance control is a long-term endeavor

requiring tenacity & cooperation from nations, institutions, healthcare systems, and the public. We can stop the development of antibiotic resistance and ensure that antibiotics remain effective for the upcoming generations by cooperating and putting these methods into practice. Here are some key strategies for the antibiotic resistance management:

Antibiotic stewardship

This entails using antibiotics sensibly and only prescribing them when essential. Medical practitioners should refrain from giving antibiotics for viral infections and instead choose the appropriate drug, dosage, and course of therapy.

Public awareness and education

It is crucial to raise public awareness of the risks associated with antibiotic resistance. It's critical that people comprehend the significance of taking antibiotics exactly as the directed, refraining from sharing medications, and not forcing physicians to prescribe drugs when none are necessary.

Surveillance and monitoring

It's important to regularly monitor patterns of antibiotic resistance. This information can direct public health initiatives and assist medical professionals in making well-informed judgments regarding which antibiotics to prescribe diseases.

Infection prevention and control

Appropriate controls for infections in healthcare environments can help stop the spread of microorganisms resistant to antibiotics. This covers precautions like hand washing, isolating patients who have infec-tions that are resistant, and following sanitation guidelines.

Research and development

To counteract the emergence of resistance, it is critical to develop novel antibiotics and complementary therapies. It is essential to the fund research into novel antibiotics and treatments.

Vaccination

Antibiotic use can be decreased using vaccines, which can help avoid numerous bacterial infections. In this sense, vaccinations against certain forms of meningitis and bacterial pneumonia, for example, have proven helpful.

One health approach

It is essential to understand how the health of people, animals, and the environment are interconnected. For instance, the use of antibiotics in agriculture may encourage the growth of resistant bacteria that may harm people. In a "One Health" strategy, the human and animal health sectors work together to manage antibiotic resistance holistically.

Regulatory measures

Governments have the power to control the use of antibiotics in both agriculture and healthcare. One way to reduce resistance is to implement and enforce laws and regulations that limit the improper use of antibiotics.

Development of fast diagnostic tests

Accurate antibiotic prescriptions can be made by the health-care professionals with the aid of enhanced diagnostic tests that can rapidly determine the kind of infection and the bacterium causing it.

International cooperation

Since antibiotic resistance is a worldwide issue, cooperation between nations is the crucial. Nations can cooperate on research and policy development, as well as exchange information and best practices.

Alternative therapies

One viable way to the fight antibiotic resistance is to investigate and develop non-antibiotic medicines like phage therapy, antibodies, and other cutting-edge techniques.

Patient engagement

One way to stop the overuse of antibiotics is to encourage patients to take an active role in their care. This entails talking about available treatment alternatives, posing inquiries, and realizing how crucial it is to adhere to recommended treatment plans.

CONCLUSION:

Antibiotic resistance is a global health crisis that the develops when bacteria develop the ability to survive and multiply in the presence of the antibiotics. This resistance might make treating bacterial infections more difficult, which might lead to infections that are more serious and the require longer recovery times. Antibiotic resistance must be controlled if antibiotics

are to be effective in the future as well as the present. To effectively control antibiotic resistance, a multidisciplinary strategy involving the medical professionals, rese-archers, legislators, and the public is needed. To counter this growing threat to the public health, cooperation is essential.

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CONFLICTS OF INTERESTS:

The authors declare no conflict of interest

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