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EXPLORING THE BACTERIAL DEFENSES AGAINST ANTIBIOTICS IN BACTERIA

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Abstract
The development of resistance in the most dangerous bacterial infections is a significant global public health concern. Antibiotic-resistant bacterial reservoirs may occur outside of hospitals due to the higher prevalence of multidrug-resistant organisms in the general population and outside of hospitals. The finest example of how bacteria have evolved and adapted is how they have responded to the antibiotic "attack." The high genetic plasticity of bacterial pathogens causes this "survival of the fittest" phenomenon by triggering specific reactions that lead to mutational adaptations, the acquisition of genetic material, or changes in gene expression that result in resistance to nearly all of the antibiotics currently used in clinical practice. Understanding the genetic and metabolic foundations of resistance is essential for preventing the emergence and spread of resistance as well as for developing innovative therapeutic strategies to address organisms that are resistant to a range of medications. This chapter will go into great detail about the primary mechanisms of antibiotic resistance that show up in clinical practice, using specific examples from relevant bacterial illnesses.
Keywords: resistance encountered, therapeutic approaches, antibiotics currently

1. INTRODUCTION

Modern medicine was transformed by the discovery, development, and broad application of antimicrobial medications to treat infections [1]. [17]. First of all, interactions between various organisms and their habitats have long been the cause of antibiotic resistance. Since the majority of antimicrobial medications are naturally occurring compounds, co-resident bacteria have developed defense mechanisms to survive [3]. [13]. According on whether Streptococcus pneumoniae isolates cause meningitis or other infections, different penicillin breakpoints have also been established [2]. These breakpoints are based on the concentrations of antibiotics that actually reach the brain fluid. In the history of medicine, antibiotics are arguably one of the most effective types of chemotherapy [4]. Antimicrobial drugs have saved many lives and made a substantial contribution to the management of infectious diseases, which were the main causes of morbidity and mortality in humans [5] [11].

Bacterial biofilms are one of a kind of bacterial lifestyle which influences and intensifies antibiotic resistance [6]. A biofilm is defined as a well-defined three-dimensional aggregation of microorganisms, single or mixed population, enclosed in a sheath of complex, self-produced extra polymeric substance (EPS) [12]. It has been reported that with global escalation of antibiotic resistance, recalcitrant nature of biofilm is playing a crucial role in intensifying resistance. Thereby, it is affecting the healthcare set up, threatening medical procedures, endangering sustainable pharmacological progress and causing an inflation of the overall economy [10]. The situation further deteriorates due to lack of new antimicrobials or its leads in clinical pipeline, lack of access to quality antimicrobials and serious antibiotic shortages affecting the entire health-care systems.

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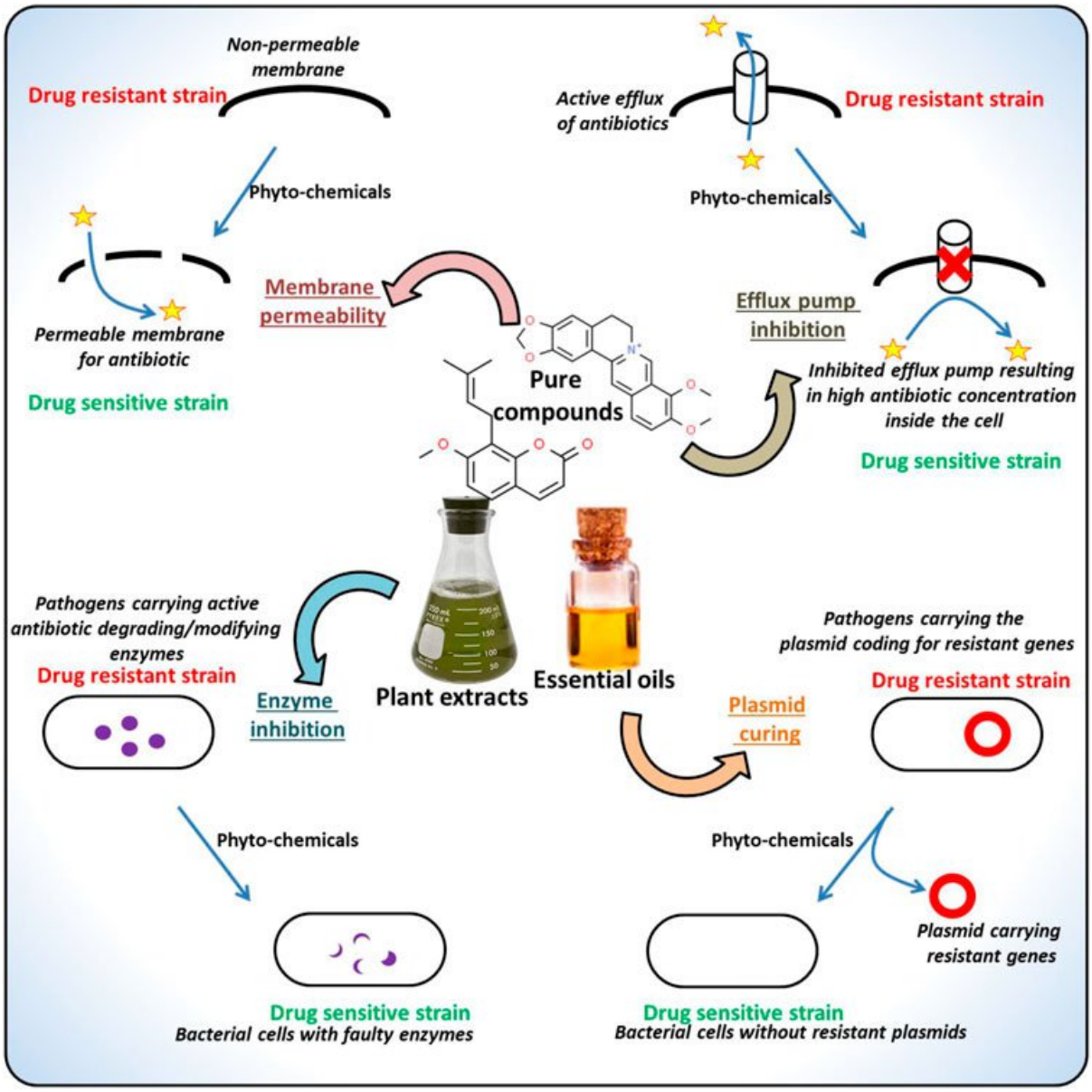

Figure 1: Antibiotic resistance in bacteria

Resistance-causing mutations only last as long as the antibiotic is present and are frequently detrimental to cell homeostasis (i.e., decreased fitness). Antimicrobial resistance mutations frequently change how antibiotics work in one of the ways listed below [14].

2. NEED OF THE STUDY

As was previously indicated, bacteria that live with toxic toxins have genetic resistance determinants built into them [8]. There is strong evidence that antibiotic resistance genes in therapeutically relevant bacteria are likely derived from these "environmental resistant" microorganisms. Moreover, resistance to numerous commonly used antibiotics has been linked to this genetic exchange [7]. High rates of conjugation, a highly effective method of gene transfer involving cell-to-cell contact, are anticipated in the gastrointestinal tract of patients receiving antibiotic treatment. It is frequently linked to the development of resistance in the hospital environment.

3. MATERIALS AND METHODS

This process was most likely developed over millions of years. Notably, resistance to a specific antibiotic class is usually produced via a number of metabolic pathways, and a single bacterial cell might employ a variety of

resistance mechanisms to withstand the effects of an antibiotic. For example, fluoroquinolone (FQ) resistance may arise from the simultaneous presence of three different metabolic pathways in the same bacteria. This can have an additional effect and often increases the resistance levels [15]. However, it appears that certain bacterial species have developed a predilection for particular resistance mechanisms. However, whereas gram-positive organisms seem to be able to produce β-lactamase under specific circumstances, they do not have this "compartmentalization" advantage.

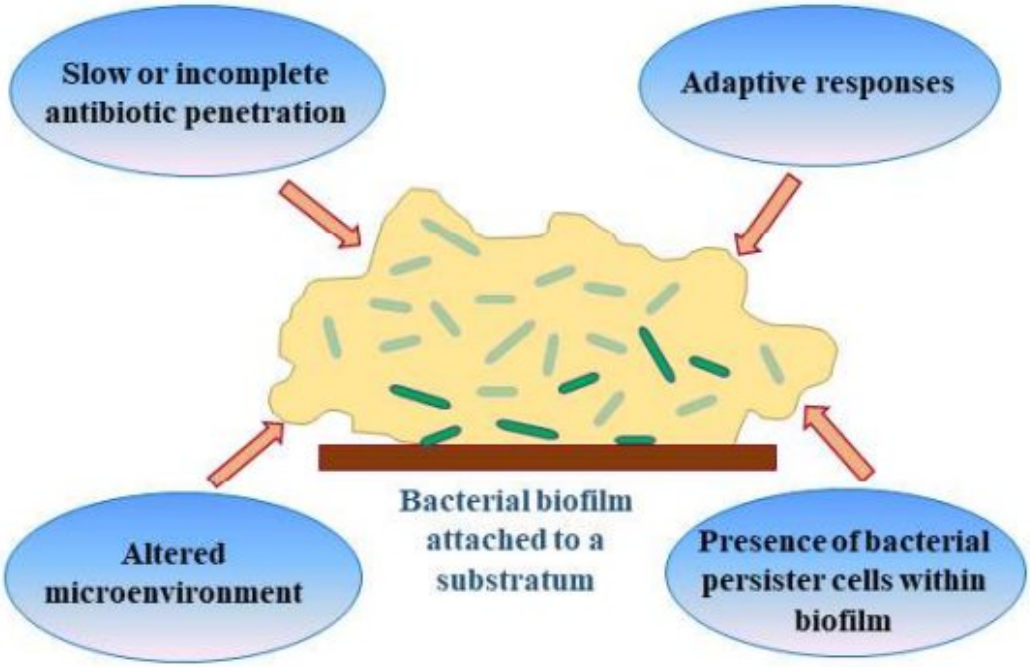

Figure 2: Major causes of antibiotic resistance of bacterial cells within a biofilm

By creating enzymes that either break down the molecule or add specific chemical moieties to the medication, bacteria can effectively fight antibiotics and stop them from reaching their intended target. AME-coding genes have been discovered on the chromosomes of certain bacterial species, such as certain aminoglycoside acetyltransferases, but these enzymes are typically located in MGEs [9].The particular aminoglycosides that these enzymes impact, the bacterial species they spread throughout, and their geographic range all exhibit significant variation.

4. RESULT AND DISCUSSION

Even within the same family, AMEs have diverse jobs and residences. For example, the genes that generate ANT are also the adenyl transferases that normally influence both gentamicin and tobramycin [16].

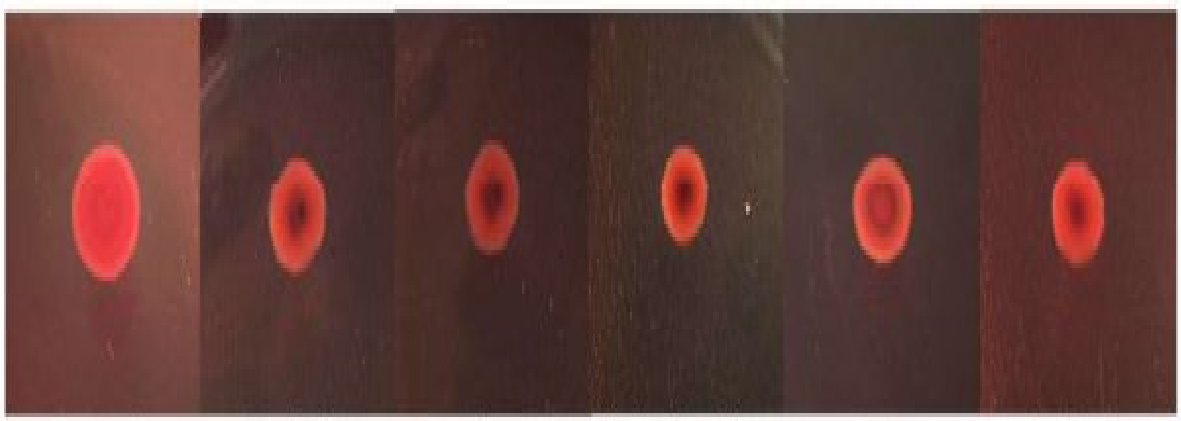

Figure 3: Effect of naringin alone and in combination with either ciprofloxacin

It's crucial to remember that the two aforementioned groups have limitations and don't entirely overlap. Furthermore, errors could result from the Ambler categorization's seeming simplicity and lack of relationship to the functional properties of the enzymes.

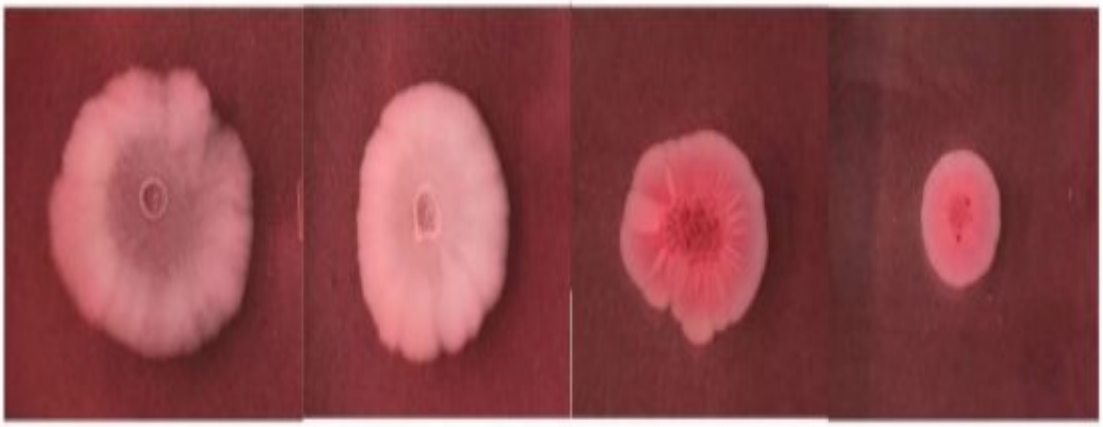

Figure 4: Effect of naringin alone and in combination with ciprofloxacin

Furthermore, enzymes that were initially categorized as belonging to a group and possessing a defined biochemical profile may develop into unique enzymes with distinct substrate specificities, typically as a result of active site mutations.

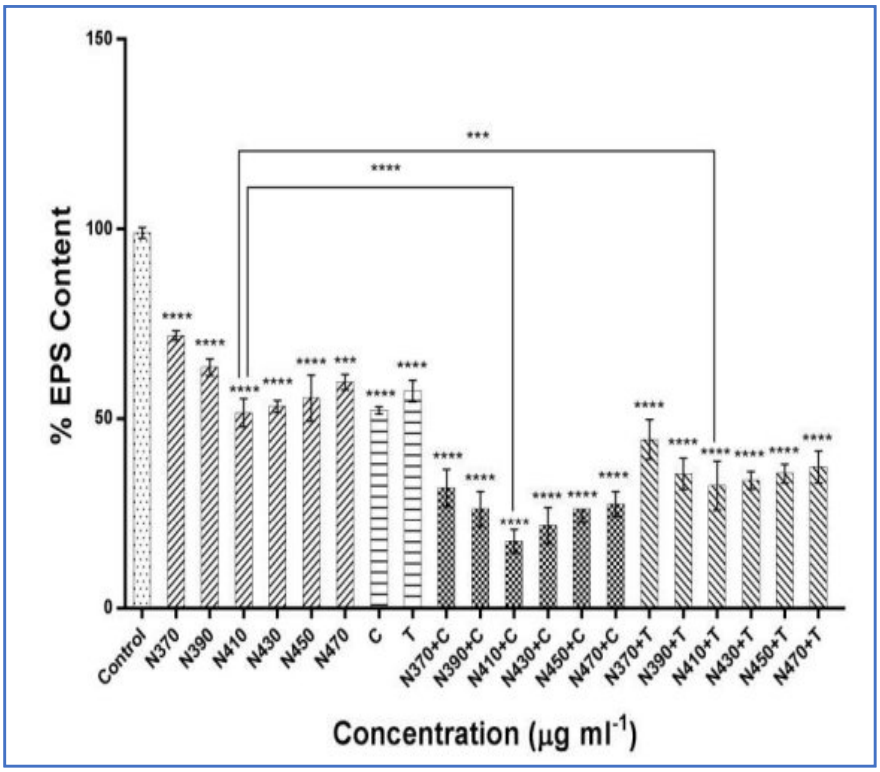

Figure 5: EPS estimation of biofilm matrix by Congo red binding assay

The appearance of NDM-1 is particularly alarming because it has been demonstrated that the blaNDM gene is easily. It swiftly expanded to numerous nations and rose to prominence as one amongst the most dreaded resistance factors in a number of global locations.

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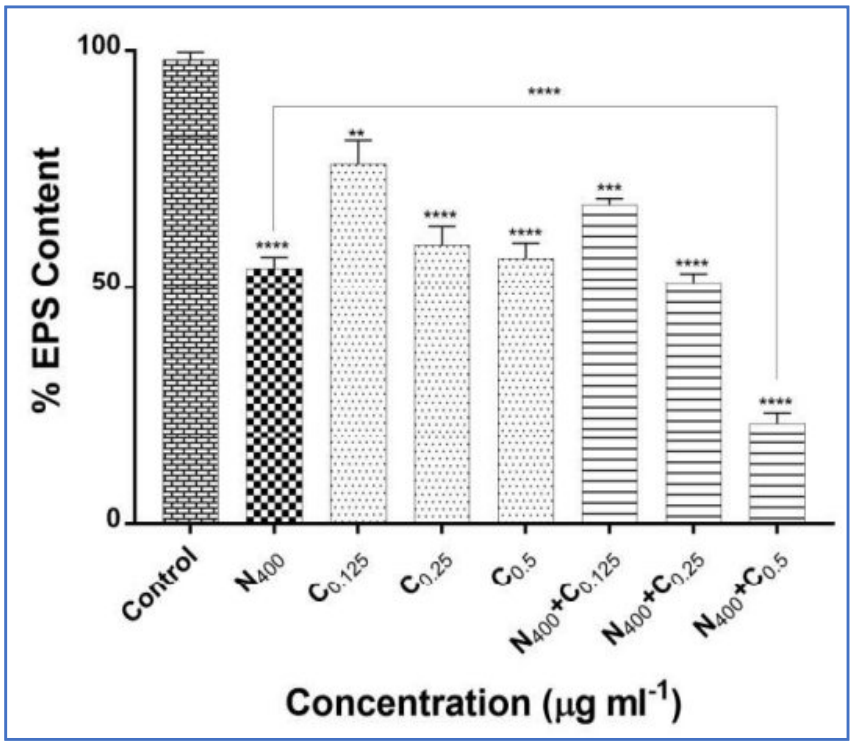

Figure 6: EPS estimation of biofilm matrix by Congo red binding assay

Furthermore, the blaNDM gene is commonly detected in isolates linked to the community in the Indian subcontinent (India and Pakistan), in addition to being widely distributed among nosocomial pathogens. Additionally, a number of studies have discovered gram-negative bacteria that produce NDM-1 in human drinking water and soil, indicating that these genes might be spreading across the human microbiota.

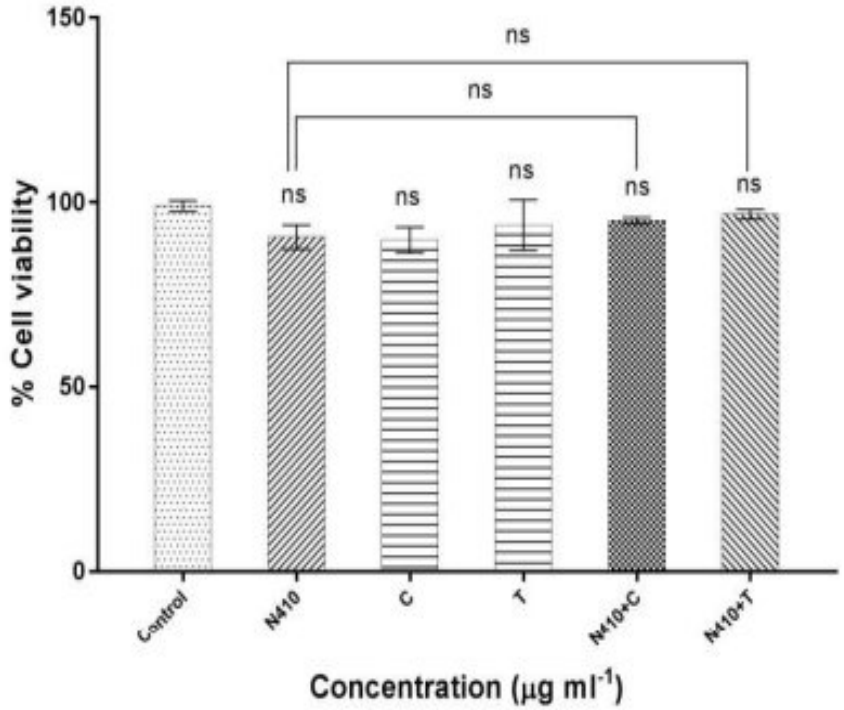

Figure 7: Cytotoxic effect of standardized combinations of naringin and antibiotics

The notion that bacteria will react to antibiotics and become resistant to them must be considered when developing new ones. Therefore, ongoing, comprehensive, and trustworthy research on antibiotic development and resistance mechanisms is essential. This "war" against creatures with a high degree of adaptability and survival is probably going to be a protracted one.

5. CONCLUSION

Because of this, sophisticated and highly developed medical techniques have been created, greatly prolonging people's lives all over the world.Antibiotic resistance, which has dramatically developed in recent decades, is one of the largest threats to public health in the twenty-first century. In fact, infections that cannot be treated because the infecting organism is resistant to many drugs have increased in frequency in clinical settings. The lack of antibiotic research and development has made this bad situation worse. Identifying new compounds became increasingly challenging, and the "golden" pipeline of antibiotic development soon dried up. The tsunami of resistance grew stronger as big pharma focused on other, more profitable, and fulfilling businesses. We must greatly expand and fund research and development efforts in order to address this problem.

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