Л.Н. Гумилев атындағы Еуразия ұлттық университетінің ХАБАРШЫСЫ.
BULLETIN of L.N. Gumilyov Eurasian National University.
ВЕСТНИК Евразийского национального университета имени Л.Н. Гумилева.
ISSN: 2616-7034. eISSN: 2663-130X

IRSTI 34.15.25
Research article

https://doi.org/10.32523/2616-7034-2025-152-3-7-19

Study of nitric oxide levels in the serum of patients with bronchial asthma

A. Sabirova[1], A. Aitkazina[2], A. Kussainova[1], O. Bulgakova[1], R. Bersimbaev[1], A. Aripova*[1]

[1]*L.N. Gumilyov Eurasian National University, Astana, Kazakhstan*
[2]*City Multidisciplinary Hospital No. 2, Astana, Kazakhstan*

Abstract. This preliminary research article is dedicated to the determination of nitric oxide (NO) levels in the serum of patients with bronchial asthma (BA). Nitric oxide plays an important role in the pathogenesis of bronchial asthma, as it contributes to bronchoconstriction and stimulates the production of inflammatory mediators, thereby exacerbating the disease. The study included 41 participants, comprising patients with BA of varying disease control categories (controlled, partially controlled, and uncontrolled asthma) as well as individuals without a diagnosis of BA (control group). The NO level was measured using the "Nitric Oxide (total)" assay kit (ADI-917-020) from Enzo Life Sciences Inc. (USA). The results demonstrated that serum NO levels in patients with BA were significantly elevated, approximately 1.34 times compared to the control group (p=0.001). Additionally, the concentration of nitric oxide was analyzed according to sex and age. A statistically significant difference was found between women older than 65 years and those younger (p=0.0347). When comparing serum, NO concentrations between partially controlled and uncontrolled asthma groups with the control group, NO levels were elevated by 1.31 times and 1.42 times, respectively (p=0.0355 and p=0.0033). ROC analysis of nitric oxide levels for the diagnosis of bronchial asthma revealed acceptable diagnostic accuracy (AUC=0.6932; p=0.013). Thus, the determination of nitric oxide concentration in the serum of patients with bronchial asthma may play an important role in diagnosis and could serve as a potential biomarker of the disease.
Keywords: Inflammation, Bronchial asthma, Nitric oxide, Serum biomarker, Serum nitric oxide levels

Introduction

Bronchial asthma (BA) is currently considered one of the most prevalent and clinically significant chronic respiratory diseases. According to the World Health Organization, over 300 million people worldwide are affected by bronchial asthma, and its global prevalence continues to rise steadily each year. These trends underscore the growing medical and social relevance of this condition [1,2].

Received: 29.07.2025. Accepted: 30.09.2025. Available online: 30.09.2025.

**Corresponding author:aripova001@gmail.com*

BA is a disease characterized by chronic airway inflammation and is primarily driven by immunological mechanisms. In its pathogenesis, inflammatory mediators – particularly molecules such as nitric oxide (NO) – play a crucial role. Nitric oxide is a key biological molecule that actively participates in airway inflammation, influencing numerous cellular processes and contributing to various physiological and pathophysiological functions in the body. It is endogenously synthesized by nitric oxide synthase (NOS) enzymes and plays an important role in airway dilation, regulation of blood circulation, and intercellular signaling [3–4].

During the inflammatory process in bronchial asthma, the production of nitric oxide (NO) is enhanced, leading to elevated levels in both the blood and airways. The use of biomarkers allows for accurate assessment of disease progression, effective treatment planning, and timely monitoring of the patient's condition – key priorities in clinical practice. Measuring nitric oxide levels in blood serum provides deeper insight into the pathogenesis of bronchial asthma and supports the development of personalized therapeutic strategies. Therefore, investigating the molecular-genetic and immunological aspects of bronchial asthma remains highly relevant.

This study aimed to determine the concentration of nitric oxide (NO) in the blood serum of patients diagnosed with bronchial asthma.

Materials and research methods

Collection of Research Materials

The study included 33 patients diagnosed with bronchial asthma (22 women and 11 men, aged 36 to 90 years) and 8 healthy individuals who served as the control group (Table 1). All participants had been residing in Kazakhstan for at least the past five years. The patients received treatment in the pulmonology department of City Hospital No. 2 in Astana. The diagnosis was established in accordance with the Global Initiative for Asthma (GINA) guidelines. Additionally, the "Nijmegen Questionnaire" and "Asthma Control Questionnaire" were administered.

The control group consisted of healthy volunteers with no history of neurological, autoimmune, allergic, or chronic diseases; no diabetes; no signs of inflammation; and no recent episodes of hyperthermia or heatstroke within the past 2–3 weeks.

Peripheral blood samples (9 mL) were collected in EDTA-K2 tubes with informed consent from the participants and ethical approval. The samples were processed at the Molecular Genetics Laboratory of the Institute of Cellular Biology and Biotechnology, L.N. Gumilyov Eurasian National University.

The patients were categorized into three groups:

Controlled BA – no or rare symptoms;

Partially controlled BA – occasional symptoms;

Uncontrolled BA – persistent symptoms and poor treatment response.

Separation of blood components

Whole blood was stored at +4°C for 4 hours, after which the plasma was separated by centrifugation at 3000g for 10 minutes. The leukocyte-free plasma was then stored at –80°C until analysis.

Table 1

Characteristics of the samples included in the study

Parameters	Control group	Patients with diagnosed BA	p-value
Number of participants	8	33	0,1374
Mean age	48	63	0,1600
Sex (female/male)	3/5	22/11	0,7927
Controlled BA	-	7	0,2593
Partially controlled BA	-	13	
Uncontrolled BA	-	13	

Determination of nitric oxide (NO) concentration

Plasma samples were diluted with reaction buffer at ratios ranging from 1:2 to 1:20 and filtered using a 10,000 MWCO ultrafiltration unit. The NO level was measured using the "Nitric Oxide (total)" assay kit (ADI-917-020) from Enzo Life Sciences Inc. (USA).

Statistical analysis

Intergroup differences in NO concentration were analyzed using Student's t-test and the Mann–Whitney U test in GraphPad Prism 8.0 software. A p-value of less than 0.05 was considered statistically significant.

Results

Determination of nitric oxide concentration in the blood serum of the bronchial asthma and control groups

In this study, the concentration of nitric oxide (NO) in the blood serum of patients diagnosed with bronchial asthma and healthy controls was determined using enzyme-linked immunosorbent assay (ELISA). A total of 33 patients with bronchial asthma and 8 healthy volunteers were included. Based on the clinical features of disease progression, the asthma group was subdivided into controlled, partially controlled, and uncontrolled subgroups. This classification was based on the frequency and severity of symptoms, pulmonary function parameters, and responsiveness to treatment.

The analysis assessed statistical differences between the asthma and control groups and identified changes in immune response levels. As shown in Figure 1, the NO concentration in the blood serum, measured at a wavelength of 570 nm, was compared across the study groups. The results revealed that the mean nitric oxide level in the control group was 50.95 μmol/L, whereas in patients with bronchial asthma, it was 68.09 μmol/L, indicating a 1.34-fold increase in the asthma group compared to healthy individuals.

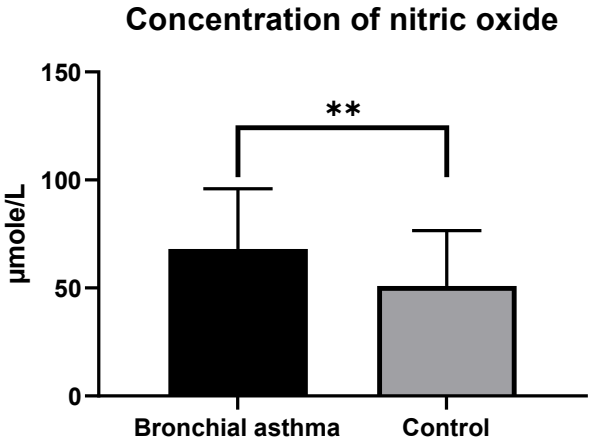

Figure 1. Nitric oxide concentration in the study groups (** p=0.001)

Analysis of nitric oxide concentration in the blood serum of bronchial asthma patients according to sex and age

In this study, we analyzed the concentration of nitric oxide (NO) in the blood serum of patients with bronchial asthma based on sex and age. Among male patients, the average NO concentration was 67 µmol/L, while among female patients it was 68 µmol/L. No statistically significant difference was observed between the sexes.

Further analysis was conducted to evaluate age-related differences. Among patients under the age of 65, the NO concentration was 75 µmol/L in women and 72 µmol/L in men. In contrast, in the group over 65 years of age, the NO levels were 58 µmol/L in women and 61 µmol/L in men. A statistically significant difference was found in NO levels between the younger and older age groups among female patients (p = 0.0347) (Figure 2).

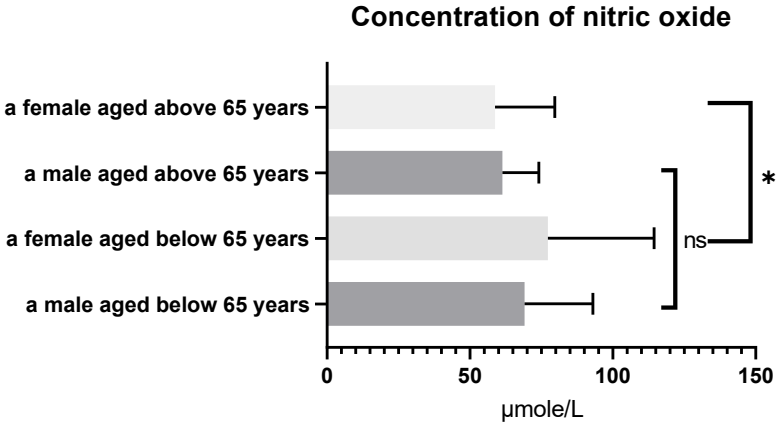

Figure 2. Age-related changes in nitric oxide (NO) concentration in the study groups (*p = 0.0347)

Analysis of nitric oxide concentration in blood serum according to asthma control level

According to the recommendations of the World Health Organization (WHO) and the Global Initiative for Asthma (GINA), bronchial asthma is classified into three categories based on symptom control and disease severity: controlled asthma, partially controlled asthma, and

uncontrolled asthma [5]. In this study, a total of 33 patients diagnosed by a pulmonologist at City Hospital No. 2 in Astana were included: 7 with controlled asthma, 13 with partially controlled asthma, and 13 with uncontrolled asthma.

When comparing the serum, NO levels between the asthma subgroups and the healthy control group, the average NO concentration in the controlled asthma group was 63.38 μmol/L, which was 1.24 times higher than in the control group (50.95 μmol/L). However, this difference was not statistically significant (p = 0.2593).

In contrast, patients with partially controlled asthma had a mean NO level of 66.53 μmol/L, significantly higher than the control group (p = 0.0355), representing a 1.31-fold increase (Figure 3a). These findings indicate that nitric oxide levels are associated with asthma control status and support the role of NO as a marker of airway inflammation [6].

Furthermore, the mean NO concentration in patients with uncontrolled asthma was 72.19 μmol/L, which is approximately 1.42 times higher than in the control group (50.95 μmol/L). This difference was statistically significant (p = 0.0033), clearly demonstrating that NO levels increase in patients with poorly controlled asthma (Figure 3b).

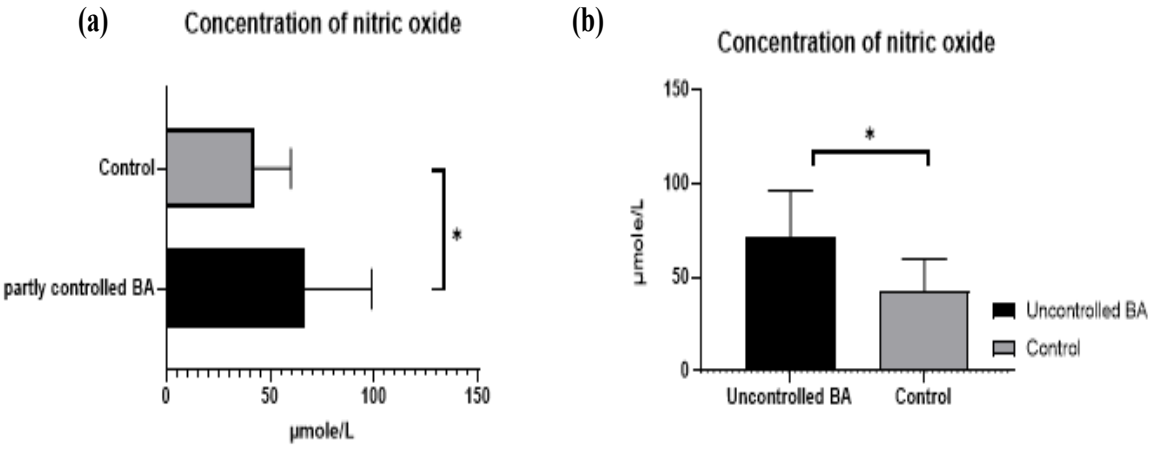

Figure 3. (a) Comparative results of nitric oxide (NO) concentration in blood plasma between patients with partially controlled bronchial asthma (BA) and the control group (measured at a wavelength of 570 nm). (b) Comparative results of nitric oxide (NO) concentration in blood plasma between patients with uncontrolled bronchial asthma (BA) and the control group (measured at a wavelength of 570 nm).

ROC curve analysis of nitric oxide levels

A Receiver Operating Characteristic (ROC) curve analysis was performed based on nitric oxide (NO) concentration to assess how effectively NO levels differentiate patients with bronchial asthma from healthy individuals, and to evaluate its potential use as a diagnostic biomarker. The area under the ROC curve (AUC) was found to be 0.6932 (p = 0.013), indicating a moderate diagnostic value of nitric oxide concentration for identifying the disease.

The analysis showed a sensitivity of 69.4% (0.69375) and a specificity of 53.9% (0.5386). The 95% confidence interval (CI) for the AUC ranged from 0.5456 to 0.8408 (Figure 4).

These findings suggest that while nitric oxide concentration alone can serve as a supportive diagnostic tool for bronchial asthma, its moderate sensitivity and specificity indicate that it may be most effective when used in combination with additional biomarkers to improve diagnostic accuracy.

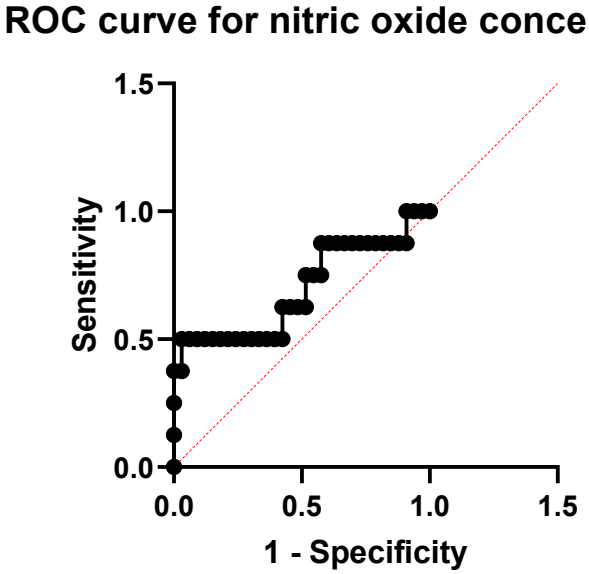

Figure 4. ROC analysis of nitric oxide levels for the diagnosis of bronchial asthma (AUC = 0.6932, p = 0.013)

Discussion

Since the early 2000s, the role of nitric oxide (NO) in the pathophysiology of airway inflammatory diseases has been the focus of active research [6-9]. Barnes [10] was the first to suggest that NO produced by nNOS (neuronal nitric oxide synthase) contributes to bronchodilation through the activation of NANC (non-adrenergic non-cholinergic) nerves. In contrast, eNOS (endothelial nitric oxide synthase) induces vasodilation and plasma extravasation, whereas excessive iNOS (inducible nitric oxide synthase) activity enhances mucus secretion, promotes eosinophil recruitment, and sustains Th2-mediated inflammation [11-15].

It has been established that NO concentrations are elevated in the exhaled air of patients with asthma [16], as well as in experimental models of chronic inflammation [13, 17]. This increase is mainly attributed to iNOS expression [10]. Treatment with inhaled corticosteroids (ICS) reduces FeNO (fractional exhaled nitric oxide) levels [18], whereas β2-agonists have little to no significant effect [19]. Administration of NOS inhibitors (L-NAME, aminoguanidine) likewise decreases FeNO [20]. Clinical studies further confirm that FeNO reflects inflammatory activity and assists in identifying the most severe asthma phenotypes, although several reports have failed to demonstrate a direct correlation with airway eosinophilia [21].

Despite some controversy, most authors recognize FeNO as a valuable biomarker for monitoring airway inflammation and assessing treatment efficacy [22,23]. In experimental

settings, NO donors induced smooth muscle relaxation and reduced bronchospasm, whereas NOS inhibition, by contrast, enhanced bronchoconstriction. The role of NO as a modulator of distal airway tone remains an area of ongoing investigation [24, 25].

Considerable attention has also been directed to the involvement of NO in airway remodeling. Evidence suggests that iNOS contributes to excessive deposition of collagen and elastin, whereas NO derived from cNOS (constitutive nitric oxide synthase) may exert protective effects [13, 26]. Positive correlations have been reported between NO metabolites and markers of remodeling, as well as with activation of arginase and matrix metalloproteinases [15, 27-29]. In addition, iNOS-driven NO production can lead to the formation of peroxynitrite and isoprostanes (8-iso-PGF2α), which further enhance bronchoconstriction and oxidative stress [30].

In parallel, extensive clinical data have accumulated supporting the diagnostic and prognostic value of FeNO. Across all systematically analyzed studies, FeNO is regarded as a reliable indicator of Th2-mediated inflammation with diagnostic, prognostic, and therapeutic relevance [31-36]. For example, de Abreu et al. [33] demonstrated that FeNO levels >30 ppb are associated with uncontrolled asthma and a decrease in response to ICS, making this parameter a convenient tool for monitoring treatment efficacy. Maniscalco et al. [34] further elaborated on the mechanisms of NO synthesis in asthma and emphasized the role of FeNO in disease phenotyping, particularly in Th2-high forms, where targeted biologics directed against IL-4, IL-5, and IL-13 have proven effective.

A large-scale meta-analysis by Murugesan et al., comprising 43 studies and over 16,000 patients, confirmed the high diagnostic accuracy of FeNO (sensitivity 72%, specificity 75%, AUC = 0.81) [35]. The greatest value was observed in children and in patients with eosinophilic inflammation. Findings by Nguyen et al. [36] demonstrated that patients with elevated FeNO levels had lower asthma control scores (ACT) and reduced FEV1. Also confirmed the clinical applicability of FeNO, emphasizing its role in optimizing ICS prescription and supporting a personalized approach to treatment [37].

In pediatric practice, FeNO is of particular importance, as nitric oxide levels are elevated in children with asthma and correlate with sensitization to aeroallergens—an observation critical for phenotyping and for the development of clinical guidelines [31, 33].

Despite the substantial body of evidence, certain limitations remain: FeNO levels may vary depending on smoking status, allergic rhinitis, infections, and ICS therapy. Accordingly, most experts recommend using FeNO as part of a multimodal assessment, alongside blood eosinophil counts, serum IgE, ACT scores, and spirometry.

In summary, nitric oxide plays a dual role in the pathogenesis of asthma: it contributes to bronchodilation and protects the extracellular matrix, yet excessive iNOS expression promotes inflammation, remodeling, and oxidative stress. FeNO, as a surrogate marker of these processes, has established itself as a valuable adjunct biomarker for the diagnosis, monitoring, and personalization of asthma therapy.

Conclusion

In conclusion, the assessment of nitric oxide (NO) levels stands out as a reliable, accessible, and informative biomarker that enables the objective evaluation of the clinical condition of

patients with bronchial asthma, the precise determination of airway inflammatory activity, and the assessment of disease control. The significant variation in NO levels depending on asthma phenotypes and control status supports its broad applicability in clinical practice, particularly for personalized therapy and disease monitoring.

Given that NO measurement is non-invasive, rapid, and easily repeatable, it can serve as a valuable supplementary tool for the early diagnosis of asthma and for monitoring treatment response. Moreover, the observed differences related to age and asthma control status further emphasize the clinical importance of this biomarker.

Future studies that integrate nitric oxide levels with other biomarkers in a comprehensive approach may provide deeper insights into asthma pathogenesis and contribute to the development of more precise therapeutic strategies.

Author Contributions

A.A.*(A. Aripova) – concept and supervision of the work; **A.A. (A. Aitkazina) –** collection of materials, medical expertise, interpretation of clinical data; **A.S. –** conducting the experiments; **A.A.*, A.K., O.B. –** discussion of the research results; **A.S., A.A.*** – writing the text; **A.A.*, R.B. –** editing the text of the article.

Funding

This research is funded by the Science Committee of the Ministry of Science and Higher Education of the Republic of Kazakhstan (AP26100907, “Identification of molecular-epigenetic markers for asthma – chronic obstructive pulmonary disease overlap syndrome for molecular diagnostics and personalized medicine”).

Conflicts of Interest

The authors declare no conflicts of interest.

Compliance with ethical standards

Peripheral blood samples for this study were collected from participants (patients and control group) with their informed consent and with the approval of the Ethics Committee of JSC "Astana Medical University" (Protocol No. 4 dated September 7, 2017).

References

1. World Health Organization. Asthma [Internet]. Geneva: WHO; [cited 2025 Feb 23]. Available from: https://www.who.int/news-room/fact-sheets/detail/asthma

2. Global Initiative for Asthma. Global strategy for asthma management and prevention. CiNii Research; 2023.

3. Abdulwehab S, Kedir F. Patient autonomy in self-managing their bronchial asthma exacerbation and its associated factors. Front Allergy. 2024;25;5:1483897. doi.org/doi:10.3389/falgy.2024.1483897

4. Tiotiu AI, Novakova P, Nedeva D, et al. Impact of air pollution on asthma outcomes. Int J Environ Res Public Health. 2020;27;17(17):6212. doi.org/10.3390/ijerph17176212

5. Нефедов ИВ, Шишиморов ИН, Магницкая ОВ. Оксид азота в выдыхаемом воздухе как предиктор эффективности базисной терапии бронхиальной астмы у детей. Научные результаты биомедицинских исследований. 2020;6(2):238–248. doi.org/10.18413/2658-6533-2020-6-2-0-8

6. Kobayashi K, Nishimura Y, Yamashita T, et al. The effect of overexpression of endothelial nitric oxide synthase on eosinophilic lung inflammation in a murine model. Int Immunopharmacol. 2006;6(7):1040–1052. doi.org/10.1016/j.intimp.2005.09.016.

7. Garvey EP, Oplinger JA, Furfine ES, et al. 1400W is a slow, tight binding, and highly selective inhibitor of inducible nitric-oxide synthase in vitro and in vivo. J Biol Chem. 1997;272(8):4959–4963. doi.org/10.1074/jbc.272.8.4959.

8. Van Camp JR, Yian C, Lupinetti FM. Regulation of pulmonary vascular resistance by endogenous and exogenous nitric oxide. Ann Thorac Surg. 1994;58(4):1025–1030. doi.org/10.1016/0003-4975(94)90448-0.

9. Poon BY, Raharjo E, Patel KD, Tavener S, Kubes P. Complexity of inducible nitric oxide synthase: cellular source determines benefit versus toxicity. Circulation. 2003;108(9):1107–1112. doi.org/10.1161/01.CIR.0000086321.04702.AC.

10. Barnes PJ. NO or no NO in asthma? Thorax. 1996;51(2):218–220. doi.org/10.1136/thx.51.2.218.

11. Meurs H, Maarsingh H, Zaagsma J. Arginase and asthma: novel insights into nitric oxide homeostasis and airway hyperresponsiveness. Trends Pharmacol Sci. 2003;24(9):450–455. doi.org/10.1016/S0165-6147(03)00227-X.

12. Prado CM, Leick-Maldonado EA, Kasahara DI, et al.Effects of acute and chronic nitric oxide inhibition in an experimental model of chronic pulmonary allergic inflammation in guinea pigs. Am J Physiol Lung Cell Mol Physiol. 2005;289(4):L677–L683. doi.org/10.1152/ajplung.00010.2005.

13. Prado CM, Leick-Maldonado EA, Yano L, et al. Effects of nitric oxide synthases in chronic allergic airway inflammation and remodeling. Am J Respir Cell Mol Biol. 2006;35(4):457–465. doi.org/10.1165/rcmb.2005-0391OC.

14. Schuiling M, Meurs H, Zuidhof AB, et al. Dual action of iNOS derived nitric oxide in allergen-induced airway hyperreactivity in conscious, unrestrained guinea pigs. Am J Respir Crit Care Med. 1998;158(5):1442–1449. doi.org/10.1164/ajrccm.158.5.9803027.

15. Meurs H, McKay S, Maarsingh H, et al. Increased arginase activity underlies allergen-induced deficiency of cNOS-derived nitric oxide and airway hyperresponsiveness. Br J Pharmacol. 2002;136(3):391–398. doi.org/10.1038/sj.bjp.0704725.

16. Kharitonov SA, Yates D, Robbins RA, et al. Increased nitric oxide in exhaled air of asthmatic patients. Lancet. 1994;343(8890):133–135. doi.org/10.1016/S0140-6736(94)90931-8.

17. Leick-Maldonado EA, Kay FU, Leonhardt MC, et al. Comparison of glucocorticoid and cysteinyl leukotriene receptor antagonist treatments in an experimental model of chronic airway inflammation in guinea-pigs. Clin Exp Allergy. 2004;34(1):145–152. doi.org/10.1111/j.1365-2222.2004.01854.x.

18. Yates DH, Kharitonov SA, Thomas PS, Barnes PJ. Endogenous nitric oxide is decreased in asthmatic patients by an inhibitor of inducible nitric oxide synthase. Am J Respir Crit Care Med. 1996;154(1):247–250. doi.org/10.1164/ajrccm.154.1.8680689.

19. Yates DH, Kharitonov SA, Barnes PJ. Effect of short- and long-acting inhaled β2-agonists on exhaled nitric oxide in asthmatic patients. Eur Respir J. 1997;10(7):1483–1488. doi.org/10.1183/09031936.97.10071483.

20. Brindicci C, Ito K, Barnes PJ, Kharitonov SA. Effect of an inducible nitric oxide synthase inhibitor on differential flow-exhaled nitric oxide in asthmatic patients and healthy volunteers. Chest. 2007;132(2):581–588. doi.org/10.1378/chest.06-3046.

21. Dweik RA, Sorkness RL, Wenzel S, et al. Use of exhaled nitric oxide measurement to identify a reactive, at-risk phenotype among patients with asthma. Am J Respir Crit Care Med. 2010;181(10):1033–1041. doi.org/10.1164/rccm.200905-0695OC.

22. Pisi R, Aiello M, Tzani P, Marangio E, Olivieri D, Chetta A. Measurement of fractional exhaled nitric oxide by a new portable device: comparison with the standard technique. J Asthma. 2010;47(7):805–809. doi.org/10.3109/02770903.2010.485667.

23. Kharitonov SA, Barnes PJ. Clinical aspects of exhaled nitric oxide. Eur Respir J. 2000;16(4):781–792. doi.org/10.1183/09031936.00.16478100.

24. Kraft M. Part III: location of asthma inflammation and the distal airways: clinical implications. Curr Med Res Opin. 2007;23(Suppl 3):S21–S27. doi.org/10.1185/030079907X226177.

25. Angeli P, Prado CM, Xisto DG, et al. Effects of chronic L-NAME treatment lung tissue mechanics, eosinophilic and extracellular matrix responses induced by chronic pulmonary inflammation. Am J Physiol Lung Cell Mol Physiol. 2008;294(6):L1197–L1205. doi.org/10.1152/ajplung.00199.2007.

26. Eynott PR, Groneberg DA, Caramori G, et al. Role of nitric oxide in allergic inflammation and bronchial hyperresponsiveness. Eur J Pharmacol. 2002;452(1):123–133. doi.org/10.1016/S0014-2999(02)02237-9.

27. Ricciardolo FLM. Multiple roles of nitric oxide in the airways. Thorax. 2003;58(2):175–182. doi.org/10.1136/thorax.58.2.175.

28. Meurs H, Maarsingh H, Zaagsma J. Arginase and asthma: novel insights into nitric oxide homeostasis and airway hyperresponsiveness. Trends Pharmacol Sci. 2003;24(9):450–455. doi.org/10.1016/S0165-6147(03)00227-X.

29. Ohbayashi H, Shimokata K. Matrix metalloproteinase-9 and airway remodeling in asthma. Curr Drug Targets Inflamm Allergy. 2005;4(2):177–181. doi.org/10.2174/1568010053586246.

30. Sugiura H, Ichinose M. Oxidative and nitrative stress in bronchial asthma. Antioxid Redox Signal. 2008;10(4):785–797. doi.org/10.1089/ars.2007.1937.

31. Haccuria A, Michils A, Michiels S, Van Muylem A. Exhaled nitric oxide: a biomarker integrating both lung function and airway inflammation changes. J Allergy Clin Immunol. 2014;134(3):554–559. doi.org/10.1016/j.jaci.2013.12.1070.

32. Backman K, Piippo-Savolainen E, Arffman M, Korppi M. Exhaled nitric oxide is a reliable biomarker of asthma in children with aeroallergen sensitization but not in other children. Pediatr Allergy Immunol. 2023;34(2):e14044. doi.org/10.1111/pai.14044.

33. de Abreu FC, da Silva Júnior JLR, Rabahi MF. The Fraction Exhaled Nitric Oxide as a Biomarker of Asthma Control. Biomark Insights. 2019;14:1177271919826550. doi.org/10.1177/1177271919826550.

34. Maniscalco M, Bianco A, Mazzarella G, Motta A. Exhaled Nitric Oxide in Asthma: From Bench to Clinical Practice. Cells. 2023;12(21):2518. doi.org/10.3390/cells12212518.

35. Murugesan K, Alzahrani S, Parambil JV, et al. Diagnostic and Prognostic Value of Exhaled Nitric Oxide in Asthma: A Comprehensive Meta-Analysis of 43 Studies. Diagnostics (Basel). 2023;13(8):1428. doi.org/10.3390/diagnostics13081428.

36. Nguyen VN, Phan NY, Hoang TV, et al. Clinical correlation between fractional exhaled nitric oxide (FeNO) levels and asthma control in Vietnamese patients. J Thorac Dis. 2020;12(2):541–550. doi.org/10.21037/jtd

37. Makarova SA, Kazina VV, Mineeva AV, et al. Fractional exhaled nitric oxide as a marker of bronchial inflammation in asthma: clinical and diagnostic value. Mod Rehabil Med. 2024;4(1):64–70.

Бронх демікпесі бар науқастардың қан сарысуынан азот оксидінің деңгейін зерттеу

А.Б. Сабирова[1], А.Т. Айтказина[2], А.А. Кусаинова[1], О.В. Булгакова[1], Р.І. Берсімбай[1], А.А.Арипова*[1]
[1]Л.Н. Гумилев атындағы Еуразия ұлттық университеті, Астана, Қазақстан
[2]№2 Қалалық көпсалалы аурухана, Астана, Қазақстан

Аңдатпа. Бұл алдын ала зерттеу жұмысы бронх демікпесі (БД) бар науқастардың сарысуындағы азот оксиді (NO) деңгейін анықтауға арналған. Азот оксиді БД патогенезінде маңызды рөл атқарады, өйткені ол бронхтардың тарылуына ықпал етіп, қабыну медиаторларының түзілуін ынталандырады, бұл өз кезегінде аурудың үдеуіне әкеледі. Зерттеуге 41 қатысушы енді, олардың қатарында ауруды бақылау деңгейі әртүрлі БД науқастары (бақыланатын, ішінара бақыланатын және бақыланбайтын астма) және БД диагнозы қойылмаған адамдар (бақылау тобы) болды. NO деңгейі Enzo Life Sciences Inc. (АҚШ) шығарған "Nitric Oxide (total)" (ADI-917-020) талдау жиынтығы арқылы өлшенді. Зерттеу нәтижелері БД бар науқастарда сарысудағы азот оксиді деңгейі бақылау тобымен салыстырғанда шамамен 1,34 есеге жоғары екенін көрсетті (p=0,001). Сонымен қатар, азот оксидінің концентрациясы жынысы мен жасына қарай талданды. 65 жастан асқан әйелдер мен жас әйелдер арасында статистикалық тұрғыдан маңызды айырмашылық анықталды (p=0,0347). Ішінара бақыланатын және бақыланбайтын БД бар науқастарды бақылау тобымен салыстырғанда NO деңгейі тиісінше 1,31 және 1,42 есе жоғары болды (p=0,0355 және p=0,0033). Бронх демікпесін диагностикалау үшін азот оксиді деңгейінің ROC талдауы қанағаттанарлық диагностикалық дәлдікті көрсетті (AUC=0,6932; p=0,013). Осылайша, бронх демікпесі бар науқастардың сарысуындағы азот оксидінің концентрациясын анықтау диагноз қоюда маңызды рөл атқаруы мүмкін және аурудың ықтимал биомаркері ретінде қолдануға болады.

Түйін сөздер: қабыну, бронх демікпесі, азот оксиді, қан сарысу биомаркері, қан сарысуындағы азот оксидінің деңгейі

Исследование уровня оксида азота в сыворотке крови у пациентов с бронхиальной астмой

А.Б. Сабирова[1], А.Т. Айтказина[2], А.А. Кусаинова[1], О.В. Булгакова[1], Р.И. Берсимбаев[1], А.А.Арипова*[1]
[1]Евразийский национальный университет имени Л.Н. Гумилева, Астана, Казахстан
[2]Городская многопрофильная больница №2, Астана, Казахстан

Аннотация. Это предварительная исследовательская статья посвящена определению уровня оксида азота в сыворотке крови у пациентов с бронхиальной астмой (БА). Оксид азота играет важную роль в патогенезе бронхиальной астмы, так как приводит к сокращению бронхов и стимулирует выработку воспалительных медиаторов. В исследование был включен 41 участник, среди которых пациенты с БА различных категорий (контролируемая, частично контролируемая и неконтролируемая астма), а также лица без диагноза БА (контрольная группа). Уровень

оксида азота измеряли с использованием набора для анализа “Nitric Oxide (total)” (ADI-917-020) компании Enzo Life Sciences Inc. (США). Результаты показали, что у пациентов с бронхиальной астмой уровень оксида азота в сыворотке крови был в 1,34 раза выше, чем в контрольной группе (p=0,001). Дополнительно была проведена оценка концентрации оксида азота в зависимости от пола и возраста. Была выявлена статистически значимая разница между женщинами старше 65 лет и женщинами младшего возраста (p=0,0347). Сравнение концентрации оксида азота у пациентов с частично контролируемой и неконтролируемой БА с контрольной группой показало повышение уровня в 1,31 раза и 1,42 раза соответственно (p=0,0355 и p=0,0033). ROC-анализ уровня оксида азота для диагностики бронхиальной астмы продемонстрировал умеренную диагностическую точность (AUC=0,6932; p=0,013). Таким образом, определение уровня оксида азота в сыворотке крови может иметь важное значение для диагностики бронхиальной астмы и использоваться в качестве потенциального биомаркера заболевания.

Ключевые слова: воспаление, бронхиальная астма, оксид азота, сывороточный биомаркер, уровень оксида азота в сыворотке

Сведения об авторах:

Сабирова Алина Болатовна – магистрант кафедры общей биологии и геномики, Евразийский национальный университет имени Л.Н. Гумилёва, ул. Сатпаева, 2, Астана, Казахстан.

Айтказина Айгерим Тлеубековна – магистр медицины, врач-пульмонолог ГКП на ПХВ «Городская многопрофильная больница №2», Т. Рыскулова, 6, 010000, Астана, Казахстан.

Кусаинова Асия Арысламовна – PhD, старший научный сотрудник Научно-исследовательского института клеточной биологии и биотехнологии, Евразийский национальный университет имени Л.Н. Гумилёва, ул. Сатпаева, 2, Астана, Казахстан.

Булгакова Ольга Владимировна – PhD, профессор кафедры общей биологии и геномики, Евразийский национальный университет имени Л.Н. Гумилёва, ул. Сатпаева, 2, Астана, Казахстан.

Берсимбай Рахметкажи Искендирович – академик НАН РК, директор Научно-исследовательского института клеточной биологии и биотехнологии, Евразийский национальный университет имени Л.Н. Гумилёва, ул. Сатпаева, 2, Астана, Казахстан.

Арипова Акмарал Алтынбаевна – PhD, и.о. доцента кафедры общей биологии и геномики, Евразийский национальный университет имени Л.Н. Гумилёва, ул. Сатпаева, 2, 010000, Астана, Казахстан.

Авторлар туралы мәліметтер:

Сабирова Алина Болатовна – жалпы биология және геномика кафедрасының білім алушысы, Л.Н. Гумилев атындағы Еуразия ұлттық университеті, Сәтбаев көш. 2, Астана, Қазақстан.

Айтказина Айгерим Тлеубековна – медицина магистрі, пульмонолог дәрігер, ШЖҚ «№2 Қалалық көпсалалы ауруханa», Т. Рысқұлов көш. 6, 010000, Астана, Қазақстан.

Кусаинова Асия Арысламовна – PhD, Жасуша биологиясы және биотехнология ғылыми-зерттеу институтының аға ғылыми қызметкері, Л.Н. Гумилев атындағы Еуразия ұлттық университеті, Сәтбаев көш. 2, Астана, Қазақстан.

Булгакова Ольга Владимировна – PhD, жалпы биология және геномика кафедрасының профессоры, Л.Н. Гумилев атындағы Еуразия ұлттық университеті, Сәтбаев көш. 2, Астана, Қазақстан.

Берсімбай Рахметқажы Іскендіруұлы – ҚР ҰҒА академигі, Клеткалық биология және биотехнология ғылыми-зерттеу институтының директоры, Л.Н. Гумилев атындағы Еуразия ұлттық университеті, Сәтбаев көш. 2, Астана, Қазақстан.

Арипова Акмарал Алтынбаевна – PhD, жалпы биология және геномика кафедрасының қауымдастырылған профессор м.а., Л.Н. Гумилев атындағы Еуразия ұлттық университеті, Сәтбаев 2, 010000, Астана, Қазақстан.

Information about the authors:

Sabirova Alina – Learner of the Department of General Biology and Genomics, L.N. Gumilyov Eurasian National University, Satpayev Street 2, Astana, Kazakhstan.

Aitkazina Aigerim – Master of Medicine, pulmonologist, Municipal State Enterprise on the Right of Economic Management "City Multidisciplinary Hospital No. 2", T. Ryskulov Street,6, 010000, Astana, Kazakhstan.

Kussainova Assiya – PhD, senior researcher, Research Institute of Cell Biology and Biotechnology, L.N. Gumilyov Eurasian National University, Satpayev str. 2, Astana, Kazakhstan.

Bulgakova Olga – PhD, Professor, Department of General Biology and Genomics, L.N. Gumilyov Eurasian National University, Satpayev str. 2, Astana, Kazakhstan.

Bersimbaev Rakhmetkazhy – Academician of the National Academy of Sciences of the Republic of Kazakhstan, Director of the Research Institute of Cell Biology and Biotechnology, L.N. Gumilyov Eurasian National University, Satpayev str. 2, Astana, Kazakhstan.

Aripova Akmaral – PhD, Acting associate professor, Department of General Biology and Genomics, L.N. Gumilyov Eurasian National University, 2 Satpayev Street, 010000, Astana, Kazakhstan.