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Infectious Diseases Caused by Bacteria and Viruses and the Challenge of Antibiotic Resistance

Rumo Bian*

Dalian Polytechnic University, Dalian, China

**Corresponding author: Rumo Bian.*

Abstract

The types of infectious diseases and the number of patients have increased significantly, posing a serious threat to global medical health. In particular, the rise in antibiotic resistance is making bacterial infections more difficult to treat. It is precisely because of the abuse of antibiotics that bacteria have rapidly developed drug resistance, especially in the case of multidrug-resistant strains, that traditional antibiotic therapy is gradually becoming ineffective. In addition, the rapid mutation of the virus also brings challenges to vaccine development. Taking SARS-CoV-2 as an example, its mutation reduces the effectiveness of the vaccine and increases the complexity of prevention and control. Bacteria and viruses have significant differences in structure and metabolism, which determine their respective treatment methods. Bacteria can be treated with antibiotics, while viruses must rely on vaccines and antiviral drugs to control them. Therefore, scientists are trying to address these challenges by studying bacterial resistance mechanisms, optimizing the composition of HPV vaccines, analyzing the impact of SARS-CoV-2 mutations, and exploring alternative treatments to antibiotics.

Keywords

antibiotics, antimicrobial resistance, vaccine optimization, infectious disease prevention

1. Introduction

The discovery and application of antibiotics was a major breakthrough in the human fight against bacterial infections. Since the beginning of the 20th century, the continuous development of antibiotics has saved countless lives. The widespread use of antibiotics has effectively suppressed the spread of many bacterial infectious diseases. From penicillin to the latest multi-antibiotics, the treatment of bacterial infections has gradually become more diverse and effective. However, with the abuse of antibiotics in clinical and agricultural settings, drug-resistant bacteria have increased rapidly, especially the spread of multidrug-resistant (MDR) and pan-drug-resistant (PDR) strains, which has brought unprecedented challenges to global public health. The formation of persistent bacteria has further complicated antibiotic treatment, reducing the effectiveness of treating bacterial infectious diseases in chronic and recurrent infections.

At the same time, the threat of viral infectious diseases remains and becomes more complex. Unlike bacteria, viruses rely on host cells to replicate and cannot be directly treated with antibiotics. The rapid mutation of viruses provides an adaptive advantage for their spread within the host, which makes the development of

vaccines and antiviral drugs more challenging. In particular, the emergence of SARS-CoV-2, whose high mutation rate of the spike protein not only affects the virus's ability to spread but also enables it to evade the protection of vaccines and antibodies to a certain extent. On the other hand, human papillomavirus (HPV) also poses a serious threat to human health. Although preventive vaccines are available, the coverage of existing vaccines is relatively limited and cannot completely prevent and control infections of multiple high-risk subtypes.

Faced with the increasingly serious problems of drug resistance and viral mutation, scientists are actively exploring new antibiotics and alternative therapies to meet the challenges posed by highly resistant bacteria. For example, a new antibiotic provides a new idea for the treatment of drug-resistant bacterial infections by inhibiting the transport of lipopolysaccharide (LPS) of Gram-negative bacteria. In addition, deep mutation scanning technology helps scientists better understand the transmission patterns of virus mutations and provides data support for vaccine optimization.

This study will integrate the history of antibiotic development, resistance mechanisms, vaccine progress, and alternative therapies in the context of bacterial and viral infectious diseases and analyze cutting-edge challenges and response strategies in infectious disease prevention and control. These explorations will not only provide new ideas for the problems caused by antibiotic resistance and viral mutations but also provide a solid scientific basis for public health policy formulation and infectious disease prevention and control.

2. Basic Differences Between Bacteria and Viruses

Bacteria and viruses are the two major types of pathogens that cause infectious diseases. Although they can both cause infection, they have significant differences in structure, physiological metabolism, replication mechanism, and treatment methods.

First, bacteria are a type of single-celled organisms that can survive and reproduce independently. They have cell walls, cell membranes, and independent metabolic systems. This ability to replicate itself allows bacteria to proliferate in an environment outside the host. The structural characteristics of bacteria make antibiotics an effective treatment. Antibiotics can target bacterial cell walls, protein steps, and DNA replication processes to inhibit or kill bacteria. However, with the abuse of antibiotics and the increase of natural selection pressure on bacteria, bacteria have developed multidrug resistance (MDR) and pan-drug resistance (PDR), increasing the complexity of treatment [1].

Viruses are different from bacteria. They do not have a complete cell structure and cannot metabolize and replicate independently. They must rely on the biological mechanisms of host cells. Viruses are composed of nucleic acids (DNA or RNA) and protein shells. After entering the host cell, they hijack the host's synthetic system for protein synthesis and gene replication. Because viral replication is completely dependent on host cells, antibiotics are not effective against viral infections. Instead, antiviral treatments usually focus on preventing the virus from entering host cells or inhibiting the viral replication steps.

In addition, although antibiotics are still the mainstay of treatment for bacterial infections, traditional antibiotics have limited effectiveness against highly resistant pathogens such as Gram-negative bacteria [2]. The fundamental differences in structure and biological characteristics between bacteria and viruses determine that they require different treatment strategies. Bacterial infections can usually be directly inhibited by antibiotics, while viral infections rely more on vaccine prevention and antiviral drug intervention. In addition, bacterial resistance and viral mutation further increase the complexity of prevention and treatment, and scientists are seeking new ways to deal with the different threats of these two types of pathogens.

3. The Formation Mechanism of Persistent Bacteria and Its Clinical Impact

3.1 The Formation Mechanism of Persistent Bacteria

Persister cells are special subpopulations of bacteria that can survive antibiotic treatment. Persisters are usually in a dormant state with very low metabolic activity and are therefore insensitive to the effects of antibiotics. The formation of persisters is closely related to environmental stresses, such as nutrient limitation, pH changes, and oxidative stress, which can induce bacteria to enter a state of slow metabolism, thereby

avoiding being killed by antibiotics [3]. In complex infection environments (such as biofilms or inside host cells), the proportion of persistent bacteria tends to increase, thereby enhancing tolerance to infection. Although the specific mechanism is not yet fully understood, studies have shown that the bacterial toxin-antitoxin system and stress response pathways may be involved in the formation of persistent bacteria [4].

3.2 Clinical Impact and Treatment Challenges

Persistent bacteria play an important role in chronic and recurrent infections. Their ability to survive antibiotic treatment makes them one of the main causes of recurrent infections. Failed root canal treatment is a typical example. In the treatment of endodontic infection, persistent bacteria can survive the treatment process and repopulate after the treatment is completed, resulting in recurrent infection. Furthermore, in chronic infections such as tuberculosis, the presence of persisters results in prolonged antibiotic treatment, as it takes longer to completely eliminate the infection [5].

The presence of persisters not only affects the treatment outcomes of individual patients but also poses a threat to public health. Although persisters do not have genetic resistance, the persistence of persister populations under antibiotic pressure increases the likelihood of developing resistance mutations, which in turn promotes the spread of resistant strains. To address the challenge of persisters, researchers are exploring new therapeutic strategies, such as targeting metabolic pathways unique to persisters or using toxin-antitoxin modules to inhibit their formation and activity recovery. Although these approaches have shown potential in experimental studies, the complexity of persisters in clinical infections remains an issue that needs to be addressed [6].

3.3 Viral Vaccine Development and Mutation Challenges

The high infectivity and rapid mutation of the virus make vaccine development a key means of controlling the spread of the virus. In recent years, scientists have effectively prevented virus-related diseases such as cervical cancer caused by HPV by developing vaccines targeting specific virus antigens. However, although current preventive vaccines have achieved remarkable results in reducing infection rates and cancer incidence, vaccines are still limited in coverage and long-term effectiveness. In particular, the weakening of vaccine effectiveness by viral mutations has made the need to update and optimize vaccine components to cope with mutations more urgent.

As research on HPV vaccines and SARS-CoV-2 vaccines progresses, we have seen different challenges faced by the two viruses in vaccine development. HPV vaccines are effective in preventing cancer, but their coverage of high-risk subtypes is limited, making it difficult to provide comprehensive protection, and therapeutic vaccines are being explored. As for SARS-CoV-2, frequent mutations make the effectiveness of vaccines uncertain, and continuous updating and broad-spectrum optimization of vaccines are essential. The following will discuss in detail the research progress of HPV vaccines and the challenges that SARS-CoV-2 mutations pose to vaccine development.

4. Research Progress of HPV Vaccines

The development of human papillomavirus (HPV) vaccines has significantly reduced the incidence of HPV infection and related cancers. Currently, the main approved HPV vaccines are preventive HPV vaccines, such as Cervarix®, Gardasil®, and Gardasil 9®, which prevent new infections by generating neutralizing antibodies against the viral L1 protein [8]. The vaccines have shown high efficacy in protecting against high-risk HPV subtypes, such as HPV-16 and HPV-18, which cause approximately 70% of cervical cancer cases [9].

Although these vaccines are highly effective in preventing HPV infection, they are limited in their effectiveness in clearing established infections and associated lesions. This is because preventive vaccines induce antibody responses primarily through virus-like particles (VLPs) targeting the L1 protein, which is not expressed in infected cells and therefore cannot clear existing HPV infection [10]. To improve the prevention effect, researchers are increasing the price of the vaccine, such as expanding Gardasil 9® to nine-valent, to cover more high-risk subtypes, thereby further reducing the risk of cancer caused by HPV infection [8].

In addition, to make up for the limitations of preventive vaccines, researchers are exploring therapeutic HPV vaccines, which aim to induce cell-mediated immune responses to clear infected cells. HPV E6 and E7

early proteins are continuously expressed in HPV-related cancers, making them ideal targets for therapeutic vaccines. Studies have shown that therapeutic vaccines can activate specific CD8+ cytotoxic T cells through E6 and E7 antigens, thereby enhancing the immune system's ability to clear infected cells [9].

With the advancement of technology, HPV vaccine research and development is still optimizing dosage regimens and adjuvant formulations. For example, by adding new adjuvants such as TLR4 agonists, the immunogenicity of the vaccine can be improved [8].

These optimizations not only enhance the protective efficacy in terms of antibody levels but also extend the duration of protection to a certain extent, making the vaccine more feasible and economical in low-income areas.

In summary, the research and development of HPV vaccines is moving towards a broader spectrum of preventive and effective therapeutic vaccines. This progress is of great significance in reducing the global burden of HPV infection-related diseases. Future research will continue to explore the optimization of multivalent vaccines and the clinical application of new therapeutic vaccines [10].

5. SARS-CoV-2 Mutations and Immune Escape

Mutations in SARS-CoV-2, especially those in the spike protein (S protein), have significantly enhanced the virus's ability to infect and evade immunity. The receptor binding domain (RBD) of the S protein mediates viral entry into cells by binding to the host cell's ACE2 receptor, and mutations in this region enable the virus to increase its infection efficiency and evade the host's immune response [11]. The early D614G mutation increased the virus's infectivity, making it quickly become one of the major SARS-CoV-2 variants worldwide. Subsequently, various “variants of concern” (VOCs) emerged one after another, such as Alpha, Beta, Delta, and Omicron. The mutations of these variants are mainly concentrated in the RBD region of the S protein [11]. Among them, the Omicron variant has more than 15 RBD mutations and variations in other antigenic domains, resulting in its strong immune escape ability against early vaccine-induced antibodies and antibodies produced by natural infection [12]. Omicron's RBD mutations, such as E484K, L452R, and N501Y, help the virus increase ACE2 binding affinity and weaken the effect of neutralizing antibodies, thereby posing a risk of reinfection in previously infected and vaccinated individuals [13].

Immune escape is a critical factor in the virus's ability to spread, especially when vaccination rates are increased, as the speed at which the virus mutates to adapt to immune pressure is significantly accelerated. Literature indicates that immune escape mutations such as E484K, K417N, and N501Y show significant neutralization resistance in the face of vaccine-induced antibodies, thereby reducing the protective efficacy of the vaccine [11]. In particular, mutations in Omicron and its sublineages (such as BA.1, BA.5, etc.) enable rapid spread among vaccinated or previously infected populations, leading to multiple waves of infection [7].

As these mutations accumulate, scientists have raised the need to develop broad-spectrum vaccines or update vaccine components to improve protection against different SARS-CoV-2 variants. The challenges posed by immune escape mutations not only place higher demands on vaccine development but also have a profound impact on the adjustment of public health strategies, especially those involving the effectiveness monitoring of existing vaccines and strategies for booster shots.

6. Challenges of Antibiotic Resistance and Countermeasures

6.1 Antibiotic Mechanisms of Action and the Development of Resistance

Antibiotics act on bacteria through a variety of mechanisms to inhibit their growth and proliferation. The main mechanisms of action include inhibiting cell wall synthesis, interfering with protein synthesis, destroying cell membranes, and inhibiting nucleic acid synthesis [14]. For example, β-lactam antibiotics (such as penicillins and cephalosporins) bind to bacterial penicillin-binding proteins (PBPs), preventing cell wall formation and leading to bacterial cell lysis. Aminoglycoside antibiotics inhibit bacterial growth by binding to the 30S subunit of bacterial ribosomes, causing errors in protein synthesis [15].

However, bacteria have developed a range of resistance mechanisms that have made antibiotic therapy less effective. The development of resistance has been attributed to several mechanisms: enzyme-mediated

inactivation of antibiotics (e.g., β-lactamases break down β-lactam antibiotics), alteration of drug targets (e.g., methicillin-resistant Staphylococcus aureus alters its PBPs to resist β-lactam antibiotics), efflux pumps (pumping antibiotics out of bacterial cells to reduce intracellular concentrations), and reduced uptake of antibiotics [14][15][16].

For example, Gram-negative bacteria restrict drug penetration through outer membrane proteins and remove antibiotics through efflux pumps, thereby increasing resistance to multiple antibiotics [17]. New research has found that new antibiotics targeting the lipopolysaccharide (LPS) transport pathway of Gram-negative bacteria, such as Zosurabalpin, can destroy the bacterial outer membrane by blocking LPS transport, showing potential for application in combating drug-resistant Gram-negative bacteria.

In addition, the emergence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) bacterial strains has further limited treatment options. For example, the diversity and combination of resistance mechanisms allow some pathogens to resist the effects of antibiotics through multiple pathways, including mutation and horizontal gene transfer [16]. Understanding these mechanisms could help develop more effective antimicrobial strategies and advance the development of new antibiotics and antimicrobial agents to extend the useful life of existing antibiotics.

6.2 Research on New Antibiotics and Alternative Therapies

As antibiotic resistance increases, researchers are actively exploring new antibiotics and alternative treatments to combat multidrug-resistant bacterial infections. The development of new antibiotics focuses on overcoming the challenges of traditional antibiotic failure, especially resistance to Gram-negative bacteria. In recent studies, new antibiotics such as Zosurabalpin significantly inhibit bacterial growth by blocking the lipopolysaccharide transport pathway of Gram-negative bacteria, providing a new approach to solving Gram-negative bacterial resistance [14].

In addition to new antibiotics, alternative therapies are also seen as an important response to antibiotic resistance. Phage therapy uses natural phages to specifically kill bacteria. Unlike broad-spectrum antibiotics, this therapy does not destroy the host's beneficial flora [18]. Although phage therapy has shown potential in treating infections, its efficacy and safety in large-scale applications still need to be further verified. At the same time, antimicrobial peptides have become a hot topic of research due to their broad antimicrobial activity. These peptides can destroy bacterial cell membranes, fight against multiple drug-resistant strains, and have relatively low risks in terms of toxicity and drug resistance [19].

In addition, microbial regulation methods such as probiotics and fecal microbiota transplantation are also used to restore or rebuild the body's normal flora, thereby indirectly reducing the colonization and infection opportunities of pathogens. These methods not only help reduce the occurrence of infections but also show potential clinical benefits in alleviating flora imbalance caused by antibiotic use [19]. For example, studies have shown that specific probiotic strains can effectively inhibit diarrhea caused by Clostridium difficile and improve the balance of intestinal microecology after antibiotic treatment.

The development of new antibiotics and alternative therapies provides new avenues for combating drug-resistant bacteria. Future studies will continue to evaluate the safety and clinical efficacy of these approaches to provide more comprehensive treatment strategies to address the global challenge of antibiotic resistance.

7. Response to Antibiotic Resistance and Future Development

The increasing problem of antibiotic resistance has posed a serious challenge to the global health system. Due to the widespread use of antibiotics in medicine and agriculture, bacteria have gradually evolved a variety of resistance mechanisms, including efflux pumps, enzyme degradation, and target modification, leading to the emergence of multidrug-resistant bacteria (MDR) and pan-drug-resistant bacteria (PDR), which has sharply reduced the effectiveness of traditional antibiotics [20]. Antibiotic resistance is expected to be a major cause of around 10 million deaths per year worldwide by 2050 [21].

To address this urgent problem, researchers are exploring multi-level strategies, from the genetic level and ecological control to new drug development, to curb the spread of drug resistance and restore the effectiveness of antibiotics. Advances in genomics and synthetic biology have brought new insights into the mechanisms of

drug resistance gene transmission. Through the gene editing tools of the CRISPR-Cas system, scientists can target and knock out drug resistance genes, thereby reversing the resistance of drug-resistant bacteria or enhancing their sensitivity to antimicrobial drugs [22]. This technology shows great potential in controlling pathogen resistance and is expected to provide an effective solution for future infection control.

Alternative therapies are also gaining attention in the treatment of drug resistance. For example, antimicrobial peptides exert broad-spectrum antibacterial activity by destroying bacterial cell membranes, and they are not prone to induce drug resistance, making them a powerful supplement for fighting drug-resistant pathogens [23]. Phage therapy uses the high specificity of phage viruses to target and kill bacteria without affecting the host's normal flora. This specificity and low side effects give phage therapy a unique advantage in treating drug-resistant bacterial infections. In addition, microecological therapies such as probiotics and fecal microbiota transplantation indirectly inhibit the growth of pathogens by regulating the host flora. These therapies have shown potential benefits in reducing the occurrence of infections and alleviating bacterial imbalance caused by antibiotics [22].

At the same time, the development of rapid diagnosis and intelligent detection technologies has made the monitoring of antibiotic resistance more accurate and efficient. For example, rapid diagnostic tools based on genome detection (such as CRISPR-biosensors) can detect the presence of resistant bacteria within hours, providing clinicians with fast and accurate resistance detection results. New food and water source resistant bacteria monitoring technologies (such as QuEChERS and fluorescent biosensors) have played an important role in reducing the risk of resistant bacteria spreading in the environment [21].

In the future, the innovative development of antibiotics will continue to integrate the results of genomics, alternative therapies, microecological therapies, and rapid detection technologies to build a multi-level, coordinated, and efficient treatment and control system. The comprehensive strategy to address antibiotic resistance will provide solid support for the global health system and is expected to significantly reduce resistance-related infections and mortality in the future.

8. Conclusion

Although significant progress has been made in the study of antibiotic resistance in recent years, there are still many gaps and deficiencies. First, the development of broad-spectrum antibiotics for multidrug-resistant bacteria (MDR) and pan-drug-resistant bacteria (PDR) is still lagging, and many new antibiotics have limited performance in dealing with complex infections and are prone to new drug resistance. Secondly, although the application of alternative therapies such as phage therapy and antimicrobial peptides has shown potential, their efficacy, safety, and standardized operation in chronic or multidrug-resistant infections are not yet mature. In addition, despite the increasing advancement of rapid diagnostic technology, the demand for low cost and portability has not been fully met, hindering its widespread application worldwide. By addressing current research gaps and strengthening multi-level antimicrobial strategies, we are expected to effectively curb the spread of antibiotic resistance, thereby providing more comprehensive protection for global public health security.

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