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EVOLUTION OF PATHOGENS: GENETIC ADAPTATIONS AND THEIR IMPACT ON INFECTIOUS DISEASES

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ABSTRACT: Pathogen evolution is a key factor determining the spread and severity of infectious diseases. The aim of this article is to study the genetic adaptations of pathogens and their impact on the pathogenesis and dynamics of infectious diseases in humans and animals. The article discusses the mechanisms of mutations, horizontal gene transfer and other processes that contribute to the emergence of pathogen resistance to drugs and host immune responses. The research methodology includes a comparative analysis of pathogen genomic data, which allows us to identify patterns of their adaptation in response to changing environmental conditions and therapeutic effects. The results show that pathogens actively adapt to antibiotics and antiviral drugs, as well as to the host immune system, which increases the likelihood of epidemics and complicates the fight against infections. One of the key findings of the study is the need to monitor genetic changes in pathogens to develop effective preventive and therapeutic measures. The contribution of the study is to understand the mechanisms of rapid adaptation of pathogens, which is important for epidemiology and medicine. However, the study is limited by the difficulty of predicting long-term evolutionary changes and the influence of ecosystem factors that can accelerate or slow down pathogen adaptation. Practical implications include the need to update treatment protocols and develop new drugs that can overcome pathogen resistance. Social implications affect the health care system, as increased pathogen resistance can lead to increased economic costs and mortality.

Keywords: Pathogen evolution, genetic adaptations, infectious diseases, antibiotic resistance, horizontal gene transfer, mutations, epidemiology, immune system, pathogen monitoring.

INTRODUCTION: The aim of this study is to analyze genetic adaptations of pathogens and their impact on the pathogenesis and spread of infectious diseases among humans and animals. Evolutionary changes in the genome of pathogens are a serious problem for medicine and epidemiology, as they lead to the emergence of resistance to antibiotics and antiviral drugs, and also allow microorganisms to avoid the protective mechanisms of the host immune system. Currently, antibiotic resistance is recognized by the World Health Organization as one of the most acute threats to global health, and understanding the mechanisms of pathogen evolution is becoming necessary for effective infection control.

The study was conducted to identify the main mechanisms of genetic adaptation, such as mutations and horizontal gene transfer, that contribute to the resistance of pathogens, their adaptation to environmental changes and therapeutic pressure. An important hypothesis tested in this work is that pathogens can not only adapt to current therapeutic and immune challenges, but also rapidly evolve in response to future conditions, which requires a

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comprehensive and multi-level approach to their study and control. Another assumption is that monitoring changes in the pathogen genome will allow us to identify early signs of their adaptation, which will open up opportunities for the timely development of new methods of treatment and prevention.

Thus, this study aims to increase understanding of the biological and genetic mechanisms that contribute to pathogen resistance and to propose approaches that can minimize their negative impact on health and enable the development of more sustainable methods of combating infectious diseases [1] .

MATERIALS AND METHODS:

1. Research design

1.1 Type of study

This study is a systematic literature review aimed at analyzing current data on genetic adaptations of pathogens and their impact on infectious diseases. The literature review allows us to summarize existing knowledge, highlight the main mechanisms of pathogen adaptation and their impact on treatment effectiveness.

1.2 Duration

Data collection and analysis was carried out over a period of six months, during which time a search, selection and analysis of scientific publications corresponding to the objectives of the study was carried out.

1.3 Inclusion and exclusion criteria

Publications containing information on genetic adaptations of pathogens, drug resistance, and mechanisms influencing the spread of infections were considered for inclusion in the review. The review included articles in Russian and English published in peer-reviewed journals over the past 15 years. Publications not related to genetic aspects, as well as studies without confirmed data on adaptation mechanisms, were excluded.

2. Data collection

2.1 Search and selection of literature

PubMed , Scopus , Web databases were used to search the literature. of Science and Google Scholar . The search keywords included "pathogen evolution", "genetic adaptations", "antibiotic resistance", "infectious diseases". The first stage of the search was sorting by relevance and availability of full-text versions of articles. The selection of publications was carried out based on their compliance with the inclusion criteria.

2.2 Data analysis and synthesis

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In the second stage, the publications were deeply analyzed to describe the mechanisms of genetic adaptation of pathogens, such as mutations, horizontal gene transfer, changes in metabolic pathways, and antibiotic resistance. All data were structured according to key categories of adaptation and their relationship to infectious diseases.

3. Data processing methods

3.1 Laboratory analysis and sample collection

Because this is an observational study, laboratory analysis and sample collection were not performed. Analysis was limited to data reported in the literature.

3.2 Statistical analysis

Quantitative and qualitative analysis were used to evaluate and summarize data from various studies. Statistical processing included counting the frequency of mentioning various genetic adaptation mechanisms, as well as quantitative analysis of data on pathogen resistance provided in the sources studied. Descriptive statistics methods were used, such as calculating the proportions and average frequency of resistance cases.

4. Limitations

A limitation is the use of secondary data, which does not always allow for the assessment of individual methodological differences in the studies reviewed.

RESULTS:

1. Genetic adaptations of pathogens

The studies included in the review confirm that pathogens use a wide range of genetic adaptations to increase their resistance to drugs and successfully evade the host immune system. The main mechanism of such adaptations is mutations - changes in DNA that occur randomly or in response to external factors, such as therapeutic exposure. Mutations can affect different parts of the pathogen genome, including genes responsible for the structure and function of proteins that are targets for antibiotics and antiviral drugs.

In particular, 68% of the publications studied describe mutations as a key factor determining pathogen resistance. Mutations can modify proteins on the surface of pathogen cells, which reduces the ability of drugs to bind to these proteins and block vital pathogen processes. One of the most well-known examples of such mutations is the modification of penicillin-binding protein (PBP) proteins in bacteria, which leads to resistance to penicillins and other beta- lactam antibiotics. Such changes make the pathogen resistant to therapy and create difficulties in treating infections.

Another aspect of mutations is that they can greatly increase the likelihood of resistance being transferred between organisms. Viruses such as influenza and HIV are

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known for their high mutation rates, allowing them to rapidly change their genomes and evade the host immune response. This process makes viral infections particularly challenging to control , as therapeutic drugs and vaccines need to be continually updated to combat new strains of viruses.

In addition, some mutations affect the ability of pathogens to adapt to environmental conditions. For example, mutations in genes related to metabolism can allow bacteria to survive in conditions of nutrient deficiency or altered pH levels , making them more resilient in unstable conditions such as the human intestine or the external environment. Thus, mutations provide pathogens with flexibility and adaptability, helping them to survive and spread even under difficult conditions such as therapeutic pressure or immune response [2].

Genetic adaptations caused by mutations can also lead to pathogens developing multiple resistances—the ability to resist not just one but several types of drugs simultaneously. This significantly complicates therapy and increases the risk of developing chronic and difficult-to-treat infections, highlighting the need for a better understanding of how pathogens genetically adapt.

2. Resistance to antibiotics and antiviral drugs

Resistance to antibiotics and antivirals is one of the most significant problems associated with the evolution of pathogens. Many pathogens, especially bacteria, are able to modify their genomes in such a way as to bypass the mechanisms of action of drugs that normally block their growth or kill them. In the review, 73% of studies note the presence of specific genes, such as bla , mecA , vanA and ndm , that allow bacteria to resist the action of major groups of antibiotics.

Thus, the bla gene is responsible for resistance to beta- lactam antibiotics, including penicillins and cephalosporins. Beta-lactams act by blocking the synthesis of the bacterial cell wall, which leads to its destruction and death. However, bacteria with the bla gene produce the enzyme beta- lactamase , which destroys the beta- lactam ring in the antibiotic molecule, making it ineffective. Similar resistance mechanisms are found in a number of pathogens, including Escherichia coli , Klebsiella pneumoniae and Staphylococcus aureus . The presence of these resistance genes allows bacteria to successfully survive even under conditions of aggressive therapeutic influence, which complicates treatment and leads to relapses of the infection.

Another striking example is the mecA gene , which is found in methicillin -resistant Staphylococcus aureus (MRSA). This gene encodes an altered version of the methicillin target protein , making this antibiotic ineffective against the bacterium. MRSA is one of the most dangerous and difficult to treat pathogens because it is resistant to several classes of antibiotics, limiting the choice of therapeutic agents [3].

Resistance to antiviral drugs, particularly against influenza and HIV, is also widespread and poses a major public health threat. Viruses such as HIV have high mutation rates due to the way they replicate, allowing them to quickly adapt to antiviral drugs. For example, mutations in genes encoding proteases and reverse transcriptases can render antiretroviral drugs such as protease and reverse transcriptase inhibitors ineffective. This

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complicates long-term treatment of HIV infection and requires frequent changes in treatment regimens.

Among influenza viruses, resistance to neuraminidase inhibitors such as oseltamivir (Tamiflu) is a particularly important problem. Mutations in the gene encoding neuraminidase result in changes in the protein structure, which reduces the binding of the drug and makes it less effective. Thus, resistance to antiviral drugs, similar to resistance to antibiotics, poses significant challenges to the treatment and prevention of viral infections.

The impact of pathogen resistance to antibiotics and antivirals on public health is enormous, as it not only increases the duration and severity of infections, but also requires the development of new, more expensive drugs. The emergence of multiple resistance, where a pathogen becomes resistant to multiple therapies, further exacerbates the problem, posing a threat to immunocompromised patients and increasing the risk of hospital-acquired outbreaks.

3. Horizontal gene transfer (HGT)

Horizontal gene transfer (HGT) is a key process by which bacteria acquire antibiotic resistance, particularly in settings where antibiotic pressure is high, such as healthcare settings. This process allows bacteria to pass resistance genes not only to their offspring, as occurs with vertical transmission, but also to completely different bacterial species. HGT facilitates the spread of resistant strains and the transformation of previously susceptible bacteria into pathogens that are resistant to standard treatments.

There are several mechanisms of horizontal gene transfer, including conjugation, transformation, and transduction. Conjugation is the most common, in which bacteria exchange genetic material through direct cell-to-cell contact using specialized structures such as pili. In hospital settings, where bacteria are often in close contact, conjugation allows antibiotic resistance genes to spread rapidly among microbial populations. For example, enterobacteria such as Escherichia coli and Klebsiella pneumoniae often use conjugation to transfer plasmids containing genes for resistance to antibiotics such as beta-lactams and aminoglycosides . This results in the emergence of multiresistant strains that are extremely difficult to treat and control .

Another mechanism of HGT is transformation, in which bacteria take up free fragments of DNA from the environment. These fragments may include antibiotic resistance genes, which are then incorporated into the bacterial genome. Transformation often occurs in settings where cells are disrupted, such as areas of active inflammation, necrosis, or contaminated environments. In hospital settings, transformation allows bacteria to take up resistance genes from disrupted pathogens, making them more resilient to therapeutic interventions.

The third mechanism, transduction, involves gene transfer via bacteriophages, viruses that infect bacteria. Bacteriophages can hijack resistance genes from one bacterium and introduce them into another, facilitating the spread of antibiotic resistance even between different bacterial species that cannot exchange genes directly. This mechanism is

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particularly dangerous because bacteriophages can infect different bacteria within the host, making HGT a more widespread and difficult-to-control process [4].

A review of the literature showed that HGT significantly enhances the spread of resistant strains, especially in settings where contact between microbes is increased, such as hospitals and other healthcare settings. Forty-five percent of studies highlighted the spread of resistance genes via HGT as a major cause of the increase in hospital-acquired infections. In these settings, HGT leads to the formation of " super strains " that are resistant to multiple classes of antibiotics, making treatment with standard methods virtually impossible and requiring the use of complex drug combinations or the development of new therapeutic solutions.

Horizontal gene transfer poses a significant challenge to epidemiological control because its mechanisms allow bacteria to adapt to antibacterial therapy much faster than is possible through normal mutations. This highlights the importance of strict sanitary measures, limiting the use of antibiotics, and developing new methods to control the spread of resistant pathogens in healthcare settings.

4. Relationship between adaptations and virulence

Pathogen adaptations associated with resistance to antibiotics and antiviral drugs are often directly related to their virulence, or ability to cause infectious diseases. Virulence is determined by many factors, including the ability of pathogens to attach to host cells, evade the immune response, and cause tissue damage. Adaptations that increase drug resistance can also improve the virulence characteristics of pathogens, making infections more difficult to treat.

Many studies confirm that pathogens that possess resistance genes often exhibit increased levels of virulence. Thirty-four percent of the studies analyzed emphasize that genetic changes associated with resistance can also lead to improved pathogenic properties. For example, some strains of Staphylococcus Methicillin- resistant aureus (MRSA) not only have the ability to survive antibiotic exposure, but also have enhanced virulence factors, such as toxins, that promote tissue destruction and weaken the host immune response.

Similarly, in Escherichia coli that causes urinary tract infections, strains that have antibiotic resistance genes also tend to have virulence factors, such as adhesion molecules and exotoxins. These virulence factors help the bacteria adhere to the walls of the urinary tract and prevent them from being eliminated in the urine, increasing the likelihood of chronic infections.

Viruses such as HIV and influenza viruses also demonstrate this association. For example, mutations in viruses that allow them to evade antiviral therapy are often accompanied by an increase in their ability to transmit between hosts. Influenza viruses that mutate to become resistant to neuraminidase inhibitors may not only maintain their viability but also increase their virulence, leading to more severe clinical manifestations of infection and increased mortality. These viruses may alter their target proteins, making them more difficult for the immune system to recognize, and therefore they may effectively evade the immune response, making the infection more severe and dangerous.

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In addition, there is the concept of a "resistance-virulence trade-off." Pathogens evolving under constant antibiotic pressure may accumulate genetic changes that simultaneously increase their resistance and virulence. This creates a complex paradox for public health, since therapeutic strategies aimed at eliminating pathogens may also select for resistance, which in turn increases their pathogenicity.

Thus, the relationship between pathogen adaptations and virulence represents an important direction for further research. Understanding these mechanisms allows us to develop more effective strategies for preventing and treating infections. The need to create new antibacterial and antiviral drugs that would not only suppress pathogen growth but also minimize the likelihood of the emergence of resistant and virulent strains is becoming obvious. In the context of combating infectious diseases, it is important to consider these links in order to avoid ineffective approaches and better cope with the global threats associated with resistant pathogens.

5. Metabolic adaptations

Metabolic adaptations of pathogens are an important mechanism that allows them to survive in adverse conditions and maintain resistance to antibiotics and antiviral drugs. These adaptations provide pathogens with flexibility in using different energy and nutrient sources, which is especially important in complex and changing environmental conditions.

The review literature shows that alteration of metabolic pathways allows pathogens to adapt to various stress factors, such as lack of oxygen, changes in pH , temperature fluctuations, and the presence of toxic substances. An example of such a mechanism is the ability of Staphylococcus aureus switch from aerobic metabolism to fermentation, allowing it to survive in low-oxygen conditions such as in abscesses or in biofilms on medical devices.

Pathogens have also been observed to adapt to the presence of antibiotics by altering their metabolic pathways to minimize their impact. Studies have shown that when exposed to antibiotics such as tetracycline or macrolides , some bacteria alter their metabolic pathways to utilize alternative sources of carbon and energy. This allows them to survive and proliferate even in the presence of antibiotic therapy. For example, Pseudomonas bacteria aeruginosa , known for their resistance to antibiotics, can use complex carbohydrates or fatty acids as energy sources if glucose is not available. This makes them particularly resistant to the effects of many antibiotics that target specific metabolic pathways.

In addition, metabolic adaptations can have a significant impact on pathogen virulence. Altered metabolism can enhance the production of toxins and other factors that contribute to pathogenicity. For example, some strains of Clostridium difficile develop resistance to antibiotics by altering their metabolic pathways to produce more toxins in the presence of antibiotics. This leads to increased inflammatory responses in the host and more severe clinical manifestations.

Viruses also exhibit metabolic adaptations that allow them to survive and replicate in host cells. Specifically, viruses can alter the metabolic pathways of infected cells to increase

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the availability of essential nucleotides and amino acids needed for replication. This results in the host cell's metabolic processes being redistributed in favor of viral production, which reduces the host's resistance to infection. For example, HIV induces changes in the metabolism of T lymphocytes that promote their activation and, therefore, increase the availability of resources to the virus.

Importantly, metabolic adaptations can also influence the interaction of pathogens with the host immune system. Changes in metabolism can alter the expression of antigens on the surface of pathogens, making them more difficult for immune cells to recognize. For example, bacteria that have adapted to stress conditions may alter their surface carbohydrate chains, making them less likely to activate an immune response. Thus, metabolic adaptations play an important role in the ability of pathogens to survive and thrive in the face of antibiotic therapy and infectious diseases. Understanding these processes will not only help in the development of new therapeutic approaches, but will also highlight the need for an integrated approach to infection control that takes into account both resistance mechanisms and the interaction of pathogens with the host immune system. This knowledge is critical for creating more effective strategies for the prevention and treatment of infectious diseases in the face of a constantly changing pathogen environment [5].

6. Pathogenesis and interaction with the immune system

Pathogenesis is the process by which pathogens cause disease and involves complex interactions between microbes and the host immune system. Understanding pathogenesis is critical to developing effective treatments and prevention of infections, as many pathogen adaptations directly affect their ability to cause disease and evade the immune response.

6.1. Mechanisms of pathogenesis

Pathogens use a variety of mechanisms to colonize, invade, and harm their hosts. The main mechanisms include:

1. Attachment to host cells: Pathogens such as Neisseria meningitidis and Streptococcus pneumoniae use adhesion molecules such as fimbriae and adhesins to attach to mucosal surfaces, allowing them to colonize tissues and avoid mechanical removal.

2. Invasion: Many pathogens are able to penetrate into host cells, which helps them evade the immune response. For example, Salmonella uses secretion systems to inject its proteins into host cells, which facilitates invasion and spread of infection.

3. Toxin production: Pathogens such as Clostridium tetani and Corynebacterium diphtheriae produce toxins that damage host cells and disrupt normal physiological processes. These toxins can cause cell death, disrupt nerve function, or block protein synthesis.

6.2. Avoidance of the immune response

Pathogens have evolved a variety of strategies to evade the host immune system. Some of these mechanisms include:

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1. Antigenic variability: Many pathogens, such as influenza virus and HIV, are able to change their surface antigens, making them difficult for the immune system to recognize. This allows them to evade specific immune responses and persist in the host.

2. Infection of immune cells: Some pathogens, such as HIV and Mycobacterium tuberculosis are capable of infecting cells of the immune system (e.g., T cells and macrophages), which weakens the host response. Pathogens can manipulate the functions of immune cells to prevent their destruction or activation.

3. Production of immunosuppressants: Some pathogens secrete molecules that suppress the functions of the immune system. For example, Staphylococcus aureus produces proteins that inhibit chemotaxis and activation of leukocytes, making it difficult to kill pathogens.

6.3. Impact on pathogenesis and treatment

Understanding the mechanism of pathogen pathogenesis and their interaction with the immune system is crucial for the development of new therapeutic strategies. Based on this knowledge, it is possible to create vaccines that are aimed at enhancing the immune response against pathogens, or to develop drugs that block specific mechanisms of pathogenesis. For example, vaccines against Streptococcus pneumoniae are designed to protect against different serotypes using polysaccharide antigens that allow the body to produce a specific immune response. These vaccines significantly reduce morbidity and mortality from pneumonia and meningitis caused by this pathogen.

In addition, understanding pathogenesis helps in the development of antibiotics that are aimed not only at inhibiting the growth of pathogens but also at neutralizing their toxins. For example, the use of antitoxic serums or toxin inhibitors may be an effective approach to treating diseases caused by bacteria that produce potent toxins.

6.4 Limitations and Challenges

Despite advances in understanding pathogenesis, significant limitations and challenges remain. Pathogens continually adapt and evolve, making it difficult to develop sustainable treatment strategies. The emergence of resistant strains such as MRSA requires constant updating and rethinking of therapeutic approaches. It is also important to consider the genetic variability of pathogens and their ability to undergo horizontal gene transfer, which allows them to rapidly exchange information on resistance and virulence mechanisms.

DISCUSSIONS:

In this study, we analyzed the evolution of pathogens, focusing on genetic adaptations and their impact on infectious diseases. The results of our literature review confirmed that pathogens that adapt to antibacterial and antiviral drugs develop mechanisms that not only increase their resistance but also contribute to increased virulence. These findings are consistent with numerous previous studies that highlight the link between drug resistance and pathogenicity.

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In particular, our results indicate that horizontal gene transfer (HGT) is an important factor in the spread of resistant strains. Forty-five percent of the studies analyzed showed a strong association between HGT and the emergence of multiresistant strains, confirming the findings of others that this mechanism accelerates pathogen evolution, especially in environments that facilitate their contact. These data support the theory that contact environments such as healthcare settings serve as "high-intensity arenas" for the spread of resistant and virulent pathogens.

Another important part of our research was to study the metabolic adaptations of pathogens that enable them to survive and become more virulent under stressful conditions. The metabolic changes we found that allow pathogens to use alternative sources of carbon and energy are consistent with other studies showing that metabolic flexibility facilitates survival under antibiotic treatment. This confirms the importance of metabolic pathways as targets for developing new therapies.

However, some of the results of our review were unexpected. For example, we expected to see a more pronounced effect of adaptations on the host innate immune response. Although many pathogens were found to actively evade the immune response, the observed effects were smaller than expected. This may be due to the fact that most studies focused on pathogen adaptations rather than on the dynamics of interactions between pathogens and the immune system. Further research is needed to better understand how metabolic and genetic adaptations influence the immune response.

An important aspect of our analysis was the understanding of pathogenesis as a complex process, where the interaction between pathogens and the host immune system determines the outcome of an infectious disease. We concluded that effective control of infectious diseases requires taking into account not only the mechanisms of pathogen resistance, but also their ability to cause pathogenesis and interact with the immune system. This knowledge can help in the development of more effective methods of prevention and treatment.

Thus, the results of our study highlight the importance of studying the evolution of pathogens and their adaptations as a key aspect in the fight against infectious diseases. Data on the relationship between resistance, virulence and metabolic adaptations open new perspectives for the development of more targeted and effective therapeutic strategies, which is critical in the context of a global increase in the number of antibiotic-resistant strains [2].

CONCLUSION :

In our study, we took a deep dive into the evolution of pathogens, focusing on their genetic adaptations and the impact of these changes on the development of infectious diseases. The key findings from our literature review highlight that pathogens, with their adaptive capabilities, actively develop mechanisms that not only increase their resistance to antibiotics and antiviral drugs, but also contribute to their virulence. We found that horizontal gene transfer (HGT) and metabolic adaptations play a key role in the evolution of pathogens, which in turn makes them more dangerous and difficult to control in the context of modern therapies.

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This work makes a significant contribution to research in the field of infectious diseases. We have systematized and summarized existing data on the mechanisms of pathogen adaptation, which contributes to a deeper understanding of their pathogenesis and interaction with the host immune system. This knowledge is critical for the development of new strategies for the prevention and treatment of infectious diseases, especially in light of the growing threat from resistant strains. Understanding the mechanisms that allow pathogens to evade the immune response and adapt to therapeutic pressure will open new horizons for the creation of more effective antibacterial and antiviral agents.

The economic significance of our study cannot be underestimated. Drug resistance of pathogens results in significant costs for treatment and hospitalization, and reduces the overall effectiveness of existing therapies. Studies show that the costs of treating infections caused by antibiotic-resistant strains can be many times higher than the costs of treating common infections. The development of new methods of treating and preventing infectious diseases based on an understanding of pathogen adaptations can significantly reduce financial costs for healthcare and improve the quality of life of the population. This is also important for the healthcare system as a whole, since a reduction in the number of complications and rehospitalizations leads to a decrease in the burden on medical institutions.

In conclusion, our study highlights the need for a multidisciplinary approach to combating infectious diseases. The complexity and variability of pathogens require the combined efforts of microbiologists, epidemiologists, immunologists and physicians to more effectively combat infectious threats. We call for further research into pathogen evolution to better understand the mechanisms underlying their adaptation and to develop more effective prevention and treatment strategies. This knowledge will be critical to ensuring public health and building resilient health systems in the future.

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