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Mini Review

ANTIBIOTIC-RESISTANT BACTERIAL INFECTIONS: CURRENT PROMISING METHODS

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ABSTRACT

One of the most pressing issues facing global health today is infections caused by antibiotic-resistant bacteria (ARB). Antibiotic resistance can be combated in a number of promising ways, including by developing new antibiotics, sensitizing ARB, or looking for alternatives to existing antibiotics. The most promising anti-ARB strategies currently in development are compiled in this review. Antibiotics can be improved in their efficacy by metabolic stimulation or by loading a novel, more efficient delivery system. Alternative antibiotics like bacteriophages and their encoded endolysins, anti-biofilm drugs, probiotics, nanomaterials, vaccines, and antibody therapies can also be developed as alternatives to conventional antibiotics. These strategies include the following: discovery of novel antibiotics by modifying existing antibiotics; screening of small-molecule libraries; or exploration of peculiar places. These treatments have been shown to be very effective against ARB in clinical or preclinical studies. It is anticipated that some anti-ARB products will soon be available for purchase in the marketplace.

Keywords: Nanomaterials; Anti-biofilm dru; Vaccine

INTRODUCTION

Antibiotic efficacy has been compromised by the rapid emergence of antibiotic resistance in numerous clinical bacteria. In addition, antibiotic misuse and overuse have made the problem of resistance even worse. Because they were all resistant to a significant number of antibiotics that are currently on the market, the World Health Organization published a list of twelve bacteria that were cause for concern.the six most prevalent nosocomial pathogens referred to as ESKAPE) that frequently evade antibiotics lethal effects, as the Infectious Diseases Society of America (IDSA) emphasized as examples of pathogenesis, transmission, and resistance. Stewardship of antibiotics and microbes is essential to the fight against antibiotic resistance. Antibiotic stewardship has been implemented in the outpatient setting in the Netherlands and Sweden, which have the lowest rates of antibiotic resistance in Europe. Antimicrobial resistance in the subsequently identified E. coli bloodstream infections has been significantly reduced in England as a result of the decrease in antibiotic prescriptions.

DISCUSSION

According to the findings of a systematic review, antibiotic stewardship programs (ASPs) could cut down on antibiotic use, costs, treatment times, and the local rate of antibiotic resistance without having a negative impact on the mortality of ICU patients. However, there are still some restrictions that prevent the proper use of antibiotic stewardship. Physicians empirically prescribe broad-spectrum antibiotics to their patients out of concern that they won't adequately treat the pathogen that is causing the problem. This treatment typically lasts too long or covers too much ground. In addition, antibiotic stewardship is not widely applicable in low- and middle-income nations due to the patient's unwillingness to pay for hospitalization costs and the high cost of management. Mutations in antibiotic-target genes or the transfer of antibiotic-resistant genes between bacterial pathogens are the primary causes of antibiotic resistance. Antibiotic resistance may be influenced by bacterial metabolism, according to a decade of research [1,2].

Treatment with antibiotics dramatically alters the metabolic state of bacteria, which in turn affects their inherent susceptibility to antibiotic side effects. In addition, numerous studies have demonstrated that altering bacterial metabolism is a highly effective strategy for increasing antibiotic efficacy. To this end, there are two metabolism-based regulatory strategies available: enhancing metabolic pathways that make bacteria more susceptible to antibiotics and inhibiting metabolic pathways that make bacteria more resistant to antibiotics. In comparison to the wild-type strain a kanamycin-resistant strain lacked glucose and L-alanine. Through activation of the TCA cycle and enhancement of the proton-motive force, these metabolites significantly improved kanamycin uptake and toxicity. Most likely, the resistance level caused by spontaneous suppressor mutations was exceeded by the elevated intracellular concentration of kanamycin. Different studies used similar potentiation strategies and came to the same conclusion: antibiotics and a variety of metabolites from glycolysis, the TCA cycle, and amino acid metabolism could control ARB [3,4].

It was discovered through the use of phenotypic screening, metabolic network modeling, and white-box machine learning that the inhibition of intracellular adenine enhanced the activity of gentamicin, ampicillin, and ciprofloxacin. Antibiotic treatment reduces adenine levels, triggering purine biosynthesis, which raises ATP demand and prompts an accelerated metabolic rate that contributes to cell death. Understanding bacterial metabolism in terms of antibiotic efficacy is still in its infancy, despite the fact that the close relationship between bacterial cell metabolism and antibiotic resistance has frequently been utilized to alter the antibiotic susceptibility of bacteria. According to the Collins lab at the Massachusetts Institute of Technology, a comprehensive understanding of the relationship between bacterial metabolism and antibiotic function can soon be used to develop highly effective and precise antibacterial therapies that can combat numerous defense mechanisms utilized by bacteria to avoid inhibition by current antibiotics. It has been reported that there are additional antibody engineering options for fighting bacterial infections [5,6].

CONCLUSION

An antibody-antibiotic conjugate known as DSTA4637S combines a rifampicin-class antibiotic with a human IgG1 that has been engineered to specifically bind to WTA's N-acetyl-glucosamine. This conjugate is made to kill S. aureus reservoirs within cells. Phagolysosomal proteases cleave the linker between the antibody and antibiotic when DSTA4637S-opsonized S. aureus is taken up by host cells, releasing enough antibiotic to kill the bacteria. PcrV, a protein at the top of the type

III secretory system, and Psl, an extracellular polysaccharide, are the targets of the bispecific antibody MEDI13902. The clinical trial evaluating this antibody therapy's efficacy and safety in patients on mechanical ventilation is currently in phase II. In cancer immunotherapy, antibody engineering has been used extensively, but only recently for bacterial infections. Antibody therapies that look promising are expected to emerge from further research that takes into account a variety of host and bacterial factors.

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