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INFECTIOUS LUNG DISEASES IN ADULTS: GENETIC DETERMINANTS, MOLECULAR BIOMARKERS, AND PERSONALIZED DIAGNOSTIC AND THERAPEUTIC STRATEGIES

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ABSTRACT

Introduction. Infectious lung diseases in adults remain clinically significant. The variability of their course is determined not only by the pathogen and comorbid conditions, but also by host genetic susceptibility, immune-response patterns, molecular biomarkers of inflammation, epithelial injury and post-infectious remodeling. Aim. To summarize current evidence on genetic determinants and molecular biomarkers of infectious lung diseases in adults and to assess their value for personalized diagnosis, prognosis and therapeutic decision-making. Materials and methods. An analytical literature review was performed. Scientific publications, clinical guidelines and systematic reviews published mainly in 2019-2026 were analyzed. The search terms included infectious lung diseases, pneumonia, genetic susceptibility, biomarkers, procalcitonin, KL-6, respiratory microbiome, metagenomic sequencing, post-infectious fibrosis and personalized medicine. Results. Promising areas include the assessment of genes involved in innate immunity and interferon signaling, the use of inflammatory and epithelial-vascular biomarkers for risk stratification, and the integration of microbiome, metagenomic and digital data into personalized models. The greatest practical value is expected from validated marker panels rather than from isolated laboratory tests. Conclusion. Infectious lung diseases in adults should be considered through a multilayer model that integrates genetic, immunological, microbiome and clinical-functional indicators. Such an approach can improve risk prediction, antimicrobial stewardship, follow-up planning and the prevention of post-infectious pulmonary complications.

Keywords: infectious lung diseases; pneumonia; genetic determinants; biomarkers; procalcitonin; KL-6; respiratory microbiome; metagenomic sequencing; personalized medicine; post-infectious fibrosis.

INTRODUCTION

Lower respiratory tract infections remain one of the leading causes of hospitalization, temporary disability and mortality in adults. Community-acquired pneumonia, viral pneumonitis and infectious exacerbations of chronic respiratory diseases continue to create a high clinical and socioeconomic burden, especially among patients with comorbidities and older adults.

The traditional model of patient management in respiratory infection is based on clinical presentation, imaging and etiological verification of the pathogen. However, this approach does not always explain why patients with comparable microbial burden and similar treatment have substantially different outcomes, ranging from rapid recovery to respiratory failure and post-infectious fibrotic remodeling.

For journals focused on genetics and biomarkers, the emphasis should be shifted from a general description of pulmonary diseases to molecular mechanisms and measurable indicators suitable for risk stratification. These indicators include variants of genes involved in innate immunity and interferon signaling, inflammatory proteins, markers of epithelial and endothelial injury, parameters of the respiratory microbiome and molecular methods for pathogen detection.

The aim of this article is to summarize current concepts of genetic determinants and biomarkers in adult infectious lung diseases and to demonstrate how these data may be used for personalized diagnosis, prognosis, selection of therapy and long-term follow-up after infection.

MATERIALS AND METHODS

This article was prepared as an analytical review of the literature. Sources were searched in PubMed/MEDLINE, Scopus, Web of Science, eLIBRARY and on the websites of professional societies. Russian and English search terms were used to reflect the genetic, molecular and clinical aspects of adult respiratory infections.

The review included clinical guidelines, systematic reviews, large cohort studies, genome-wide association studies, meta-analyses and publications addressing the clinical interpretation of biomarkers. Priority was given to publications from recent years, while classical studies were included when they were necessary to describe established genetic or molecular mechanisms.

Genetic determinants of susceptibility and severity in respiratory infections

Genetic predisposition to severe respiratory infection is mediated through several interconnected mechanisms: pathogen recognition, activation of the interferon response, regulation of inflammation, maintenance of the epithelial barrier, mucociliary clearance and tissue repair. These mechanisms are especially important in viral pneumonias, severe bacterial pneumonia and mixed infections.

The interferon pathway is of particular importance. Rare pathogenic variants in genes involved in type I interferon production and signaling, as well as autoantibodies against interferons, have been associated with life-threatening viral infection in some patients. Such findings show that severe respiratory infection may result not only from the virulence of the pathogen, but also from a specific defect in host antiviral defense.

For bacterial pneumonia and mixed infections, genetic associations have been studied less consistently. Potentially relevant variants include polymorphisms of innate immune receptors, complement components, mannose-binding lectin and cytokine genes. However, most of these markers are not yet ready for routine clinical use because their effect sizes are modest and depend on population, comorbidity and environmental factors.

Another important area is the genetics of post-infectious outcomes. The rs35705950 variant in the promoter region of MUC5B is one of the best studied genetic risk factors for interstitial lung abnormalities and pulmonary fibrosis [12, 13]. In the context of infection, such markers may help identify patients at increased risk of persistent radiological changes and fibrotic remodeling.

Genetic data are also relevant in chronic airway diseases, which increase the risk of complicated infection. Large studies have demonstrated the polygenic nature of reduced lung function and chronic obstructive pulmonary disease, as well as partly distinct genetic architectures of childhood-onset and adult-onset asthma. These findings are important for interpreting infectious exacerbations and selecting long-term anti-inflammatory strategies.

Table 1. Genetic and molecular areas relevant to infectious lung diseases

Marker group	Biological significance	Clinical interpretation
Interferon-response genes: IFNAR2, OAS1/OAS2/OAS3, TYK2	Participate in early antiviral defense, activation of innate immunity and regulation of inflammatory signaling.	Promising for assessing the risk of severe viral infections; require validation in multicenter cohorts.
Rare immune defects and autoantibodies against type I interferons	May lead to insufficient antiviral response despite otherwise preserved immune function.	Relevant in patients with unexpectedly severe viral infection, especially in the absence of clear clinical risk factors.
MUC5B rs35705950 and epithelial-repair genes	Associated with mucociliary clearance, epithelial remodeling and the risk of fibrotic changes.	May be used in research models of post-infectious fibrosis together with CT findings and functional testing.
Polygenic profiles of lung function and COPD	Reflect the inherited contribution to reduced FEV1/FVC, airway remodeling and emphysematous phenotype.	Useful for scientific stratification, but in routine practice remain secondary to spirometry, smoking history and assessment of

		exacerbations.
Genetics of asthma and T2 inflammation	Associated with epithelial cytokines, eosinophilic inflammation and response to biologic therapy.	Important for interpreting infectious exacerbations of asthma and choosing a long-term anti-inflammatory strategy.

Molecular biomarkers of diagnosis and prognosis

Biomarkers in infectious lung diseases serve several purposes: they support the probability assessment of bacterial or viral disease, reflect the intensity of systemic inflammation, help estimate the risk of respiratory failure and adverse outcome, and can guide follow-up after recovery. At the same time, biomarkers should not replace clinical judgment; they should be interpreted together with symptoms, imaging, microbiological data and functional status.

C-reactive protein and procalcitonin remain the most accessible laboratory indicators of the inflammatory response. Procalcitonin may help reduce unnecessary antibiotic use in acute respiratory infections when applied within validated clinical algorithms. However, low procalcitonin values should not be used as the only reason to withhold antibiotics in severe pneumonia or septic states.

Cytokine and immunological markers, including interleukin-6, ferritin, the neutrophil-to-lymphocyte ratio and markers of endothelial activation, reflect the intensity of inflammation and may have prognostic value. Their clinical utility is highest when serial measurements are interpreted in the context of disease severity, oxygen requirement and organ dysfunction.

Markers of alveolar epithelial injury and fibrotic risk include KL-6, surfactant protein D, soluble receptor for advanced glycation end products, matrix metalloproteinases and profibrotic mediators. KL-6 is considered a marker of type II alveolar epithelial injury and interstitial lung disease activity, and it is increasingly discussed in relation to post-infectious lung injury.

Modern etiological diagnosis increasingly uses molecular methods: multiplex PCR, targeted sequencing and metagenomic next-generation sequencing. Metagenomic sequencing can detect a broad spectrum of bacterial, viral, fungal and atypical pathogens, as well as potential resistance determinants. Its main limitation is the need to distinguish true infection from colonization and contamination.

Table 2. Main biomarkers and molecular methods in adult infectious lung diseases

Biomarker / method	**What it reflects**	**Potential clinical application**
C-reactive protein	Systemic inflammation and acute-phase activity.	Assessment of inflammatory dynamics; adjunct to clinical presentation and imaging.
Procalcitonin	Probability of bacterial inflammation and systemic response to infection.	Rationalization of antibiotic therapy and control of treatment duration when validated algorithms are used.
IL-6, ferritin, neutrophil-to-lymphocyte ratio	Hyperinflammatory response and immune dysregulation.	Prediction of severity and decision-making regarding enhanced monitoring.
D-dimer and markers of endothelial activation	Coagulopathy, vascular injury and risk of thrombotic complications.	Risk stratification in severe viral pneumonia and respiratory failure.
KL-6, SP-D, sRAGE	Alveolar epithelial injury, interstitial inflammation and risk of fibrosis.	Selection of patients for long-term follow-up, CT monitoring and lung-function assessment after infection.
Multiplex PCR	Presence of genetic material from specific respiratory pathogens.	Rapid etiological verification, especially in viral and atypical infections.
Metagenomic sequencing	Broad spectrum of microbial	Diagnosis of severe, mixed, rare

	genomes and potential resistance genes.	and culture-negative infections; requires strict clinical interpretation.

Respiratory microbiome as a biomarker and risk modifier

The respiratory microbiome is a dynamic ecosystem in which bacteria, viruses and fungi interact with the airway epithelium and the host immune system. Dysbiosis may amplify inflammation, impair colonization resistance and increase susceptibility to infection or exacerbation of chronic lung disease.

Unlike classical microbiological diagnostics, microbiome analysis evaluates not only the presence of a specific pathogen, but also the structure of the community. Reduced microbial diversity, dominance of potentially pathogenic taxa, changes in the viral or fungal components of the microbiome and shifts in the gut-lung axis may be considered potential biomarkers of risk.

Clinical use of microbiome data requires caution. Samples from the upper respiratory tract do not always reflect the lower airways, and detection of a microorganism does not prove infection. The most promising approach is the integration of microbiome data with clinical, radiological and inflammatory markers, rather than interpretation of sequencing results in isolation.

Post-infectious lung lesions and biomarkers of fibrotic remodeling

After respiratory infection, some patients continue to have dyspnea, reduced exercise tolerance, restrictive ventilatory defects, impaired diffusion capacity and abnormalities on computed tomography. Post-infectious changes may include organizing pneumonia, residual interstitial abnormalities, bronchiectasis, vascular injury and fibrotic remodeling.

From the perspective of genetics and biomarkers, the most important task is to identify signs that distinguish reversible inflammation from early fibrotic remodeling. Research models may combine genetic risk factors, including MUC5B, with biomarkers of epithelial injury such as KL-6 and SP-D, inflammatory markers, high-resolution CT patterns and functional parameters such as FVC and DLCO.

Dynamic follow-up is clinically important for high-risk patients: older adults, patients after severe pneumonia or acute respiratory distress syndrome, patients with COPD, interstitial lung diseases, marked residual CT changes and persistent reduction in diffusion capacity. Biomarker-guided follow-up could help determine the frequency of imaging, the need for pulmonary consultation and the indications for rehabilitation.

Biomarkers in personalized therapy

A personalized approach to infectious lung diseases does not mean abandoning standard clinical guidelines. Rather, it refines guideline-based decisions according to the individual risk profile. At the initial assessment stage, biomarkers may help determine the place of care, the intensity of monitoring and the probability of bacterial infection.

For antibacterial therapy, rational drug use is crucial. Procalcitonin and clinical dynamics may be used to shorten unnecessary antibiotic courses; however, the decision to start treatment in severe pneumonia should remain clinically driven. Biomarkers are most useful when they support de-escalation and treatment-duration decisions rather than when they are used as a single diagnostic criterion.

In chronic respiratory diseases, biomarkers help distinguish infectious exacerbation from worsening non-infectious inflammation. Blood eosinophils, the T2 inflammatory phenotype and clinical features of asthma or COPD may influence the choice of maintenance therapy and indirectly affect the frequency and severity of infectious exacerbations.

Table 3. Practical model for biomarker use in personalized patient management

Clinical stage	Recommended marker block	Decision that may be refined
Initial assessment	CRP, procalcitonin, oxygen saturation, complete blood count, severity scores, CT/X-ray findings.	Place of care, need for hospitalization, start of empirical therapy and intensity of monitoring.
Etiological diagnosis	PCR panels, culture methods, antigen tests and, when indicated, metagenomic sequencing.	Identification of the pathogen, de-escalation or change of antibacterial therapy, detection of mixed infections.

Assessment of hyperinflammation and organ risk	IL-6, ferritin, D-dimer, endothelial injury markers and blood gases.	Decision on intensive monitoring, thrombosis prevention and expanded laboratory follow-up.
Monitoring treatment response	Dynamics of temperature, symptoms, CRP/procalcitonin, oxygen requirement and microbiological results.	Shortening antibiotic duration, de-escalation of therapy and search for complications if response is absent.
Post-infectious follow-up	KL-6, SP-D and sRAGE when available; spirometry, DLCO and high-resolution CT.	Assessment of fibrotic risk, follow-up frequency, referral to rehabilitation and pulmonary consultation.

Integration of omics data and artificial intelligence

A promising direction is the integration of genomic, transcriptomic, proteomic, metabolomic, microbiome and clinical-radiological data into a unified risk model. This approach allows the transition from isolated marker assessment to a multidimensional patient profile, which can better reflect the heterogeneity of infectious lung diseases.

Artificial intelligence technologies can be used for CT image analysis, interpretation of pulmonary function tests, prediction of exacerbations and identification of hidden phenotypes of respiratory disease [21, 22]. However, clinical implementation requires external validation, transparent algorithms, assessment of bias and integration into real clinical workflows.

For scientific publications on genetics and biomarkers, it is important to emphasize not only the promise of digital solutions, but also their limitations: small samples, lack of unified thresholds, unequal access to laboratory tests, population specificity of genetic data and the need to prove that model-based decisions improve clinical outcomes.

LIMITATIONS AND UNRESOLVED ISSUES

Despite rapid growth in research, most genetic and molecular markers in infectious lung diseases do not yet have sufficient evidence to be used independently for clinical decision-making. Genetic variants usually have a probabilistic value and cannot be interpreted without clinical context. In addition, many studies focus on selected populations, which limits the generalizability of their results.

Biomarkers also face problems of standardization: measurement methods, thresholds, timing of sampling and outcome definitions differ across studies. Therefore, the most realistic direction is the development of validated panels in which genetic, inflammatory, microbiome and functional indicators complement each other.

CONCLUSION

Infectious lung diseases in adults should be considered as the result of interaction among the pathogen, genetically and immunologically determined host characteristics, the respiratory microbiome and environmental factors. For the field of genetics and biomarkers, the main scientific value lies in identifying measurable indicators that can improve risk stratification and personalize patient management. The most justified approach is not the use of a single universal test, but the formation of a multilayer model of personalized care. Such a model should include clinical assessment, molecular verification of the pathogen, dynamics of inflammatory biomarkers, indicators of epithelial and endothelial injury, functional tests and, in selected cases, genetic and microbiome data. This strategy may improve prognosis, rationalize antimicrobial therapy and reduce the burden of post-infectious pulmonary complications.

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CONTRIBUTION OF THE AUTHORS

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CONFLICT OF INTERESTS

The authors declare that there is no conflict of interest

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