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CLINICAL GROUP

ARCHIVES OF Pulmonology and Respiratory Care OPEN ACCESS JOURNAL

ISSN: 2581-3684 DOI: https://dx.doi.org/10.17352/aprc

Review Article

What Successes can be Expected in the Fight Against Infection in Acute Pneumonia in the Context of the Side Effects of Antimicrobial Therapy?

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Received: 30 October, 2025
Accepted: 06 November, 2025
Published: 07 November, 2025

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Keywords: Acute pneumonia; Antimicrobial resistance; Antibiotic therapy; Pathogenesis; Evolution of the etiology of AP; Review analysis, Didactic influence of antibiotics

Abstract

Acute pneumonia (AP) is still one of the most unpredictable and dangerous inflammatory diseases, even with significant progress in antimicrobial treatment. This review looks at how medical understanding and treatment of AP have changed over time. It focuses on the side effects and limitations of long-term antibiotic use. The analysis uses both historical and modern literature to show how the germ theory of disease and the reliance on antibiotics have created current misunderstandings about the causes and treatment of AP. The paper discusses the rise of antibiotic resistance, the changing nature of pneumonia germs, and the increasing occurrence of viral pneumonia in the post-antibiotic era. It also argues that the focus on antimicrobials has caused us to overlook the mechanisms related to the host's response. By bringing together evidence from a century of microbiological and clinical studies, the author calls for a shift in thinking. We need to look beyond antibiotics as the only treatment and adopt a broader view of the mechanisms behind AP. The review ends by stating that if we do not reevaluate the role of microbial factors and the limits of antibiotics, progress in treating pneumonia will remain limited.

Introduction

Acute pneumonia (AP) has always been considered a severe and unpredictably dangerous disease and remains so despite impressive medical advances in many fields. For many centuries, the causes and characteristics of this disease remained unexplored, but the ancient postulate that pneumonia is not something a person catches, but something they become ill with, remains relevant to this day. A lack of necessary knowledge did not prevent ancient physicians from empirically finding approaches to treating such patients. In such cases, pathogenetic interventions played a key role, but their use was not clearly justified or regulated. Nevertheless, emergency treatment methods such as bloodletting, cupping therapy, or short-term cooling of the patient's body have a centuries-old history of use, demonstrating their relevance.

Advances and discoveries in microbiology in the late 19th century drew attention to a new understanding of the nature of AP and gradually shaped a narrow concept of the disease that defined approaches to solving this problem for almost a century. During this period, the fundamental principle of this concept—the primacy of the microbial factor—was not yet firmly established in professional thought, but it served as a stimulus for the search for and development of etiotropic agents. Thus, the task of developing antimicrobial drugs became a natural consequence for pharmacists with the advent of microbiological diagnostics of AP.

Since the first etiologic studies, it has been established that this disease can be caused by more than one microbe [1], and these inflammatory factors can include representatives of the body's commensal microflora [2]. The lack of an etiologic monopoly in this disease led to its designation as a nonspecific inflammatory process. Streptococcus pneumoniae, although one of the first pathogens discovered and consistently accounting for 95% or more of pathogens in the pre-antibiotic era [3], and the disease itself could rightfully be called

Citation: Klepikov I. What Successes can be Expected in the Fight Against Infection in Acute Pneumonia in the Context of the Side Effects of Antimicrobial Therapy?. Arch Pulmonol Respir Care. 2025;11(1):017-021. Available from: https://dx.doi.org/10.17352/aprc.000093

"pneumococcal pneumonia," was not a factor influencing morbidity. The inconsistency of its presence in the etiology of AP began to manifest itself after the advent of antibiotics, when its undisputed leadership began to rapidly decline and has since been irrevocably lost [4]. On the other hand, its non-aggressive presence in the commensal microflora of healthy individuals has been established.

The most successful etiotropic treatment of patients with AP was achieved shortly before the advent of antibiotics, thanks to the use of sulfonamides and antipneumococcal antiserum [3]. The use of these drugs improved treatment outcomes and demonstrated the therapeutic potential of their targeted antimicrobial action, raising hopes for further improvements in this therapy. In the absence of scientific explanations for the nature of AP at the time, the triumphant start of antibiotic therapy arose against a psychologically favorable backdrop and contributed to the emergence of the so-called germ theory of disease [3]. The essence of this doctrine was the recognition of the microbial factor as the primary cause of disease development, and, accordingly, antimicrobials were presented as the primary therapeutic resource. From this point on, suppression of the causative agent of AP became the primary goal in the treatment of this group of patients.

Although the rapid development of antibiotic resistance in microflora exposed to them was known even in the preclinical stage of research [5,6], and Alexander Fleming, one of the pioneers of this therapy, warned of the dangers of the uncontrolled use of these drugs [7], in clinical practice, everything was subordinated to the desire to achieve the desired result at any cost. Moreover, already in the early stages of this therapy, antibiotics began to be used not only for treatment but also for disease prevention, prescribing them to apparently healthy individuals, significantly exceeding the reasonable limits of their use [8-11]. However, even the founders of this therapeutic innovation could not foresee the scale and severity of side effects that could be expected from prolonged exposure of bacteria to "related" antagonist components.

Discussion

The development of bacterial resistance as a side effect of antibiotics became evident soon after the widespread use of this therapy, and the official countdown since the emergence of MRSA has already exceeded six decades [12,13]. However, real concern about this phenomenon has arisen only in the last couple of decades. A detailed and critical analysis of data from the entire history of this therapy shows that this side effect is not as dramatic as it currently appears, if the nature of AP is considered from the perspective of fundamental biomedical science. Bacterial resistance can be considered a serious problem only if the erroneous idea persists that antimicrobial therapy is the primary and only method of treating AP. However, even with this approach to treatment, resistant microorganisms are statistically rare causative agents of AP, accounting for no more than 1% - 2% of the etiology of the disease [14-16]. At the same time, some categories of healthy individuals are latent carriers of such flora, with a frequency of up to 6% - 10% [17-19]. The latter situations remain a subject of discussion in the context of sanitizing antimicrobial therapy [20]. It should be noted that currently, the proportion of some resistant microorganisms in the general population of the planet's inhabitants is 2% - 3% [21,22], but if the current trend continues, this figure may increase.

By focusing on the single side effect of antibiotic therapy—the development of microbial resistance—medicine has overlooked the phenomenon of periodic renewal of active pneumonia pathogens. This side effect of antibiotics began to be observed soon after their practical use, when pneumococcus began to lose its traditional role as the dominant pathogen [4]. In the early days of antibiotic therapy, the focus was on the dynamic replacement of bacterial microflora, but over the past three to four decades, a steady increase in viral pneumonias has been observed [23,24], which appears to be a natural response of the microbiome to prolonged antimicrobial activity.

Attempts to early identify AP pathogens have been underway for a long time, but their lack of success is reflected in two approaches to finding alternative solutions. On the one hand, leading expert forums do not consider the results of bacteriological diagnostics of AP to be important, recommending empirical antibacterial therapy [25,26]. On the other hand, microbiological diagnostic methods continue to be refined in the hope of achieving progress in the application of early, targeted etiotropic therapy [27,28]. However, as is evident from the nature of these efforts, no significant innovations or changes have occurred. Etiotropic treatment, represented by antibiotics, continues to play a leading role, and the significant increase in the viral etiology of AP has not led to significant changes in treatment.

The gradual transformation of the etiologic spectrum of AP has led to a significant reduction in the justified use of antibiotics. As a result, a situation has arisen in which antimicrobial drugs, by virtue of their action, have initiated a process of self-elimination from the therapeutic arsenal for AP. Statistics on the identification of AP pathogens in recent years show that, despite improvements in microbiological diagnostic methods, up to half or more of the tests are negative [29]. Among the remaining positive tests, viral forms of inflammation are increasingly common, and only a minority confirm bacterial inflammation of the lung tissue [30-32]. These data allow us to estimate the proportion of patients with AP for whom antibacterial therapy is justified.

It is no coincidence that, on the one hand, the observed transformation of the etiology of AP and the decline in the clinical efficacy of antibiotics, and on the other, attempts to reduce the impact of these drugs on the further development of resistant strains, have led to some successful attempts to treat such patients without antimicrobials [33]. The results of such observations should not be surprising, given the fact that a similar number of patients with COVID-19 pneumonia during the SARS-CoV-2 pandemic were cured without traditional etiotropic therapy due to its unavailability. At the same time, the number of patients with severe AP remains a challenge for modern medicine, and the usual reliance on antimicrobials in such cases does not produce the expected results and requires additional support and measures.

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The more severe the disease, the more pronounced its manifestations, causing concern for the patient's condition and requiring urgent and intensive care. These principles have been used in recent decades to distinguish between patients with severe AP [34,35]. However, some experts point to the unpredictability of this process even in cases that initially raised no concerns [36,37]. In previous years, attempts were made to explain such variants of AP development by the virulence of the pathogens. However, differential diagnosis based on etiology proved ineffective not only for bacterial forms of inflammation but also in distinguishing between bacterial and viral processes [38-40]. Moreover, the main clinical manifestations of AP remained stable regardless of the pathogen.

This last circumstance indicates the dependence of clinical symptoms on the functional characteristics of the affected organ, the impairment of which occurs as a result of inflammatory damage to its structures. This obviousness is a manifestation of the classic fifth sign of inflammation (loss of function) and emphasizes the importance of understanding the pathogenesis of this disease. Thus, the mechanism of AP development follows a general pattern, but the dynamics of this process and, consequently, the severity of its manifestations are individual. This individual characteristic of the body's response has long been known, but only the experience of the SARS-CoV-2 pandemic, when the coronavirus demonstrated completely different reactions to its effects, sparked discussion in this area [41,42].

Discussion and research into the various mechanisms of the body's response to inflammation, which has increased due to the pandemic, are undoubtedly an important step in addressing the problem of AP. However, further progress in this area is only one aspect of a broader topic. The root cause of this problem, without which further progress in this field is impossible, lies in professional misconceptions associated with the existing concept of AP. This confusion arose with the emergence of the germ theory of AP as the first scientific interpretation of the nature of this disease. In the era of effective antibiotic use, the foundations of this concept have been significantly strengthened, and a system for training physicians to strictly adhere to these principles has emerged. Due to the declining effectiveness of antimicrobial therapy in recent decades, there has been no attempt to constructively analyze the accumulated counterarguments, while a reverent attitude toward antibiotics persists. Despite these changes, these drugs retain a reputation as the only cure for AP, perpetuating the false notion of the pathogen's leading role in the development of the disease and leading to inappropriate treatment choices.

Summarizing at least the above brief descriptions of the facts accumulated over the long period of antibiotic use, the leading role of the pathogen in the development of AP appears unproven. The same can be said about antibiotics as the primary treatment for such diseases. By using antimicrobials as the basis of treatment and continuing to rely on the success of such therapy, modern medicine today cannot accurately determine which infection it is combating in each specific case. Therefore, after the obvious failures of many years of accurate diagnosis of AP pathogens, the use of antibiotics has ultimately become logically considered empirical. However, the continued widespread use of these drugs against the backdrop of a significant increase in viral diseases may at first seem incomprehensible.

This latter circumstance is due, on the one hand, to inflated expectations regarding the therapeutic potential of antibiotics and a persistent belief in their therapeutic effect. On the other hand, the unjustified use of antibiotics is due to the ignoring of certain side effects of this therapy, which go unnoticed. At the same time, as noted above, excessive attention to resistant microflora as the sole consequence of antibiotics further emphasizes their important place in the treatment of AP, ascribed to them by modern medicine. However, such attention only complicates a comprehensive assessment of the problem and its solution. The constant change in active AP pathogens and the gradual displacement of antibiotics from the therapeutic arsenal for such inflammatory processes have an increasingly significant impact on the choice of etiotropic agents, but, unfortunately, this side effect of antibiotics remains unaccounted for and underestimated. An even more global and important consequence of the antibiotic era is its didactic mission and psychological impact on professional worldviews, which have not undergone an evolution commensurate with the observed changes of recent decades.

Conclusion

Thus, over the long period of antibiotic use, the initial conditions under which this therapy was initiated have changed significantly. Significant biological transformations were caused by the inevitable decline in the effectiveness of these drugs and the adaptation of the microflora to such prolonged aggression. Many bacterial strains, having acquired resistance to antimicrobials, have not become more aggressive and, simply by participating in the inflammatory process, can complicate the task of etiotropic treatment. At the same time, the long-term use of antimicrobials was accompanied by the suppression and weakening of pathogens causing acute inflammatory diseases, which forced nature to gradually update the list of pathogens and, ultimately, forced viruses to replace bacteria as a natural countermeasure to antibiotics. Currently, a significant increase in viral forms of inflammation raises important questions about the future of antibiotics and their use, further exacerbating the consequences of their action.

The new circumstances that have emerged in the antibiotic era and characterize the problem of AP being discussed today are a prerequisite for harmonizing professional views. This requires a balanced and critical reassessment of the persistent desire to restore the past by developing and using new generations of antibiotics. Without a comprehensive assessment of previous experience with this therapy and the absence of conclusions regarding the reasons for the significant decline in its effectiveness, the unpredictability and danger of such a move are completely obvious. Stubborn adherence to outdated conceptual understandings of the nature of AP is a kind of anchor that hinders progress in solving the problem as a whole and perpetuates the principles of inadequate treatment.

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Deeply held beliefs about the dominant role of microbial factors in the development of AP and long-held hopes for success solely through antibiotics are refuted by abundant evidence, yet they remain the guiding concept. Understanding the nature of the problem under discussion is the primary goal, and without a radical shift in our understanding, achieving this key goal will remain a pipe dream.

References

1. Gram C. On the isolated staining of schizomycetes in section and dry preparations. Fortschritte der Medizin. 1884;2(6):185–189.
2. Jaccoud. Scientific American. New York (NY): Munn & Company; 1887;196.
3. Podolsky SH. The changing fate of pneumonia as a public health concern in 20th-century America and beyond. American Journal of Public Health. 2005;95(12):2144-2154. Available from: https://doi.org/10.2105/ajph.2004.048397
4. Gadsby NJ, Musher DM. The microbial etiology of community-acquired pneumonia in adults: from classical bacteriology to host transcriptional signatures. Clinical Microbiology Reviews. 2022;35:e00015-22. Available from: https://doi.org/10.1128/cmr.00015-22
5. Abraham EP, Chain E. An enzyme from bacteria is able to destroy penicillin. Reviews of Infectious Diseases. 1988;10(4):677-678. (Originally published 1940). Available from: https://pubmed.ncbi.nlm.nih.gov/3055168/
6. Rammelkamp T. Resistance of Staphylococcus aureus to the action of penicillin. Experimental Biology and Medicine. 1942;51:386-389. Available from: https://doi.org/10.3181/00379727-51-13986
7. Fleming A. The Nobel Prize in Physiology or Medicine 1945 – Penicillin: Nobel lecture. NobelPrize.org; 1945. Retrieved 2020 Oct 17. Available from: https://www.nobelprize.org/prizes/medicine/1945/fleming/lecture/
8. Greene HJ, Alture-Werber E. Penicillin as a prophylactic in abdominal surgery. Proceedings of the Society for Experimental Biology and Medicine. 1945;58(3):211-212.
9. Tupper WRC, et al. The prophylactic use of penicillin in obstetrics. American Journal of Obstetrics and Gynecology. 1949;57(3):569-574.
10. Cope S, Sanderson G, Hill S, Chamberlain EN. Prophylactic use of oral penicillin in rheumatic fever, chorea, and carditis. British Medical Journal. 1960;1:913. Available from: https://www.bmj.com/content/1/5177/913
11. Rammelkamp CH Jr. Prophylaxis of bacterial disease with antimicrobial drugs. American Journal of Medicine. 1965;39(5):804-811. Available from: https://doi.org/10.1016/0002-9343(65)90099-9
12. Jevons MP. "Celbenin"-resistant staphylococci. British Medical Journal. 1961;1:124-125. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC1952888/
13. University of Chicago Medical Center. MRSA history timeline: the first half-century, 1959–2009. Chicago (IL): University of Chicago Medical Center; 2010. Archived 2020 Feb 18. Retrieved 2012 Apr 24.
14. Sakamoto Y, Yamauchi Y, Jo T, Michihata N, Hasegawa W, Takeshima H, et al. In-hospital mortality associated with community-acquired pneumonia due to methicillin-resistant Staphylococcus aureus: a matched-pair cohort study. BMC Pulmonary Medicine. 2021;21:345. Available from: https://bmcpulmmed.biomedcentral.com/articles/10.1186/s12890-021-01713-1
15. Ding H, Mang NS, Loomis J, Ortwine JK, Wei W, O'Connell EJ, et al. Incidence of drug-resistant pathogens in community-acquired pneumonia at a safety net hospital. Microbiology Spectrum. 2024;12:e00792-24. Available from: https://journals.asm.org/doi/10.1128/spectrum.00792-24
16. Gohil SK, Septimus E, Kleinman K, Avery TR, Varma N, Sands KE, et al. Initial antibiotic selection strategy and subsequent antibiotic use—insights from the INSPIRE trials. JAMA. 2025;334(12):1107-1109. Available from: https://doi.org/10.1001/jama.2025.11256
17. Aubry-Damon H, Grenet K, Ndiaye-Sall P, Che D, Corderio E, Bougnoux M, et al. Antimicrobial resistance in commensal flora of pig farmers. Emerging Infectious Diseases. 2004;10:873-879. Available from: https://doi.org/10.3201/eid1005.030735
18. Albrich WC, Harbarth S. Health-care workers: source, vector, or victim of MRSA? Lancet Infectious Diseases. 2008;8:289-301. Available from: https://doi.org/10.1016/s1473-3099(08)70097-5
19. Graveland H, Wagenaar JA, Heesterbeek H, Mevius D, van Duijkeren E, Heederik D. Methicillin-resistant Staphylococcus aureus ST398 in veal calf farming: human MRSA carriage related with animal antimicrobial usage and farm hygiene. PLoS One. 2010;5:e10990. Available from: https://doi.org/10.1371/journal.pone.0010990
20. Liu C, Holubar M. Should an MRSA nasal swab guide empiric antibiotic treatment? NEJM Evidence. 2022;1(12). Available from: https://doi.org/10.1056/evidccon2200124
21. Centers for Disease Control and Prevention (CDC). Clinical overview of methicillin-resistant Staphylococcus aureus (MRSA) in healthcare settings. Health Care Providers. 2025. Available from: https://www.cdc.gov/mrsa/hcp/clinical-overview/index.html
22. Centers for Disease Control and Prevention (CDC). Antibiotic-resistant Streptococcus pneumoniae. Public Health. 2024. Available from: https://www.cdc.gov/pneumococcal/php/drug-resistance/index.html
23. World Health Organization. Revised global burden of disease 2002 estimates. Geneva: WHO; 2004. Available from: http://www.who.int/healthinfo/global_burden_disease/estimates_regional_2002_revised/en/
24. Ruuskanen O, Lahti E, Jennings LC, Murdoch DR. Viral pneumonia. Lancet. 2011;377(9773):1264-1275. Available from: https://doi.org/10.1016/s0140-6736(10)61459-6
25. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, et al. Diagnosis and treatment of adults with community-acquired pneumonia: an official clinical practice guideline of the American Thoracic Society and Infectious Diseases Society of America. Am J Respir Crit Care Med. 2019;200(7):e45-e67. Available from: https://doi.org/10.1164/rccm.201908-1581st
26. Martin-Loeches I, Torres A, Nagavci B, Aliberti S, Antonelli M, Bassetti M, et al. ERS/ESICM/ESCMID/ALAT guidelines for the management of severe community-acquired pneumonia. Intensive Care Med. 2023;49:615-632. Available from: https://link.springer.com/article/10.1007/s00134-023-07033-8
27. Ling L, Lai CKC, Rhee C. Bacterial multiplex polymerase chain reaction tests for the diagnosis and management of pneumonia: ready for prime time? Thorax. 2025;80:862-872. Available from: https://doi.org/10.1136/thorax-2024-222297
28. Zhang JH, Chou SF, Wang PH, Yang CJ, Lai YH, Chang MY, et al. Optimizing patient outcomes in severe pneumonia: the role of multiplex PCR in the treatment of critically ill patients. Front Med (Lausanne). 2024;11:1391641. Available from: https://doi.org/10.3389/fmed.2024.1391641
29. Shoar S, Musher DM. Etiology of community-acquired pneumonia in adults: a systematic review. Pneumonia (Nathan). 2020;12:11. Available from: https://pneumonia.biomedcentral.com/articles/10.1186/s41479-020-00074-3
30. Jain S, Self WH, Wunderink RG, Fakhran S, Balk R, Bramley AM, et al. CDC EPIC Study Team. Community-acquired pneumonia requiring hospitalization among U.S. adults. N Engl J Med. 2015;373:415-427. Available from: https://doi.org/10.1056/nejmoa1500245

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31. Shorr AF, Zilberberg MD, Micek ST, Kollef MH. Viruses are prevalent in non-ventilated hospital-acquired pneumonia. Respir Med. 2017;122:76-80. Available from: https://doi.org/10.1016/j.rmed.2016.11.023

32. Palomeque A, Cilloniz C, Soler-Comas A, Canseco-Ribas J, Rovira-Ribalta N, Motos A, et al. A review of the value of point-of-care testing for community-acquired pneumonia. Expert Rev Mol Diagn. 2024;1–14. Available from: https://doi.org/10.1080/14737159.2024.2391027

33. Walker PJ, Wilkes C, Duke T, Graham HR; ARI Review group. Can child pneumonia in low-resource settings be treated without antibiotics? A systematic review & meta-analysis. J Glob Health. 2022;12:10007. Available from: https://doi.org/10.7189/jogh.12.10007

34. Niederman MS, Bass JB Jr, Campbell GD, Fein AM, Grossman RF, Mandell LA, et al. Guidelines for the initial management of adults with community-acquired pneumonia: diagnosis, assessment of severity, and initial antimicrobial therapy. Am Rev Respir Dis. 1993;148(5):1418-1426. Available from: https://doi.org/10.1164/ajrccm/148.5.1418

35. ERS Task Force. Guidelines for management of adult community-acquired lower respiratory tract infections. Eur Respir J. 1998;11(4):986-991. Available from: https://doi.org/10.1183/09031936.98.11040986

36. Jiang J, Yang J, Jin Y, Cao J, Lu Y. Role of qSOFA in predicting mortality of pneumonia: a systematic review and meta-analysis. Medicine (Baltimore). 2018;97:e12634. Available from: https://doi.org/10.1097/md.0000000000012634

37. Peyrani P, Arnold FW, Bordon J, Furmanek S, Luna CM, Cavallazzi R, et al. Incidence and mortality of adults hospitalized with community-acquired pneumonia according to clinical course. Chest. 2020;157:34-41. Available from: https://doi.org/10.1016/j.chest.2019.09.022

38. Heneghan C, Plueddemann A, Mahtani KR. Differentiating viral from bacterial pneumonia. Oxford: Centre for Evidence-Based Medicine; 2020. Available from: https://www.cebm.net/covid-19/differentiating-viral-from-bacterial-pneumonia/

39. Kamat IS, Ramachandran V, Eswaran H, Guffey D, Master DM. Procalcitonin to distinguish viral from bacterial pneumonia: a systematic review and meta-analysis. Clin Infect Dis. 2020;70(3):538-542. Available from: https://doi.org/10.1093/cid/ciz545

40. Lhommet C, Garot D, Grammatico-Guillon L, Jourdannaud C, Asfar P, Faisy C, et al. Predicting the microbial cause of community-acquired pneumonia: can physicians or a data-driven method differentiate viral from bacterial pneumonia at patient presentation? BMC Pulm Med. 2020;20:62. Available from: https://bmcpulmmed.biomedcentral.com/articles/10.1186/s12890-020-1089-y

41. Meyer N. Dysregulated host immune response is the driver of disease progression and severe patient outcomes. Respir AMJ. 2023;1(1):26-35. Available from: https://doi.org/10.33590/respiramj/10304417

42. Sinha P. Severe viral lower respiratory tract infections pose a significant burden on patients and healthcare systems. Respir AMJ. 2023;1(1):26-35. Available from: https://doi.org/10.33590/respiramj/10304417

Citation: Klepikov I. What Successes can be Expected in the Fight Against Infection in Acute Pneumonia in the Context of the Side Effects of Antimicrobial Therapy?. Arch Pulmonol Respir Care. 2025;11(1):017-021. Available from: https://dx.doi.org/10.17352/aprc.000093