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Antibiotic Resistance

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Abstract: *Antibiotics have been the key to treating and preventing bacterial infections since the discovery of penicillin. However, the improper and widespread use of antibiotics has resulted in the development of antibiotics resistance. This is one of the main issues facing the world today. Antibiotic resistance is the ability of bacteria to survive the drugs that have been effective against them in the past. In the pharmacological sense, there are several ways through which antibiotic resistance can develop. This includes the breakdown of the drugs, changing the target of the drugs, reducing the permeability of the drugs, and expelling the drugs from the bacterial cells. This makes the commonly used antibiotics less effective and complicates the process of treating bacterial infections.*

Today, the number of multidrug-resistant pathogens is on the rise. This has resulted in increased illness, death, and healthcare costs around the world. This is mainly because of the widespread and improper use of antibiotics. In order to combat the growing menace of antibiotic resistance, several pharmacological strategies have been put in place. This review article will discuss the pharmacological basis of antibiotic resistance and the ways to prevent it.

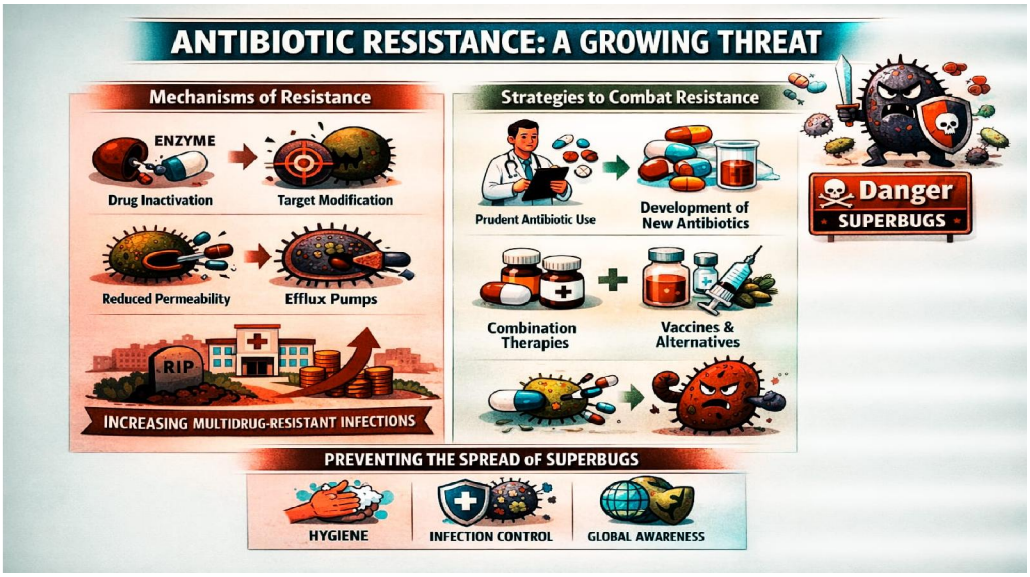

Fig 1 : A Growing therapy

Keywords: antibiotic resistance

I. INTRODUCTION

Currently, antibiotics remain some of the most important therapeutic agents in contemporary medicine for treating bacterial infections. The availability of antibiotic therapy since the introduction of penicillin in the mid-20th century has resulted in the decreased incidence of death and disease due to infectious diseases due to the availability of antibiotics. Antibiotics exert their antimicrobial effect through a variety of pharmacological mechanisms that selectively target bacterial cells as opposed to producing deleterious effects on the host. These pharmacological

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mechanisms include the inhibition of bacterial cell wall biosynthesis, the inhibition of protein synthesis, the inhibition of nucleic acid biosynthesis, and the disruption of metabolic pathways.

With increasing use and abuse of antibiotics, the number of bacteria becoming resistant to the effects of antibiotics is increasing because of the emergence and dissemination of antibiotic-resistant bacteria at an accelerated pace. Antibiotic-resistant bacteria are capable of surviving and replicating in levels of antibiotic that would previously have caused a reduction in their ability to reproduce or die. There are two primary methods by which bacteria become resistant to the effects of antibiotics: 1) through genetic mutations, and 2) horizontal gene transfer through transformation, transduction, or conjugation.

From a pharmacological point of view, antibiotic resistance is mediated by a number of factors. Amongst them are the enzymatic degradation and modification of antibiotics, such as the production of β-lactamase enzymes. Other factors include alterations in the site of action of antibiotics and reduced permeability of the cell membranes of bacteria, as well as the active efflux of antibiotics from the cells of bacteria.

The emergence of antibiotic resistance on a global scale is a major concern to health care today. The improper and excessive use of antibiotics in practice and medicine, self-medication, non-compliance with treatment regimens, and excessive use of antibiotics in agriculture and cattle rearing have contributed to the emergence of resistant microbial populations. As a consequence, infections that were previously easy to treat are now becoming hard to therefore, understanding how pharmacology creates resistance against antibiotics is important if we want to create effective strategies for preventing such problems. This review will explore several topics about the pharmacology of antibiotic resistance, including its underlying pharmacological reasons, causes and prevention methods.

II. PATHOPHYSIOLOGY OF ANTIBIOTIC RESISTANCE

Antibiotics are often used to successfully kill background bacteria but may not be effective against these resistant types as they have adapted to antibiotics through genetic modification or gaining resistance genes. There are numerous genetic mutations within bacteria that develop during the course of an antibiotic therapy (for example; certain target proteins that an antibiotic may interact with). In addition, bacteria can acquire resistance genes through various horizontal gene transfer mechanisms (e.g. conjugation, transduction, transformation). The genetic material containing the resistance gene(s) can be on different types of DNA structures (e.g. plasmids, transposes, Integrants). Bacteria are able to exchange their DNA with adjacent bacteria through these means, many bacteria can destroy or change an antibiotic before it reaches its site of action by producing enzymes. An example of this is the production of β-lactamase enzymes that hydrolyze the β-lactamase ring of antibiotics (e.g., penicillin and cephalosporin). β-lactamase antibiotics are no longer effective against bacteria that secrete β-lactamase since enzymatic processes have rendered these antibiotics ineffective and have allowed for the survival of bacteria in the presence of antibiotic exposure facilitate the rapid transmission of resistant types throughout the entire population of a bacterial species. [1]

Enzymatic inhibition of antibiotics

Many bacteria can destroy or change an antibiotic before it reaches its site of action by producing enzymes. An example of this is the production of β -lactamase enzymes that hydrolyze the β-lactamase ring of antibiotics (e.g., penicillin and cephalosporin). B-lactamase antibiotics are no longer effective against bacteria that secrete β-lactamase since enzymatic processes have rendered these antibiotics ineffective and have allowed for the survival of bacteria in the presence of antibiotic exposure.[2]

Changing/ Modifying Drug Targets Bacteria can alter the structure of molecular targets where antibiotics normally bind. Most commonly, this results in the reduced affinity of antibiotics to their binding sites, thus resulting in decreased therapeutic efficacy. Some examples of this are that alterations of penicillin-binding proteins result in resistance to β-lactamase antibiotics, whereas alterations of ribosomal subunits will confer resistance to macro ides and aminoglycosides.[3]

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Decreased Permeability Of Bacteria To Antibiotics

Certain species of bacteria prevent antibiotics from entering their cells through alterations to their membrane permeability. For example, many gram negative bacteria have an outer membrane of porins that act as tunnels for antibiotic entry. A mutation or decreased expression of these porins will decrease the amount of antibiotic inside a bacterial cell, decreasing efficacy against that organism. [4]

Active Efflux Of Antibiotics

Efflux pumps are transport proteins that expel antibiotics from the cytoplasm of bacteria. Overexpression of efflux systems lowers the amount of antibiotic in bacterial cells below a level that is able to exert a therapeutic effect. As a result, the bacteria can survive treatment with an antibiotic. Efflux pumps are also commonly involved in developing multidrug resistance because they can expel multiple classes of antibiotics.[5]

Biofilm Formation

Many bacteria form biofilms (i.e., communities of microorganisms enclosed in a matrix), which impede the penetration of antibiotics and allow the bacteria to remain alive in hostile environments. Biofilms also change the metabolism of the bacteria in the biofilm and contribute greatly to the bacteria's ability to remain resistant to antimicrobial agents and to evade host immune responses.[6]

Selective pressure and spread of resistance

Selective pressure favouring the survival and propagation of resistant bacteria is induced by inappropriate prescribing of antibiotic medications. The result of this is that susceptible bacteria are eliminated from the local environment and those bacteria that are resistant have the opportunity to grow and spread throughout the community and/or healthcare system. The result is that the emergence and spread of multidrug-resistant pathogens is now a global phenomenon [7].

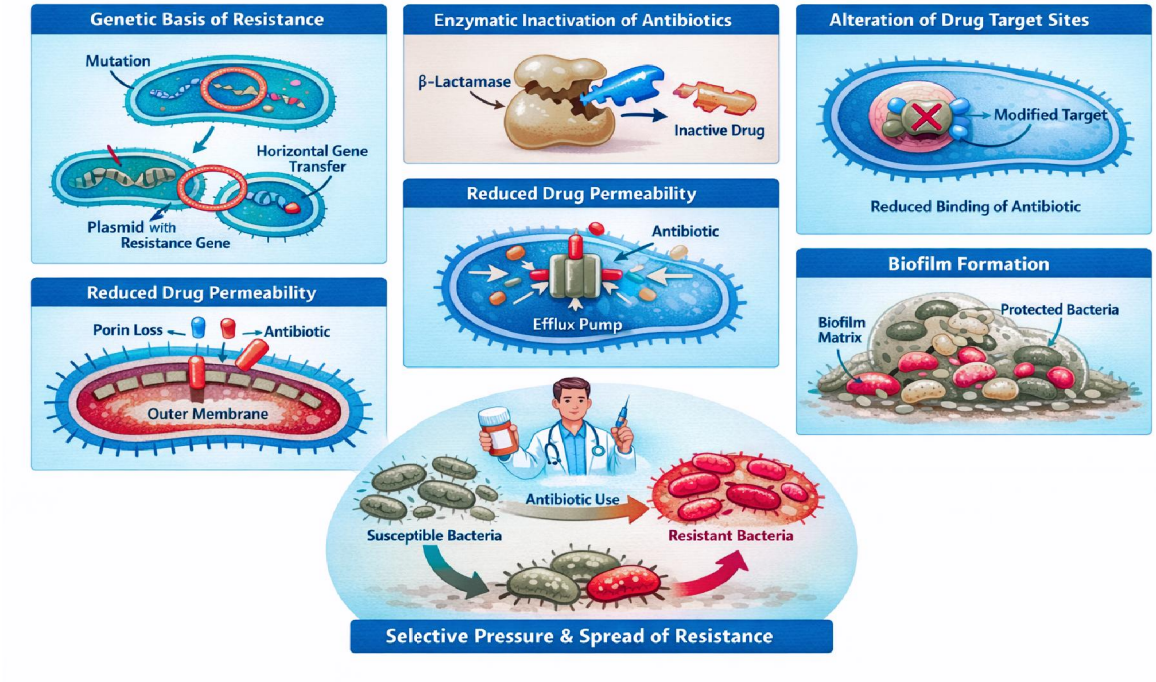

Fig 2: Mechanism and spread of antibiotic resistance in bactria

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III. MECHANISM OF ACTION OF CIPROFLOXACIN

Ciprofloxacin, which is a fluoroquinolone (also known as a "fluoroquinolone antibiotic"), is effective against both Gram-negative and some Gram-positive bacteria (e.g., some bacteria that live in your body) in treating infections such as urinary tract infections (UTI), gastrointestinal infections (GI), and respiratory tract infections (RTI). In general, ciprofloxacin kills bacteria by preventing or disrupting the normal processes of replication and transcription of their DNA.[8]

Inhibition of Bacterial DNA Gyrase

One of the ways that ciprofloxacin works to kill bacteria is through the inhibition of one of the enzymes needed to build DNA in bacteria, called DNA gyrase (also known as topoisomerase II). This enzyme is very important for bacteria because it helps make sure that DNA is supercoiled in the correct direction (i.e., negative supercoiling) to enable its replication and transcription. When ciprofloxacin binds to the DNA–gyrase complex, it forms a stable bond with them following cleavage of the DNA strand and does not allow that strand of DNA to be re-ligated to the gyrase again. The result is that the bacterium will have a lot of broken DNAs and will not be able to divide.[9]

Topoisomerase IV Inhibition

Topoisomerase IV is a second major type of bacteria topoisomerase; it facilitates the separation of replicated (daughter) chromosomes that allow cells to divide properly. When this is inhibited, daughter chromosomes will not be separated correctly and therefore bacterium will die.[10]

Bactericidal Action

Unlike bacteriostatic antibiotics, ciprofloxacin has bactericidal action due to its capacity to accumulate DNA damage via inhibition of both bacterial DNA gyrase (topoisomerase II) and topoisomerase IV enzymes. The lethal effectiveness of ciprofloxacin relative to Gram-negative bacteria is dependent on obtaining sufficient concentrations of ciprofloxacin to exceed the pathogen's minimum inhibitory concentration (MIC).[11]

Pharmacological Importance

Ciprofloxacin has a wide range of effects due to the mechanism through which it works by targeting the enzymes responsible for replicating the DNA of bacteria. This makes it very effective against various Gram negative species of bacteria including E. coli and Pseudomonas aeruginosa. How ever, bacteria may develop resistance to this drug via alterations in the DNA gyrase gene and or the topoisomerase IV gene, by decreased permeability of the cell wall for this drug, or by increased activity of the efflux pump protein.[12]

IV. CLINICAL EVIDENCE FOR CIPROFLOXACIN

1. Evidences in Urinary Tract Infection

Ciprofloxacin is a proven medication for the treatment of urinary tract infections (UTIs) caused by various types of Gram-negative bacteria such as E. coli. In a randomized clinical trial, Ciprofloxacin was found to be able to eliminate bacteria, as well as provide quicker relief from symptoms than older medications. In the same study, the cure rate for uncomplicated UTIs was greater than 80% to 90%. [13]

2. Evidence in Gastrointestinal infections

Ciprofloxacin has also been shown to effectively treat various types of bacterial diarrhea and gastrointestinal infections caused by bacteria such as Salmonella, Shigella and Campylobacter. Research has demonstrated that the use of Ciprofloxacin leads to a shorter duration of illness and less severe diarrhea in patients. This medication is commonly used to treat traveler's diarrhea due to its ability to effectively treat enteric Gram-negative bacteria. [14]

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3. Ciprofloxacin and its Clinical Effectiveness for Respiratory Infections

Ciprofloxacin has shown clinical effectiveness against pathogens causing respiratory infections, particularly those associated with gram-negative bacteria (e.g. aerobic Pseudomonas aeruginosa). Studies have shown that ciprofloxacin has been effective in improving the outcomes of both chronic lower respiratory tract infections (LRI's) and acute respiratory infections (ARI's) and that ciprofloxacin users have had a higher probability of their bacteria being cleared or experiencing a more favourable outcome than did patients who did not use ciprofloxacin.[15]

4. The Pharmacokinetic/Pharmacodynamic Data for Ciprofloxacin

Ciprofloxacin has a high oral bioavailability (~70%) and achieves relatively high concentrations within the human body in all body fluids and tissues. Ciprofloxacin has been shown to be bactericidal in nature at high concentrations, and, thus, as the concentration is increased so is the amount of bacteria killed. These pharmacokinetic/pharmacodynamic characteristics of ciprofloxacin have been helpful in determining clinical efficacy and its ability to treat various common types of infections.[16]

5. Evidence From International Organizations and Guidelines

According to the World Health Organization (WHO) and other guidelines on the management of infectious diseases, there is also agreement that ciprofloxacin is a valid therapy for treating many types of bacterial infections (i.e., while it is an effective treatment, there are also significant concerns about possible adverse reactions to the medication, as well as about the development of anti-microbial resistance) and, therefore, it should not only be administered according to established guidelines but with caution given the continued development of anti-microbial resistance.[17]

V. RECENT TUBERCULOSIS ANTIBIOTIC ADVANCES

The recent increase in antimicrobial resistance has led to the development of new drugs for infections and new drug therapy. Some of the major advances currently being researched are directed toward enhancing the effectiveness of drugs already available, recovering the effectiveness of existing drugs, and finding entirely new antimicrobiaL agents.

New Antibacterial Agents

Due to continued bacterial resistance to antibiotics, several new antibiotics have been approved by FDA recently to treat infections due to these resistant bacteria. One of the newest of these, ceftazidime–avibactam, has been shown to kill multidrug-resistant gram-negative bacilli (e.g., Acinetobacter baumannii or enterobacteriaceae) and is characterized by its ability to inhibit the action of β-lactamase by producing a β-lactamase inhibitor that also inhibits the normal function of β-lactams. Another recently approved fluoroquinolone is delafloxacin, which has been reported to inhibit the growth of resistant organisms more effectively than.[18]

COMBINATION THERAPY

Combining multiple types of antimicrobial medications (including oral and injectable) may result in better outcome for patients by reducing the chance of resistance developing. For example, when using penicillin-type beta-lactam (penicillin-like) antibiotics with beta-lactamase inhibitors (such as clavulanic acid), activity is restored against resistant bacteria that produce beta-lactamase enzymes. This is often called combination therapy.[19]

BACTERIOPHAGE THERAPY

The use of bacteriophages, which are viruses that specifically infect and kill bacteria, to treat patients with infections from multi-drug resistant organisms is a newly growing area of medical research. Bacteriophages have become a potential alternative to traditional treatments for infections caused by multi-drug resistant organisms.[20]

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ANTIMICROBIAL PEPTIDES:

Antimicrobial peptides are a type of naturally occurring molecule produced by animals and other living beings as part of their innate immune system and have the ability to disrupt the cell membrane of bacteria. Researchers are investigating the potential for therapeutic use of antimicrobial peptides to treat patients with infections from resistant organisms.[21]

NANOTECHNOLOGY BASED DRUG DELIIVERY :

Nanotechnology is being explored to develop ways to deliver antibiotics more effectively and increase their bioavailability, improve how they are targeted to resistant bacteria, and cause infections directly to the infected areas of the body by providing a means to bypass the body's normal methods of protecting itself from bacteria [22].

FUTURE STRATAGIES TO CONTROL ANTIBIOTIC RESISTANCE :

Likewise, there are several areas where coordinated efforts need to occur to develop effective strategies for controlling antibiotic resistance both at the level of the individual healthcare facility, as well as at the community and global levels.

ANTIBIOTIC STEWARDSHIP PROGRAMS :

One such coordinated effort is through the use of antibiotic stewardship programs. Such programs focus on improving the way antibiotic medications are prescribed and used in health care settings by ensuring that a physician utilizes the appropriate medication, the correct dose is prescribed, and the proper amount of time is spent in therapy. By reducing the amount of time a patient receives an antibiotic unnecessarily, the program helps to minimize the likelihood of developing antibiotic resistance [23].

Monitoring and Surveillance

There are worldwide networks that keep track of how people are affected by antibiotics, which helps inform how they should be treated. Internationally, groups like the WHO have begun tracking how individuals with resistant bacteria and other pathogens will be treated from a global perspective through international surveillance programs. [24]

Developing New Antibiotics

To fight resistant bacteria, ongoing research needs to be performed on developing new antibiotics using different modes of action. Antimicrobial targets may be identified through better technology in genomics and drug development.[25]

Vaccination Programs

When vaccines are developed for a disease, the amount of bacterial infections will decrease and subsequently reduce the number of antibiotic prescriptions. Vaccinating people before they are infected is a key tool in fighting resistant bacteria. Changing Perspectives and Understanding of Resistance.[26]

Public awareness and education :

Educating healthcare workers and the population on appropriate ways to use antibiotics, avoiding self-treatment, and completing prescribed antibiotic Educational material to healthcare providers and the general public is critical to the prevention of resistance.[27]

Clinical Challenges

Despite advancements in antimicrobial therapy over several decades, clinical challenges exist that limit the clinical usefulness of antibiotics.

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VI. INCREASING MULTI DRUG RESISTANCE

The development of multidrug resistant bacteria limits the number of options for treatment of infections by pathogens that are resistant to multiple antibiotic classes. These pathogens are associated with increased morbidity, mortality, and costs within the system.[28]

New Antibiotic Development Not Working

Currently, the development of new antibiotics remains limited due to the high research costs, regulatory challenges, and lower financial incentives than with other therapeutic areas.[29]

Adverse Clinical Events from Antibiotic Use

Commonly used antibiotics have associated side effects such as gastrointestinal upset, allergic reactions, nephrotoxicity, and hepatotoxicity, so patients may not take them as prescribed, which could limit the use of the antibiotics in clinical practice.[30]

Biofilm Related Infections

The presence of bacteria within biofilms provide protection from both antibiotic treatment and immune response from the host. Medical device and implant related infections that develop due to biofilm presence are often difficult to treat.[31]

Mulitiplication of Resistance Genes

Bacteria can multiply swiftly by exchanging genetic material (via horizontal gene transfer). Resistance to antibiotics is spreading rapidly within bacteria and complicates infection control significantly.[32]

VII. DIFFRERNT TYPES OF ANTIBIOTICS :

Class of Antibiotics	Example	Mechanism of Action	Major Uses
Penicillins	Amoxicillin,Ampicillin	Inhibit bacterial cell wall synthesis	Respiration infections,UTIs,skin infections
Cephalosporins	Ceftriaxone,Cephalexin	Inhibit Cell wall synthesis	Pneumonia, meningitis,sepsis
Macrolides	Azithromycin, Erythromycin	Prevent bacteria from building proteins	Respiratory infections, atypical pneumonia
Tetracyclines	Tetracyclin,Doxycyclin	Inhibitprotine synthesis[30S Ribosomes]	Acne, cholera,rickettsial infection
Aminoglycosides	Gentamicin,streptomycin	Inhibitprotine synthesis[30S Ribosomes]	Severe Gram-negative infections
Fluoroquinolones	Ciprofloxacin,Levofloxacin	Inhibit DNA gyrase and topoisomerase IV	UTLs, Gastrointestinal infections
Glycopeptides	Vancomycin	Inhibit bacterial cell wall synthesis	MRSA infections

Table 1 : Different type of antibiotics

VIII. CONCLUSION

To Summarize One Of The Greatest Dangers In Modern Medicine Is The Growing Emergence Of Bacterial Strains That Are Resistant To Antibiotics, Known As Antibiotic Resistance. The Use Of Antibiotics Has Changed The Course Of Infectious-Related Diseases; Antisocially, The Use Of Antibiotics Has Helped To Reduce The Rate Of Many

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Infectious-Disease Related Mortality And Morbidity. However, Over The Decades, There Has Been An Increased Rate Of Development Of Resistant Bacterial Strains Due To The Overuse Of Antibiotics In Human Medicine, Agriculture, And Animal Husbandry.

Antibiotic Resistance Is The Result Of A Variety Of Mechanisms That Cause The Drug To Become Inactive Or Less Effective Than Before The Mutation Of A Bacterium Cause Antibiotics To Lose Their Therapeutic Effectiveness. The Mechanisms By Which Antibiotic Resistance Develops Include Enzymatic Inactivation By Bacteria, Altered Targets Of Antibiotic Action, Decreased Permeability Of The Bacterial Cell Membrane, And Efflux Of Antibiotics From Bacteria. In Addition To Making It Difficult To Manage Infections, The Resultant Infection Prolongations Result In Higher Costs To The Healthcare System And Higher Deaths Associated With Infections That Were Previously Easily Treated (i.e., Most Bacterial Infections).

There have been some recent developments in the area of antimicrobial research that provide potential solutions for fighting against resistant pathogens — these include advancement of novel antibiotics, combination therapy, bacteriophage therapy, antimicrobial peptides, and new technology to create methods of drug delivery. In addition, there have been numerous global initiatives that promote antibiotic stewardship programs, surveillance systems, vaccination strategies, and public awareness in order to reduce the spread of resistance (e.g., the World Health Organization).

In spite of these positive developments, there remain a number of significant challenges to overcome including the lack of developing new antibiotics, apparatus that spread resistance genes rapidly, and the continued existence of multidrug resistant pathogens. Therefore, the collection of coordinated efforts between healthcare professionals, researchers, policymakers, and the general public will be critical to not only protecting the current efficacy of existing antibiotics but also ensuring that future antimicrobial therapies continue to be successful.

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