

ИНФЕКЦИОННЫЕ БОЛЕЗНИ INFECTIOUS DISEASES

RISK PREDICTION OF POST-COVID CONDITIONS USING NLRP3 AND GSDMD MARKERS IN PATIENTS WITH BRONCHIAL ASTHMA AFTER COVID-19

**Lobova T.G.,
Vitkina T.I.**

Vladivostok Branch of the Far Eastern
Scientific Center of Physiology and
Pathology of Respiration – Institute
of Medical Climatology and Rehabilitative
Treatment (Russkaya str. 73g, Vladivostok
690105, Russian Federation)

Corresponding author:
Tatyana G. Lobova
e-mail: lobova.tg@dvfu.ru

ABSTRACT

Background. One of the key factors in triggering the hyperinflammation cascade in patients with bronchial asthma and COVID-19 is the activation of NLRP3 cytosolic complexes and the GSDMD proteins by SARS-CoV-2, which are effector molecules for the lytic and inflammatory forms of pyroptosis.

Aim. To determine prognostic criteria for worsening of mild bronchial asthma following an infectious disease caused by SARS-CoV-2.

Materials and Methods. In patients with mild, partially controlled bronchial asthma ($n = 86$), who were divided into 2 groups: asthma without COVID-19 ($n = 34$) and asthma following mild COVID-19 ($n = 52$), peripheral blood inflammatory markers were evaluated. The patients were examined immediately after recovery and confirmation of a negative SARS-CoV-2 test result, and six months post-infection. The control group included conditionally healthy volunteers ($n = 15$) with no history of COVID-19. Pulmonary function was monitored by measuring FVCL, OFV1, and OFV1/FVCL. NLRP3 and gasdermin D inflammasomes were measured by enzyme-linked immunosorbent assay.

Results. The study revealed that in a number of patients with mild asthma, who had post-COVID conditions 6 months after COVID 19, there were elevated levels of NLRP3 inflammasome and gasdermin D (GSDMD), as well as a decreased pulmonary function, indicating a hyperinflammatory process. A predictive model for worsening of mild asthma after COVID-19 was developed: concurrent elevation of NLRP3 > 0.04 ng/ml and GSDMD > 0.38 ng/ml levels during the convalescence period serves as a marker of a high risk of post-COVID complications.

Conclusion. The role of NLRP3 and GSDMD in exacerbating the course of asthma after COVID-19 has been established, and predictive threshold values of biomarkers in this patient population have been identified.

Keywords: bronchial asthma, post-COVID syndrome, inflammatory markers, NLRP3, gasdermin D

Received: 29.10.2025
Accepted: 10.07.2026
Published: 23.07.2026

For citation: Lobova T.G., Vitkina T.I. Risk prediction of post-COVID conditions using NLRP3 and GSDMD markers in patients with bronchial asthma after COVID-19. *Acta biomedica scientifica*. 2026; 11(3): 12-18. doi: 10.29413/ABS.2026-11.3.2

ПРОГНОЗ РИСКА РАЗВИТИЯ ПОСТКОВИДНЫХ НАРУШЕНИЙ С ИСПОЛЬЗОВАНИЕМ МАРКЕРОВ NLRP3 И GSDMD У ПАЦИЕНТОВ С БРОНХИАЛЬНОЙ АСТМОЙ, ПЕРЕНЕСШИХ COVID-19

**Лобова Т.Г.,
Виткина Т.И.**

Владивостокский филиал Федерального государственного бюджетного научного учреждения «Дальневосточный научный центр физиологии и патологии дыхания – Научно-исследовательский институт медицинской климатологии и восстановительного лечения» (690105, г. Владивосток, ул. Русская, 73 г, Россия)

Автор, ответственный за переписку:
Лобова Татьяна Геннадьевна
e-mail: lobova.tg@dvfu.ru

РЕЗЮМЕ

Обоснование. Одним из ключевых факторов запуска каскада гипервоспаления при сочетании бронхиальной астмы и COVID-19 предполагается активация вирусом SARS-CoV-2 цитозольных комплексов NLRP3 и белка GSDMD, являющихся эффекторными молекулами для литической и воспалительной формы пироптоза.

Цель. Определение прогностических критериев утяжеления течения легкой бронхиальной астмы (БА) на фоне перенесённого инфекционного заболевания, вызванного SARS-CoV-2.

Материалы и методы. У пациентов с легкой, частично контролируемой бронхиальной астмой ($n = 86$), которые были разделены на 2 группы: БА без COVID-19 ($n = 34$) и БА после легкой формы течения COVID-19 ($n = 52$) проведена оценка маркеров воспаления в периферической крови. Пациенты были обследованы сразу после выздоровления и подтверждения отрицательного результата теста на SARS-CoV-2, и через шесть месяцев после заражения. В контрольную группу вошли условно здоровые добровольцы ($n = 15$), которые не болели COVID-19. Проводился мониторинг функции внешнего дыхания ФЖЕЛ, ОФВ1, ОФВ1/ФЖЕЛ. Инфламмосомы NLRP3, газдермин D определяли методом иммуноферментного анализа.

Результаты. Исследование выявило, что у ряда пациентов с легкой астмой, у которых спустя 6 месяцев после COVID-19 сохраняются постковидные нарушения, отмечено повышение уровней инфламмосомы NLRP3 и газдермина D (GSDMD), а также снижение функции внешнего дыхания, что указывает на гипервоспалительный процесс. Разработан способ прогноза утяжеления лёгкой астмы после COVID-19: одновременное превышение уровней $NLRP3 > 0,04 \text{ ng/ml}$ и $GSDMD > 0,38 \text{ ng/ml}$ в периоде реконвалесценции служит маркером высокого риска постковидных осложнений.

Заключение. Установлена роль NLRP3, GSDMD в утяжелении течения БА после COVID-19, выделены прогностические пороговые значения биомаркеров у данного контингента пациентов.

Ключевые слова: бронхиальная астма, постковидные нарушения, маркеры воспаления, NLRP3, газдермин D

Статья поступила: 29.10.2025
Статья принята: 10.07.2026
Статья опубликована: 23.07.2026

Для цитирования: Лобова Т.Г., Виткина Т.И. Прогноз риска развития постковидных нарушений с использованием маркеров NLRP3 и GSDMD у пациентов с бронхиальной астмой, перенесших COVID-19. *Acta biomedica scientifica*. 2026; 11(3): 12-18. doi: 10.29413/ABS.2026-11.3.2

INTRODUCTION

Although COVID-19 is no longer classified as a pandemic, it remains a significant clinical problem. The consequences of the infection are particularly serious, especially in immunocompromised individuals, who include patients with asthma [1–3]. According to the WHO, CENTRAL, and Rub Med electronic databases, the overall prevalence of asthma among COVID-19 patients was 8% between December 1, 2019 and July 2021, [4]. In Russia, the proportion of asthma patients who have had COVID-19 reaches up to 15%, depending on the region. This group of patients experiences prolonged recovery [3, 4]. The systematic review “Post-COVID-19 Symptoms in Adults with Asthma” reported post-COVID-19 disorders in asthmatic individuals, including shortness of breath during exercise (75.4%) and at a rest (34.4%), cough (2.1%), and increased fatigue (21.2–65.6%). According to some data, asthmatics have a 1.5–2 times higher risk of these disorders than people without asthma [5].

A pressing issue remains the search for key links in the pathogenesis of persistent systemic inflammation in people with respiratory diseases in the post-COVID period. Current evidence suggests that SARS-CoV-2 directly or indirectly activates polyprotein complexes (inflammasomes) in the intracellular space. NOD-like receptors (NLRs) are a family of intracellular sensors that initiate the assembly of multi-protein complexes (inflammasomes) in response to tissue damage or pathogenic invasion. These structures act as a molecular platform for the activation of caspase-1, a cysteine protease that processes and secretes the active forms of the proinflammatory cytokines IL-1 β and IL-18 [6]. NLRP3, an inflammasome, is involved in the recognition of a wide range of viral, bacterial, and fungal agents, as well as endogenous danger signals, and has been characterized in great detail. The high level of NLRP3 expression in the respiratory tract epithelium and immunocompetent cells (monocytes, neutrophils, T-lymphocytes) determines its key role in modulating the respiratory immune response [7]. This mechanism triggers the pyroptotic pathway of inflammation, which may contribute to the formation of post-COVID disorders in patients with asthma [7, 8]. However, currently, there is limited information on the effect of an inflammasome-mediated inflammatory response on the long-term consequences of COVID-19 in people with asthma.

AIM

To determine prognostic criteria for worsening of mild bronchial asthma following an infectious disease caused by SARS-CoV-2.

MATERIALS AND METHODS

A total of 101 subjects were enrolled after providing written informed consent. Of these, 52 individuals

had mild persistent, partially controlled asthma and had experienced mild COVID-19 confirmed by PCR testing. Thirty-four patients had mild partially controlled asthma and had not been infected with SARS-CoV-2. The control group consisted of 15 individuals without asthma, chronic bronchopulmonary disease, and a history of COVID-19, as confirmed by PCR and ELISA tests at the time of sample collection. The study groups were comparable in terms of sex and age. 62% of them were women and 38% were men. The average age of the subjects was 42.0 ± 5.3 years. The diagnosis of bronchial asthma was verified based on the criteria of the Global Strategy for the Treatment and Prevention of Bronchial Asthma (GINA), Federal Clinical Guidelines, and in accordance with the International Classification of Diseases 10th Revision (ICD-10).

The ACQ-5 (Asthma Control Questionnaire) test was used to determine the level of disease control. A score from 0.75 to 1.5 indicates partially controlled bronchial asthma, and less than 0.75 indicates good asthma control [GINA 2024]. The study was conducted in accordance with the Declaration of Helsinki (1978, revised in 2024) and approved by the Ethics Committee of the Vladivostok branch of the Far Eastern Scientific Center for Physiology and Pathology of Respiration (Protocol No. 9, dated November 24, 2021).

Inclusion criteria were: mild partially controlled asthma. Before enrollment, patients with a confirmed diagnosis of mild asthma had been under regular follow-up for at least 1 year and were regularly receiving maintenance therapy with inhaled formoterol + budesonide (6+160 mcg/dose, 1–2 doses twice daily and as needed).

Exclusion criteria included acute infectious diseases, chronic internal organ diseases in the acute phase, decompensated chronic heart failure, and uncontrolled asthma.

Patients were enrolled in the first half of 2021 and examined at two time points: early (after recovery and confirmation of a negative SARS-CoV-2 test) and at 6 months. At the time of inclusion in the study and throughout the entire observation period, all subjects underwent PCR testing for SARS-CoV-2 and ELISA testing for immunoglobulins of class G (IgG) to SARS-CoV-2. Participants completed questionnaires on the course and condition of asthma, underwent ACQ testing, and providing information on the timing of COVID-19 infection, symptoms, prescribed medications, and allergic reactions. Post-COVID disorders included asthenic syndrome (increased fatigue, weakness, shortness of breath or palpitations, a feeling of air hunger, sweating; during significant, accelerated or particularly prolonged exertion (effort); impaired concentration, memory loss, sleep disorders, and anxiety). The state of the external pulmonary function (EPF) was assessed using a SPIROLAB III device, with measurement of forced vital lung capacity (FVC), forced expiratory volume in 1 second (FEV1), and the FEV1/FVC ratio. Values were recorded as absolute values and as percentages of predicted values. Disease control was assessed using the ACQ test.

Venous blood was collected in the morning on an empty stomach into tubes with a clot activator

for determination of the inflammatory markers NLRP3 and gasdermin D. Blood samples were centrifuged and serum was collected and frozen at -20°C , then stored at -80°C . ELISA was performed using a BEP 2000 analyzer (Siemens, Germany) with commercially available kits for NOD-like receptor protein 3 (NLRP3) (BIOMATIK ELISA Kit, Israel) and gasdermin D (GSDMD) (Human GSDMR Simple Step ELISA Kit Abcam, Great Britain) according to the manufacturers' instructions. The study of SARS-CoV-2 was conducted by quantitative ELISA method using Vector-Best JSC (Russia) on an ALISEI analyzer (Italy).

SARS-CoV-2 viral RNA was detected using the PCR and the SARS-CoV-2 AmpliTest test system. The DTprime detection amplifier (NPO DNA Technology LLC, Russia) and Rotor-Gene Q (Qiagen, Germany) were used. The study for the presence of antibodies to SARS-CoV-2 was carried out using the quantitative ELISA method, following the instructions for the reagent kits (Vector-Best JSC, Russia) on an ALISEI analyzer (Italy).

Statistical processing was performed using Statistica 10. Data was presented as medians and quartiles. Statistical significance was set at $p < 0.05$. Between-group differences were assessed using the Mann – Whitney and Kruskal – Wallis tests. The Bonferroni correction was used to solve the problem of multiple comparisons. Asthma patient values were compared with corresponding values from healthy individuals (control group), taken as 100 %.

ROC analysis was used to determine the cutoff thresholds for the investigated biomarkers. The ROC curve is a graphical representation that allows for the evaluation of the quality of a diagnostic test. It reflects the ratio between the proportion of true-positive test results [sensitivity of the test] and the proportion of false-positive test results [test specificity] with varying separation thresholds. The quantitative interpretation of the ROC curve

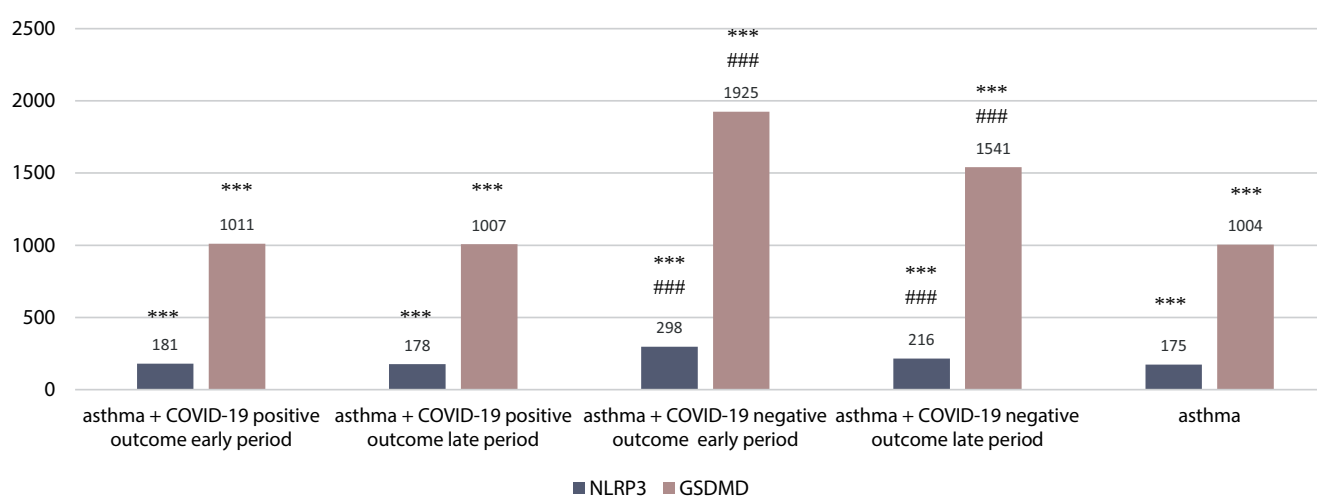
is provided by the AUC indicator. The higher the AUC value, the better the classifier. A value of 0.5 indicates the unsuitability of the chosen classification method [9].

RESULTS

Based on six-month follow-up results, the main group was divided into 2 subgroups of patients based on their clinical and laboratory status. In the subgroup with positive outcomes ($n = 34$, 67 %; no post-COVID disorders), normalization of clinical and functional parameters and a decrease in hyperinflammatory rates were observed during this period, reaching values comparable to those in the asthma group without SARS-CoV-2.

The following dynamics of inflammatory markers were observed in patients with a positive outcome: normalization of clinical and functional parameters and a decrease in hyperinflammation indicators to values comparable to those found in the group of individuals with A, who did not have SARS-CoV-2.

High levels of hyperinflammation markers (Fig. 1) were detected in the subgroup with negative outcomes ($n = 18$, 33 %; post-COVID disorders) at 6 months after COVID-19. These patients had symptoms indicative of persistent post-COVID disorders (asthenic syndrome with increased fatigue, weakness, shortness of breath or palpitations, a feeling of air hunger, sweating during significant, accelerated or particularly prolonged exertion; impaired concentration, memory loss, sleep disorders, and anxiety). In patients with a negative outcome, the following marker dynamics were observed: at the initial stage, they were significantly (nearly twice) higher than in the comparison group. At the late stage, there is a decrease in both indicators (NLRP3 by 82 % ($p \leq 0.001$), and gasdermin D by 384 % ($p \leq 0.001$)) relative to the initial values. However,



Note. Hyperinflammation markers are expressed as a percentage relative to the control group. * – significant difference compared to the control group; # – significant difference between favorable and unfavorable outcome subgroups. *** – $p < 0.001$, ### – $p < 0.001$.

FIG. 1.

NLRP3 and gasdermin D levels in patients with asthma following COVID-19, stratified by clinical outcome (positive and negative outcome)

NLRP3 levels is 41 % ($p \leq 0.01$) and gasdermin D is 537 % ($p \leq 0.001$) higher than the values of the comparison group. This contributes to prolonged recovery and, in some cases, to the development of post-COVID syndrome.

In the negative outcome group in the late period, asthma exacerbations were recorded 1.5–2 times more frequently than in the positive outcome group during the same period (Table). Pulmonary function parameters also differed between the groups. At an early stage, FVC was reduced by 8.8 %, and by 4.7 % at a late stage compared to the positive outcome group. FEV1 was 76.1 % at an early stage and 82.6 % at a late stage. The FEV1/FVC ratio was 60.4 % at an early stage and 65.5 % at a late stage suggesting mild obstruction (Table).

Based on the data obtained, cutoff values for hyperinflammation markers associated with adverse outcomes were determined for gasdermin D and NLRP3.

The threshold determination algorithm comprised three stages. First, an association between marker levels and post-COVID complication risk was assessed. The analysis showed statistically significant differences ($p < 0.05$) between NLRP3 levels in the positive outcome group and in the COVID-19 negative outcome group ($p < 0.001$). Statistically significant differences ($p < 0.05$) were found between gasdermin D levels in the COVID-19 positive outcome group compared to the negative outcome group ($p = 0.002$). Second, ROC analysis was used to determine the separating threshold for gasdermin D and NLRP3, and performance characteristics of the diagnostic test were evaluated. The optimal threshold value for gasdermin D was 0.38 ng/ml, with a sensitivity (Se) of 85 % and specificity (Sp) of 91 %. The area under the curve (AUC) was 0.89, indicating good quality for the diagnostic model (Fig. 2).

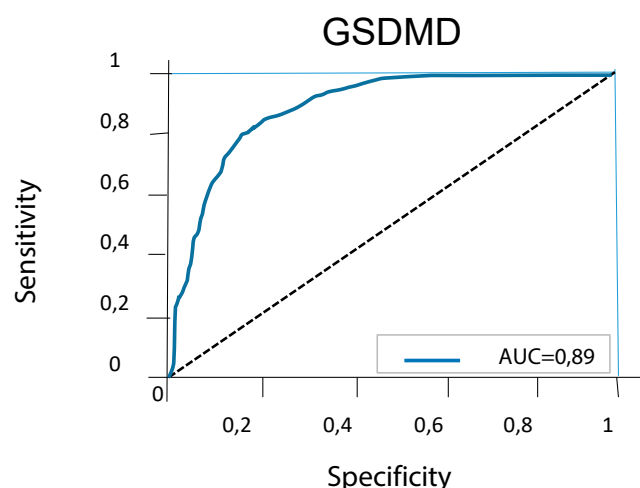


FIG. 2. ROC-curve analysis of gasdermin D for predicting adverse outcomes in asthma patients after COVID-19

For NLRP3, the cutoff value was determined at 0.04 ng/ml (Se = 82%, Sp = 87%, AUC = 0.858) (Fig. 3), also indicating good prognostic value of this marker.

DISCUSSION

The obtained data indicate the key pathogenetic role of the NLRP3 inflammasome in the initiation and maintenance of hyperinflammatory cascades in patients with asthma following COVID-19. This interaction is of particular interest, as it combines the chronic inflammatory processes characteristic of asthma and the virus-induced

TABLE

COMPARATIVE CLINICAL AND FUNCTIONAL CHARACTERISTICS OF THE STUDIED GROUPS AT DIFFERENT STAGES OF CONVALESCENCE

Parameter	Group with mild partially controlled asthma, positive outcome after COVID-19 (n=34)		Group with mild partially controlled asthma, negative outcome after COVID-19 (n=18)		Group with partially controlled mild asthma (n=34)
	Early stage	Late stage (after 6 months)	Early stage	Late stage (after 6 months)	
Number of asthma exacerbations		No more than 1 time in 6 months		1–2 times in 6 months	No more than 1 time in 6 months
ACQ test, points	1.2 *** (1.05–1.34)	1.01 *** (0.97–1.14)	***1.38 *** (1.38–1.45)	***1.43 *** (1.36–1.49)	***1.0 *** (0.91–1.06)
EPF parameters, % of predicted					
FVC % from the proper	94.2 *** (91.3–96.7)	95.4 *** (92.7–97.6)	***85.4 *** (82.2–89.7)	***90.7 *** (86.4–94.9)	***95.9 *** (93.8–99.2)
FEV1 % from the proper	88.6 *** (84.5–92.5)	90.2 *** (85.2–96.1)	***76.1 *** (70.2–79.5)	***82.6 *** (76.8–86.2)	***92.2 *** (87.5–96.3)
FEV1/FVC %	71.8 *** (65.9–75.8)	75.2 *** (70.4–79.6)	***60.4 *** (55.7–67.1)	***65.5 *** (61.3–67.6)	***77.9 *** (71.5–84.1)

Note. Right* indicates statistical significance compared to the control group; left* indicates differences between the subgroups with positive and negative outcomes. *** – $p < 0.001$.

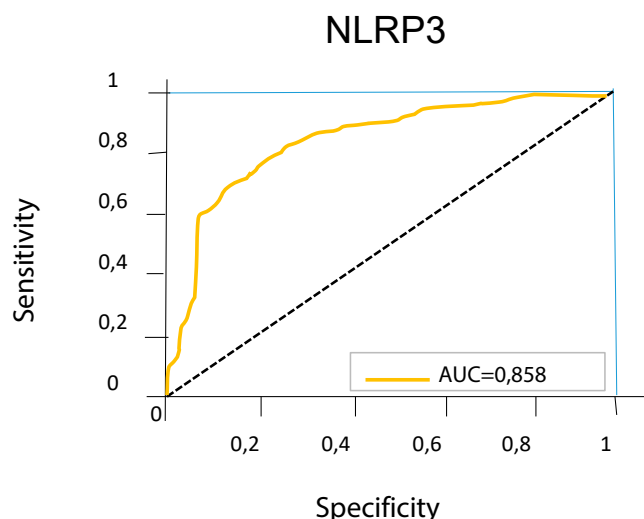


FIG. 3.
ROC-curve for NLRP3 in asthma patients with a history of COVID-19

inflammatory response in COVID-19. The combination of these factors creates a unique pathogenetic setting that predisposes to prolonged inflammation and the development of post-COVID disorders [1–3].

Inflammasomes, in particular the NLRP3 complex, are an important element of the innate immune defense, providing recognition of pathogens and triggering inflammatory cascades. NLRP3 refers to receptors that activate intracellular signaling pathways in response to pathogen-induced damage [5, 6].

The inflammatory response arising in this context in patients with asthma and COVID-19 becomes cyclical: virus-induced NLRP3 activation triggers excessive cytokine release, leading to epithelial damage of the respiratory tract, vascular endothelium injury, increased tissue sensitization, and NLRP3 reactivation [7]. Prolonged activation of NLRP3 and GSDMD can lead to persistent hyperinflammation and disruption of tissue repair mechanisms. The present study demonstrates that even six months after COVID-19, patients with bronchial asthma continue to have elevated NLRP3 and GSDMD levels, accompanied by asthenic syndrome, decreased pulmonary function, and impaired asthma control. These findings are consistent with previous studies reporting prolonged activation of proinflammatory pathways after COVID-19 in individuals with chronic bronchopulmonary disease [8–10].

The persistence of elevated concentrations of NLRP3 and GSDMD in patients with asthma after COVID-19 may explain the presence of clinical signs such as increased fatigue, shortness of breath, decreased exercise tolerance, and frequent asthma exacerbations. Hyperactivation of inflammasomes may not only contribute to the persistence of inflammation but also reduce the efficacy of maintenance therapy by decreasing sensitivity to inhaled glucocorticoids [11, 12]. In this study, ROC analysis demonstrated that NLRP3 and GSDMD levels have high diagnostic accuracy for predicting negative outcomes. These markers may serve as potential biomarkers

for the risk of post-COVID disorders and worsening of asthma control.

CONCLUSION

The results confirm the involvement of the NLRP3 and GSDMD in the formation of the hyperinflammation phenotype in patients with asthma in the post-COVID period. The developed prognosis method demonstrates that a concurrent elevation of NLRP3 above 0.04 ng/ml and GSDMD above 0.38 ng/ml indicates a high risk of worsening of the underlying disease, as well as the need for personalized therapy and long-term follow-up of this patient category. These findings open up prospects for further research aimed at developing new diagnostic and prognostic criteria for post-COVID disorders, as well as the identification of therapeutic strategies capable of modulating the activity of inflammasome-mediated inflammatory pathways in mild asthma following COVID-19.

Conflicts of interest

The authors declare no conflicts of interest.

REFERENCES

1. Assaf S, Stenberg H, Jesenak M, Tarasevych SP, Hanaia NA, Diamant Z. Asthma in the era of COVID-19. *Respir Med.* 2023; 218: 107373. doi: 10.1016/j.rmed.2023.107373
2. Kiseleva NV, Tarlovskaya EI, Boldina MV. Features of the course of bronchial asthma in patients who have suffered a new coronavirus infection. *South Russian Journal of Therapeutic Practice.* 2023; 4(4): 34–40. (In Russ.). doi: 10.21886/2712-8156-2023-4-4-34-40
3. Philip KEJ, Buttery S, Williams P, Vijayakumar B, Tonkin J, Cumella A, et al. Impact of COVID-19 on people with asthma: a mixed methods analysis from a UK wide survey. *BMJ Open Respir Res.* 2022; 9(1): e001056. doi: 10.1136/bmjresp-2021-001056
4. Kruglova OS, Sobko EA, Demko IV, Kraposhina AY, Khramova YuA, Geyl' SA. Asthma and COVID-19 (review). *Bulleten' fiziologii i patologii dyhaniâ = Bulletin Physiology and Pathology of Respiration.* 2025; (95): 135–148 (In Russ.). doi: 10.36604/1998-5029-2025-95-135-148
5. Osipova VV, Osipova GL, Zaryanova EA, Terekhov DV. Asthma and COVID-19: An overview of the guidelines for the management of asthma patients during the COVID-19 pandemic. *Pul'monologiya.* 2021; 31(5): 663–670 (In Russ.). doi: 10.18093/0869-0189-2021-31-5-663-670
6. Swanson KV, Deng M, Ting JP. The NLRP3 inflammasome: molecular activation and regulation to therapeutics. *Nat Rev Immunol.* 2019; 19(8): 477–489. doi: 10.1038/s41577-019-0165-0
7. Branchett WJ, Lloyd CM. Regulatory cytokine function in the respiratory tract. *Mucosal Immunol.* 2019; 12(3): 589–600. doi: 10.1038/s41385-019-0158-0

8. Lobova TG, Vitkina TI. Assessment of the inflammatory response in convalescents of a new coronavirus infection in the catamnesis. *Acta Biomedica Scientifica*. 2025; 10(1): 115–122. doi: 10.29413/ABS.2025-10.1.12
9. Sharashova EE, Kholmatova KK, Gorbatova MA, Grijbovski AM. Application of the multivariable logistic regression analysis in healthcare using SPSS software. *Nauka i Zdravookhranenie [Science & Healthcare]*. 2017; 4: 5–26.
10. Garrigues E, Janvier P, Kherabi Y, Le Bot A, Hamon A, Gouze H, et al. Post-discharge persistent symptoms and health-related quality of life after hospitalization for COVID-19. *J Infect*. 2020; 81(6): e4–e6. doi: 10.1016/j.jinf.2020.08.029
11. Swanson KV, Deng M, Ting JPY. The NLRP3 inflammasome: molecular activation and regulation to therapeutics. *Nat Rev Immunol*. 2019; 19(8): 477–89. doi: 10.1038/s41577-019-0165-0
12. Borukaeva IKh, Edilov KG, Dzueva AS, Labazanova MI, Gidizov Kh-AR. The Role of the NLRP3 Inflammasome in the Pathogenesis of Bronchial Asthma: Inflammation Mechanisms and emerging Therapeutic Perspectives. *Kazan Medical Journal*. 2025; 106(2): 287–297. (In Russ.). doi: 10.17816/KMJ646818

Information about the authors

Tatyana G. Lobova – Graduate Student, Vladivostok Branch of the Far Eastern Scientific Center of Physiology and Pathology of Respiration – Institute of Medical Climatology and Rehabilitative Treatment; e-mail: lobova.tg@dvfu.ru, <https://orcid.org/0000-0003-1721-6548>

Tatyana I. Vitkina – Dr. Sc. (Med.), Professor, Corresponding Member of the Russian Academy of Sciences, Head of the Laboratory of Medical Ecology and Recreational Resources, Vladivostok Branch of the Far Eastern Scientific Center of Physiology and Pathology of Respiration – Institute of Medical Climatology and Rehabilitative Treatment; <https://orcid.org/0000-0002-1009-9011>