

DOI: <https://doi.org/10.17816/dent626993>



Effect of bronchial asthma on the condition of the oral cavity

Natalia A. Latysh, Svetlana N. Razumova, Nikolai V. Sturov, Angela S. Brago

Peoples' Friendship University of Russia named after Patrice Lumumba, Moscow, Russia

ABSTRACT

BACKGROUND: High-quality dental rehabilitation of patients suffering from bronchial asthma is one of the priority tasks of an integrated approach to the treatment of a general somatic pathology. To date, changes in the oral cavity are polyetiological and can be associated with xerostomia, nutrition, nature of treatment, and poor hygiene. Thus, the microbial factor may play an important role in the development of pathological changes in the oral cavity.

AIM: Through a literature review, this study aimed to examine typical and atypical types of microbial colonies developing in the oral cavity of patients with bronchial asthma and study ways to correct the pathogenic microflora of the oral cavity.

MATERIALS AND METHODS: An analysis was made of information sources in the international databases of Google Scholar, PubMed, and eLIBRARY.RU from 2000 to 2023. The keywords of the query were "oral cavity", "bronchial asthma", "microbes", "antimicrobial therapy".

RESULTS: The literature search extracted 118 sources, of which 34 were relevant. Patients diagnosed with bronchial asthma were found to have dental problems associated with conditions associated with bacterial flora, such as caries, periodontal diseases, and candidiasis. Inflammatory and autoimmune processes were activated by the action of pathogenic microflora.

CONCLUSION: Changes in the microbial balance have affected the development of oral cavity pathologies. Correct hygiene and use of antibacterial and antifungal agents, intake of low-dose drugs, or avoidance of drugs that stimulate the increase in pathogenic microbes, contribute to a decrease in the microbial pathological potential in the oral cavity.

Keywords: oral cavity; bronchial asthma; microbes; antimicrobial therapy.

To cite this article:

Latysh NA, Razumova SN, Sturov NV, Brago AS. Effect of bronchial asthma on the condition of the oral cavity. *Russian Journal of Dentistry*. 2024;28(3):305–315. DOI: <https://doi.org/10.17816/dent626993>

DOI: <https://doi.org/10.17816/dent626993>

К вопросу о влиянии бронхиальной астмы на состояние полости рта

Н.А. Латыш, С.Н. Разумова, Н.В. Стуров, А.С. Браго

Российский университет дружбы народов имени Патриса Лумумбы, Москва, Россия

АННОТАЦИЯ

Актуальность. Качественная стоматологическая реабилитация пациентов, страдающих бронхиальной астмой, — одна из приоритетных задач комплексного подхода к лечению общесоматической патологии. На сегодняшний день считается, что изменения в полости рта имеют полиэтиологичный характер и могут быть связаны с ксеростомией, питанием, характером проводимого лечения, неудовлетворительной гигиеной. Микробный фактор может быть ведущим в развитии патологических изменений в полости рта за счёт изменения качества и количества слюны, являющейся буферной системой, а также других сопутствующих факторов.

Цель исследования — изучить по литературным источникам данные о состоянии полости рта пациентов, страдающих бронхиальной астмой и применяющих ингаляционные препараты для её коррекции.

Материалы и методы. Проведён анализ работ, опубликованных с 2000 по 2023 год в международных базах данных Google Scholar, PubMed, eLIBRARY.RU. Ключевыми словами запроса были “oral cavity”, “bronchial asthma”, “microbes”, “antimicrobial therapy”.

Результаты. Обнаружено 118 источников, из которых 34 были релевантными. На основании полученных литературных данных у пациентов, страдающих бронхиальной астмой, выявлены стоматологические проблемы, связанные с возможным влиянием бактериальной флоры, среди них кариес, болезни пародонта, кандидоз. Вышеуказанные патологии могут развиваться вследствие воспалительного и аутоиммунного процессов, активизируя действие патогенной микрофлоры.

Заключение. Изменение микробного баланса оказывает влияние на развитие патологий полости рта. Коррекция гигиены и использование антибактериальных, противогрибковых средств, а также снижение концентрации или отмена лекарственных средств, стимулирующих увеличение количества патогенных микробов, способствуют снижению микробного патологического потенциала в полости рта.

Ключевые слова: полость рта; бронхиальная астма; микробы; противомикробная терапия.

Как цитировать:

Латыш Н.А., Разумова С.Н., Стуров Н.В., Браго А.С. К вопросу о влиянии бронхиальной астмы на состояние полости рта // Российский стоматологический журнал. 2024. Т. 28, № 3. С. 305–315. DOI: <https://doi.org/10.17816/dent626993>

BACKGROUND

According to the World Health Organization (WHO), there are 262 million patients with asthma worldwide, with more than 250,000 deaths each year [1]. Asthma is a prevalent chronic inflammatory respiratory disease. Its prevalence in children and adults is steadily increasing, as are mortality rates, making asthma a significant cause of disability [2]. The air spaces in the lungs diminish due to inflammation and muscle strain around the small airways, causing cough, wheezing, shortness of breath, and tightness in the chest. These symptoms are intermittent and frequently increase at night or on exertion. Moreover, there are other common triggers that can worsen asthma symptoms. These triggers differ between individuals, but generally include changes in oral microbiota, viral infections (common cold), and weather changes. Other common triggers are dust, smoke, fumes, pollen, animal fur and feathers, various detergents, and perfumes [1]. There are numerous factors that increase the risk of asthma; however, it is sometimes difficult to find the main cause.

Asthma is more likely to affect individuals who have a family history of this condition (in parents or siblings). Moreover, the risk of asthma is higher in patients with allergies, eczema, and rhinitis. Low birth weight, prematurity, exposure to tobacco smoke, air pollution, and viral respiratory infections affect the development of lungs and increase the risk of asthma.

Children and adults with overweight or obesity have a higher risk of asthma. In adults, the risk of asthma increases due to environmental allergens, indoor and outdoor air pollution, house dust, mold, and occupational exposure to chemicals [1, 3, 4].

The treatment of asthma is aimed at eliminating airway inflammation (anti-inflammatory drugs) and smooth muscle spasms (bronchodilators). There are two types of asthma drugs: those for quick relief (rescue medications) and those for long-term disease control. Quick-relief drugs include short-acting bronchodilators, systemic corticosteroids, and anticholinergic agents, whereas long-term control drugs comprise anti-inflammatory agents, long-acting bronchodilators, and leukotriene modifiers [5, 6]. The majority of asthma drugs are administered using various types of inhalers or nebulizers. Patients are taught to use these devices properly and informed about the required frequency and duration of drug therapy.

Microbiota composition varies between individuals and body areas. In asthma, oral microbiota may become the critical factor for upper and lower respiratory tract infections [6–12], considering that the lungs are not sterile, as was previously thought [8]. Inhaled microorganisms and pathogens discharged

from the upper and lower respiratory tract via mucociliary mechanisms and found in saliva promote oropharyngeal infections [13]. The clinical significance of the oropharyngeal wall microbiota is poorly understood. However, antibiotics, probiotics, diet, and intestinal microflora have been shown to affect the oral microbiota composition [14, 15].

Several studies [6, 16, 17] assessed the association between respiratory tract microbiota and chronic respiratory diseases. However, only lower respiratory tract samples, like sputum and bronchoalveolar lavage, were examined. The upper respiratory tract microbiota in patients with asthma is insufficiently studied. The induction or inhibition of systemic immune tolerance to infection-associated antigens can be explained by the association between oral tolerance and airway tolerance. Bacterial infections have been shown to be involved in the development and progression of respiratory diseases; however, there have been no systemic studies of respiratory tract microorganisms [12]. Therefore, it is critical to determine the characteristics of oropharyngeal microbiota and the pathogenicity of its components. This may aid in identifying potential targets and developing new approaches to the prevention and treatment of oral complications of systemic disorders such as asthma [18, 19].

Dental prophylaxis and adequate oral hygiene can help reduce the role of microorganisms in oropharyngeal diseases [20].

The prevalence of asthma is steadily increasing; therefore, problems associated with asthma drugs may cause serious disorders, including in the oral cavity.

This review aimed to assess published data on the oral health of patients with asthma who receive inhaled asthma drugs.

SEARCH METHODOLOGY

The review included works published in Russian and English between 2000 and 2023. The search was performed in Google Scholar, PubMed, and eLIBRARY.RU. The following search terms in Russian and English were used: полость рта / oral cavity, бронхиальная астма / bronchial asthma, микробы/microbes, противомикробная терапия / antimicrobial therapy, бронходилататоры короткого и длительного действия / short- and long-acting bronchodilators, and модификаторы лейкотриенов / leukotriene modifiers. Eligible publications included clinical and laboratory studies and meta-analyses that assessed changes in the oral cavity during asthma treatment. The search yielded 118 publications; of these, 34 were included in the study conducted between January 2023 and December 2023.

RESULTS

The review identified oral diseases in asthma patients with the greatest significance in terms of microorganism involvement. These included xerostomia, disorder of hard tissues of teeth, periodontal diseases, and fungal infections. Eligible publications included 11 articles on the association between asthma and disorder of hard tissues of teeth, 9 articles on the association between asthma and periodontal diseases, 3 articles on the association between asthma and xerostomia, and 11 articles on the association between asthma and fungal infections of the oral mucosa (Table 1).

Samec and Jan [21] and Ersin et al. [22] described the development of dental caries in patients who received asthma drugs that act by decreasing salivary flow rate and modifying saliva pH. The duration and treatment of asthma significantly affect the risk of dental caries. Reddy et al. [23] and Chuang et al. [24] found a high prevalence of dental caries in patients with asthma, which increases with the severity of asthma.

Stensson et al. [25] reported that preschool children with asthma have a higher prevalence and severity of dental caries than children without asthma due to mouth breathing and increased consumption of sugary drinks. Another study by the same authors found that patients with asthma aged 18–24 years have a higher prevalence and severity of dental caries than healthy individuals [26]. Mehtonen et al. [27] and Alavaikko et al. [28] also reported a higher prevalence of dental caries in patients with asthma.

Shashikiran et al. [29] found a high severity of dental caries in patients with asthma who use salbutamol inhalers. Ryberg et al. [30] and Gani et al. [31] reported that a higher risk of dental caries in children with asthma who received beta-2-agonists was most frequently associated with decreased salivary flow rate, xerostomia, and increased *Lactobacilli* and *Streptococcus mutans* counts.

Any factor that reduces the quality and quantity of saliva can compromise oral homeostasis, where saliva

plays a critical role [32]. According to studies, long-term use of beta-2-agonists can reduce salivary flow rates [30, 33]. Patients must be instructed to wash their mouths after using an inhaler. Dry mouth can be prevented by using artificial saliva, drinking more water, or using fluoride mouthwash on a daily basis.

Samec and Jan [21] and Kargul et al. [34] reported a significant decrease in saliva pH to 5.5 in patients with asthma 30 minutes after using beta-2-agonist inhalers, which is below the threshold for enamel demineralization. Kargul et al. [34] found that chewing sugar-free gum for at least one minute after using an inhaler improved dental plaque pH. Moreover, it is recommended to use sugar-free gum to promote saliva production and buffer oral acids. Reduced saliva production may impair the removal of fermentable substances from the oral cavity and decrease its buffering capacity.

Ryberg et al. [30] found that parotid saliva production decreased by 26% and 36%, respectively, in patients with asthma who received drug therapy compared to controls without asthma. In patients with asthma, parotid saliva showed decreased production of total protein, amylase, hexosamine, peroxidase, lysozyme, and IgA per minute [35, 36].

The high incidence of dental caries in patients with asthma can be also associated with fermentable carbohydrates in asthma drugs. Some powder inhalers contain lactose monohydrate to improve the drug's taste. Fathima et al. [37] found that frequent oral inhalations of sugar-containing drugs, along with reduced salivary flow rates, may increase the risk of dental caries. Reddy et al. [23] reported that patients receiving asthma syrups have the highest incidence of dental caries.

Frequent consumption of cariogenic drinks due to excessive thirst may increase the incidence of dental caries in patients with asthma. Increased consumption of these drinks may be associated with several factors, including attempts to drown out the taste of inhaled drug, mitigate the drying effect of mouth breathing,

Table 1. Publications included in the review

Oral diseases in patients with asthma	<i>n</i>	%
Association between asthma and disorder of hard tissues of teeth	11	32.3
Association between asthma and xerostomia	3	8.8
Association between asthma and periodontal diseases	9	26.5
Association between asthma and fungal infections	11	32.3

or reduce salivation caused by beta-2-agonist therapy [25]. Moreover, patients with asthma may overlook oral hygiene due to their condition [23]. Prevention measures such as more frequent dental check-ups, use of fluorides, and dental caries prophylaxis may reduce the risk of dental caries in patients with asthma. According to Gani et al. [31], fluoride supplementation is required in all patients with asthma, especially those receiving beta-2-agonists (Table 2 [38–45]).

The association between asthma and periodontal diseases was assessed in 9 articles (26.5%).

The association between asthma and periodontal diseases may include abnormal immune and inflammatory responses, side effects of asthma drugs, or a combination of these factors. According to Mehta et al. [38], asthma is associated with periodontal diseases. Ferreira et al. [39] reported a high prevalence of periodontal diseases in patients with asthma. Inhaled corticosteroids (ICS) can enter the circulation through the lungs or by swallowing, when the drug does not reach the lungs but is deposited in the posterior pharynx [46, 40]. According to studies, ICS may reduce bone mineral density [40–43, 47]. As early as in 1995, Hanania et al. [47] reported that regular use of standard ICS doses in patients with asthma may inhibit adrenal gland function and reduce bone mineral density in a dose-dependent manner. Systemic bone loss caused by these drugs, especially in long-term use at high doses, may promote periodontal disease progression [48]. Han et al. [49] and Choi et al. [50] reported tooth loss in patients with asthma due to long-term treatment with potent topical ICS, followed by a decrease in bone mineral density, especially in the mandible. Therefore, bone mineral density must be regularly assessed in patients who receive ICS, particularly in the presence of risk factors for osteoporosis. Minimum permissible doses of ICS must be used for asthma control [21, 31, 37, 40]. The association between bacterial and immunological factors is the main cause of periodontal

tissue destruction. Saliva undoubtedly influences this association via its protective mechanisms. Many asthma drugs alter saliva secretion in a substantial proportion of patients, which impairs periodontal health.

The association between asthma and fungal infections was assessed in 11 articles (32.3%). Oropharyngeal candidiasis is typically associated with inhaled corticosteroids [51]. This may be due to the topical effect of these drugs on the oral mucosa, because only 10%–20% of the dose actually reach the lungs, while the majority of the drug remains in the oropharynx. The prevalence of oral candidiasis in ICS therapy may reach 77%, with variability attributable to differences in detection methods. This local side effect is especially common in patients who regularly receive high ICS doses [52]. The general immunosuppressive and anti-inflammatory effects of steroids are thought to play a critical role in candidiasis [53]. Fukushima et al. [44, 45] reported that ICS may decrease IgA levels in saliva, promoting oral candidiasis. Ellepola and Samaranayake [53] also reported that ICS and topical steroids may cause candidiasis. Moreover, many dry-powder inhalers contained 10–25 mg of lactose monohydrate per dose [54, 55]. Elevated glucose levels may also promote the growth, proliferation, and adhesion of *Candida* to oral mucosa cells [55, 56]. Several preventive measures can be taken to minimize the risk of candidiasis during ICS inhalations [53]. Selroos et al. [57] found that mouthwash reduced *Candida* colonization in the oral cavity of patients who used dry powder inhalers. Spacers may reduce the topical effect of steroids that cause oral candidiasis by minimizing drug deposition in the oropharynx and facilitating its passage into the lungs [58]. Salivary flow stimulation in patients with low salivary flow rates may also prevent oral candidiasis. Artificial saliva and sugar-free gum can help increase salivary flow rates. Other methods to reduce *Candida* colonization include antifungal mouthwashes, such as nystatin [59].

Table 2. Impact of asthma drugs on dental problems in patients with asthma

Problem	Manifestation	References
Inflammatory periodontal diseases	Gingivitis and periodontitis secondary to asthma	Mehta et al. [38] Ferreira et al. [39]
Osteoporosis	Reduced bone mineral density secondary to inhaled corticosteroid therapy	Irwin, Richardson [40] Mortimer et al. [41] Heffler et al. [42] Shen et al. [43]
Reduced IgA and IgE levels	Reduced IgA and IgE levels secondary to inhaled corticosteroid therapy may cause candidiasis	Fukushima et al. [44] Fukushima et al. [45]

DISCUSSION

There is disagreement among dental practitioners on the association between asthma and oral diseases, such as dental caries, periodontal diseases, and changes in oral mucosa. The association between asthma and oral diseases, including dental caries, and its primary cause (beta-2-agonist therapy) were studied and confirmed by Almeida et al. [32], Johansson and Ericson [33], Ryberg et al. [30], Shashikiran et al. [29], Stensson et al. [25], and Fathima et al. [37].

McDerra et al. [60] reported a higher prevalence of dental caries affecting permanent teeth in patients with asthma. Kenny and Somaya [61] found that long-term use of liquid oral sugar-containing formulations can increase the incidence of dental caries.

However, Reddy et al. [23], Kargul et al. [34], and Samec and Jan [21] reported that dental caries in patients with asthma is associated with a carbohydrate-rich diet and carbohydrates used as sweeteners in asthma drugs.

In contrast, some other studies found no positive correlation between asthma and dental caries. Bjerkeborn et al. [62] found that asthma or its severity have no effect on the prevalence of dental caries. Eloit et al. [63] also found no correlation between the severity of asthma, treatment duration, and prevalence of dental caries.

However, the association between asthma and periodontal diseases reported by Kenny and Somaya [61], Hyypä et al. [64], Wactawski-Wende et al. [48], and Han et al. [49], was not disputed in subsequent studies.

For example, Hyypä et al. found that gingivitis in patients with asthma may be associated with altered immune response. Patients with asthma have elevated IgE levels in periodontal tissues, which may cause their destruction [64].

Problems associated with candidiasis during ICS therapy, reported by Roland et al. [51], Kurt et al. [52], Ellepola and Samaranayake [53], Selroos et al. [57], and Salzman and Pyszczyński [58], were not disputed. Knight and Fletcher [65] found that patients who received corticosteroids had higher glucose levels in saliva than patients who did not.

CONCLUSION

Controversial data on the association between asthma and oral diseases, such as dental caries, associated with microbial activity in the oral cavity and reduced protective mechanisms due to asthma therapy with agents such as salbutamol, dipropionate, and budesonide, necessitate further research on the pathogenesis of dental caries in patients with asthma. The introduction of novel pathogen identification methods and asthma drugs highlights the importance of oral disease prevention in patients with asthma.

ADDITIONAL INFORMATION

Funding source. The authors state that there is no external financing in this work.

Competing interests. The authors declare that they have no competing interests.

Authors' contribution. All authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work. N.A. Latysh — concept of the article, literature review, writing the article; S.N. Razumova — literature search, preparation and editing of the article; N.V. Sturov — writing the text editing the article; A.S. Brago — article design.

REFERENCES

1. The Global Asthma Report. *Int J Tuberc Lung Dis.* 2022;26 (Suppl. 1):1–104. doi: 10.5588/ijtld.22.1010
2. Chang J, Mosenifar Z. Differentiating COPD from asthma in clinical practice. *J Intensive Care Med.* 2007;22(5):300–309. doi: 10.1177/0885066607304445
3. Chuchalin AG. Pneumonia: the urgent problem of 21st century medicine. *Terapevticheskii arkhiv.* 2016;88(3):4–12. EDN: VOONWP doi: 10.17116/terarkh20168834-12
4. Lötvall J, Akdis CA, Bacharier LB, et al. Asthma endotypes: a new approach to classification of disease entities within the asthma syndrome. *J Allergy Clin Immunol.* 2011;127(2):355–360. doi: 10.1016/j.jaci.2010.11.037
5. Sag C, Ozden FO, Acikgoz G, Anlar FY. The effects of combination treatment with a long-acting beta2-agonist and a corticosteroid on salivary flow rate, secretory immunoglobulin A, and oral health in children and adolescents with moderate asthma: a 1-month, single-blind clinical study. *Clin Ther.* 2007;29(10):2236–2242. doi: 10.1016/j.clinthera.2007.10.014
6. Ramsey CD, Gold DR, Litonjua AA, et al. Respiratory illnesses in early life and asthma and atopy in childhood. *J Allergy Clin Immunol.* 2007;119(1):150–156. doi: 10.1016/j.jaci.2006.09.012
7. Al-Ramahi N, Al-Najjar MAA, Jabaley A, et al. Variations in the respiratory microbiota amongst asthmatic and non-asthmatic subjects in Jordan. *Saudi J Biol Sci.* 2022;29(10):103406. doi: 10.1016/j.sjbs.2022.103406
8. Charlson ES, Bittinger K, Haas AR, et al. Topographical continuity of bacterial populations in the healthy human respiratory tract. *Am J Respir Crit Care Med.* 2011;184(8):957–963. doi: 10.1164/rccm.201104-0655OC
9. Innes JA, Reid PT. Respiratory diseases. In: Boon NA, Colledge NR, Walker BR, Hunter JA, editors. *Davidson's principles*

and practice of medicine. 20th ed. Churchill Livingstone: Elsevier; 2006. P. 670–678.

10. Peng X, Cheng L, You Y, et al. Oral microbiota in human systematic diseases. *Int J Oral Sci.* 2022;14(1):14. doi: 10.1038/s41368-022-00163-7
11. Tuganbaev T, Yoshida K, Honda K. The effects of oral microbiota on health. *Science.* 2022;376(6596):934–936. doi: 10.1126/science.abn1890
12. Belibasakis GN, Bostanci N, Marsh PD, Zaura E. Applications of the oral microbiome in personalized dentistry. *Arch Oral Biol.* 2019;104:7–12. doi: 10.1016/j.archoralbio.2019.05.023
13. Zhu J, Chu W, Luo J, et al. Dental materials for oral microbiota dysbiosis: an update. *Front Cell Infect Microbiol.* 2022;12:900918. doi: 10.3389/fcimb.2022.900918
14. Wade WG, Prosdociimi EM. Profiling of oral bacterial communities. *J Dent Res.* 2020;99(6):621–629. doi: 10.1177/0022034520914594
15. Bourdin A, Gras D, Vachier I, Chanez P. Upper airway x 1: allergic rhinitis and asthma: united disease through epithelial cells. *Thorax.* 2009;64(11):999–1004. doi: 10.1136/thx.2008.112862
16. Erb-Downward JR, Thompson DL, Han MK, et al. Analysis of the lung microbiome in the “healthy” smoker and in COPD. *PLoS One.* 2011;6(2):e16384. doi: 10.1371/journal.pone.0016384
17. Garzoni C, Brugger SD, Qi W, et al. Microbial communities in the respiratory tract of patients with interstitial lung disease. *Thorax.* 2013;68(12):1150–1156. doi: 10.1136/thoraxjnl-2012-202917
18. Noverr MC, Huffnagle GB. The ‘microflora hypothesis’ of allergic diseases. *Clin Exp Allergy.* 2005;35(12):1511–1520. doi: 10.1111/j.1365-2222.2005.02379.x
19. Reddel HK, FitzGerald JM, Bateman ED, et al. GINA 2019: a fundamental change in asthma management: treatment of asthma with short-acting bronchodilators alone is no longer recommended for adults and adolescents. *Eur Respir J.* 2019;53(6):1901046. doi: 10.1183/13993003.01046-2019
20. Song X, Greiner-Tollersrud OK, Zhou H. Oral microbiota variation: a risk factor for development and poor prognosis of esophageal cancer. *Dig Dis Sci.* 2022;67(8):3543–3556. doi: 10.1007/s10620-021-07245-2
21. Samec T, Jan J. Developmental defects of enamel among Slovenian asthmatic children. *Eur J Paediatr Dent.* 2022;23(2):121–124. doi: 10.23804/ejpd.2022.23.02.14
22. Ersin NK, Gülen F, Eronat N, et al. Oral and dental manifestations of young asthmatics related to medication, severity and duration of condition. *Pediatr Int.* 2006;48(6):549–554. doi: 10.1111/j.1442-200X.2006.02281.x
23. Reddy DK, Hegde AM, Munshi AK. Dental caries status of children with bronchial asthma. *J Clin Pediatr Dent.* 2003;27(3):293–295.
24. Chuang CY, Sun HL, Ku MS. Allergic rhinitis, rather than asthma, is a risk factor for dental caries. *Clin Otolaryngol.* 2018;43(1):131–136. doi: 10.1111/coa.12912
25. Stensson M, Wendt LK, Koch G, et al. Oral health in preschool children with asthma. *Int J Paediatr Dent.* 2008;18(4):243–250. doi: 10.1111/j.1365-263X.2008.00921.x
26. Stensson M, Wendt LK, Koch G, et al. Oral health in young adults with long-term, controlled asthma. *Acta Odontol Scand.* 2011;69(3):158–164. doi: 10.3109/00016357.2010.547516
27. Mehtonen IT, Rantala AK, Hugg TT, et al. Dental caries is associated with lower respiratory tract infections: a population-based cohort study. *Respir Med.* 2019;158:1–5. doi: 10.1016/j.rmed.2019.09.002
28. Alavaikko S, Jaakkola MS, Tjäderhane L, Jaakkola JJ. Asthma and caries: a systematic review and meta-analysis. *Am J Epidemiol.* 2011;174(6):631–641. doi: 10.1093/aje/kwr129
29. Shashikiran ND, Reddy VV, Raju PK. Effect of antiasthmatic medication on dental disease: dental caries and periodontal disease. *J Indian Soc Pedod Prev Dent.* 2007;25(2):65–68. doi: 10.4103/0970-4388.33450
30. Ryberg M, Möller C, Ericson T. Effect of beta 2-adrenoceptor agonists on saliva proteins and dental caries in asthmatic children. *J Dent Res.* 1987;66(8):1404–1406. doi: 10.1177/00220345870660082401
31. Gani F, Caminati M, Bellavia F, et al. Oral health in asthmatic patients: a review: asthma and its therapy may impact on oral health. *Clin Mol Allergy.* 2020;18(1):22. doi: 10.1186/s12948-020-00137-2
32. de Almeida Pdel V, Grégio AM, Machado MA, et al. Saliva composition and functions: a comprehensive review. *J Contemp Dent Pract.* 2008;9(3):72–80. doi: 10.5005/jcddp-9-3-72
33. Johansson I, Ericson T. Saliva composition and caries development during protein deficiency and beta-receptor stimulation or inhibition. *J Oral Pathol.* 1987;16(3):145–149. doi: 10.1111/j.1600-0714.1987.tb01482.x
34. Kargul B, Tanboga I, Ergeneli S, et al. Inhaler medicament effects on saliva and plaque pH in asthmatic children. *J Clin Pediatr Dent.* 1998;22(2):137–140.
35. Ryberg M, Möller C, Ericson T. Saliva composition and caries development in asthmatic patients treated with beta 2-adrenoceptor agonists: a 4-year follow-up study. *Scand J Dent Res.* 1991;99(3):212–218. doi: 10.1111/j.1600-0722.1991.tb01887.x
36. Yalçın SS, Emiralioğlu N, Yalçın S. Evaluation of blood and tooth element status in asthma cases: a preliminary case-control study. *BMC Pulm Med.* 2021;21(1):201. doi: 10.1186/s12890-021-01565-9
37. Fathima R, Shenoy R, Jodalli PS, et al. Evaluation of salivary parameters and oral health status among asthmatic and nonasthmatic adult patients visiting a tertiary care hospital. *Cureus.* 2019;11(10):e5957. doi: 10.7759/cureus.5957
38. Mehta A, Sequeira PS, Sahoo RC, Kaur G. Is bronchial asthma a risk factor for gingival diseases? A control study. *N Y State Dent J.* 2009;75(1):44–46.
39. Ferreira MKM, Ferreira RO, Castro MML, et al. Is there an association between asthma and periodontal disease among adults? Systematic review and meta-analysis. *Life Sci.* 2019;223:74–87. doi: 10.1016/j.lfs.2019.03.005
40. Irwin RS, Richardson ND. Side effects with inhaled corticosteroids: the physician’s perception. *Chest.* 2006;130(Suppl. 1):41S–53S. doi: 10.1378/chest.130.1_suppl.41S
41. Mortimer KJ, Harrison TW, Tattersfield AE. Effects of inhaled corticosteroids on bone. *Ann Allergy Asthma Immunol.* 2005;94(1):15–79. doi: 10.1016/S1081-1206(10)61280-X
42. Heffler E, Madeira LNG, Ferrando M, et al. Inhaled corticosteroids safety and adverse effects in patients with

- asthma. *J Allergy Clin Immunol Pract.* 2018;6(3):776–781. doi: 10.1016/j.jaip.2018.01.025
43. Shen TC, Chang PY, Lin CL, et al. Risk of periodontal disease in patients with asthma: a nationwide population-based retrospective cohort study. *J Periodontol.* 2017;88(8):723–730. doi: 10.1902/jop.2017.160414
44. Fukushima C, Matsuse H, Saeki S, et al. Salivary IgA and oral candidiasis in asthmatic patients treated with inhaled corticosteroid. *J Asthma.* 2005;42(7):601–604. doi: 10.1080/02770900500216259
45. Fukushima C, Matsuse H, Tomari S, et al. Oral candidiasis associated with inhaled corticosteroid use: comparison of fluticasone and beclomethasone. *Ann Allergy Asthma Immunol.* 2003;90(6):646–651. doi: 10.1016/S1081-1206(10)61870-4
46. Pandya D, Puttanna A, Balagopal V. Systemic effects of inhaled corticosteroids: an overview. *Open Respir Med J.* 2014;8:59–65. doi: 10.2174/1874306401408010059
47. Hanania NA, Chapman KR, Sturtridge WC, et al. Dose-related decrease in bone density among asthmatic patients treated with inhaled corticosteroids. *J Allergy Clin Immunol.* 1995;96(5 Pt 1):571–579. doi: 10.1016/s0091-6749(95)70254-7
48. Wactawski-Wende J. Periodontal diseases and osteoporosis: association and mechanisms. *Ann Periodontol.* 2001;6(1):197–208. doi: 10.1902/annals.2001.6.1.197
49. Han ER, Choi IS, Kim HK, et al. Inhaled corticosteroid-related tooth problems in asthmatics. *J Asthma.* 2009;46(2):160–164. doi: 10.1080/02770900802553102
50. Choi H, Bae KH, Lee JW. Association between age at asthma diagnosis and tooth loss. *Acta Odontol Scand.* 2018;76(7):466–472. doi: 10.1080/00016357.2018.1436723
51. Roland NJ, Bhalla RK, Earis J. The local side effects of inhaled corticosteroids: current understanding and review of the literature. *Chest.* 2004;126(1):213–219. doi: 10.1378/chest.126.1.213
52. Kurt E, Yildirim H, Kiraz N, et al. Oropharyngeal candidiasis with dry-powdered fluticasone propionate: 500 microg/day versus 200 microg/day. *Allergol Immunopathol (Madr).* 2008;36(1):17–20. doi: 10.1157/13115666
53. Ellepola AN, Samaranayake LP. Inhalational and topical steroids, and oral candidosis: a mini review. *Oral Dis.* 2001;7(4):211–216. doi: 10.1034/j.1601-0825.2001.70402.x
54. Tootla R, Tumba KJ, Duggal MS. An evaluation of the acidogenic potential of asthma inhalers. *Arch Oral Biol.* 2004;49(4):275–283. doi: 10.1016/j.archoralbio.2003.11.006
55. Torres SR, Peixoto CB, Caldas DM, et al. Relationship between salivary flow rates and Candida counts in subjects with xerostomia. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2002;93(2):149–154. doi: 10.1067/moe.2002.119738
56. Samaranayake LP, Hughes A, Weetman DA, MacFarlane TW. Growth and acid production of Candida species in human saliva supplemented with glucose. *J Oral Pathol.* 1986;15(5):251–254. doi: 10.1111/j.1600-0714.1986.tb00617.x
57. Selroos O, Löfroos AB, Pietinalho A, Riska H. Asthma control and steroid doses 5 years after early or delayed introduction of inhaled corticosteroids in asthma: a real-life study. *Respir Med.* 2004;98(3):254–262. doi: 10.1016/j.rmed.2003.10.007
58. Salzman GA, Pyszczynski DR. Oropharyngeal candidiasis in patients treated with beclomethasone dipropionate delivered by metered-dose inhaler alone and with Aerochamber. *J Allergy Clin Immunol.* 1988;81(2):424–428. doi: 10.1016/0091-6749(88)90911-6
59. Lyu X, Zhao C, Yan ZM, Hua H. Efficacy of nystatin for the treatment of oral candidiasis: a systematic review and meta-analysis. *Drug Des Devel Ther.* 2016;10:1161–1171. doi: 10.2147/DDDT.S100795
60. McDerra EJ, Pollard MA, Curzon ME. The dental status of asthmatic British school children. *Pediatr Dent.* 1998;20(4):281–287.
61. Kenny DJ, Somaya P. Sugar load of oral liquid medications on chronically ill children. *J Can Dent Assoc.* 1989;55(1):43–46.
62. Bjerkeborn K, Dahllöf G, Hedlin G, et al. Effect of disease severity and pharmacotherapy of asthma on oral health in asthmatic children. *Scand J Dent Res.* 1987;95(2):159–164. doi: 10.1111/j.1600-0722.1987.tb01824.x
63. Eloit AK, Vanobbergen JN, De Baets F, Martens LC. Oral health and habits in children with asthma related to severity and duration of condition. *Eur J Paediatr Dent.* 2004;5(4):210–215.
64. Hyypä TM, Koivikko A, Paunio KU. Studies on periodontal conditions in asthmatic children. *Acta Odontol Scand.* 1979;37(1):15–20. doi: 10.3109/00016357909004680
65. Knight L, Fletcher J. Growth of Candida albicans in saliva: stimulation by glucose associated with antibiotics, corticosteroids, and diabetes mellitus. *J Infect Dis.* 1971;123(4):371–377. doi: 10.1093/infdis/123.4.371

СПИСОК ЛИТЕРАТУРЫ

1. The Global Asthma Report // *Int J Tuberc Lung Dis.* 2022. Vol. 26(Suppl. 1). P. 1–104. doi: 10.5588/ijtld.22.1010
2. Chang J., Mosenifar Z. Differentiating COPD from asthma in clinical practice // *J Intensive Care Med.* 2007. Vol. 22, N 5. P. 300–309. doi: 10.1177/0885066607304445
3. Чучалин А.Г. Пневмония: актуальная проблема медицины XXI века // *Терапевтический архив.* 2016. Т. 88, № 3. С. 4–12. EDN: VOONWP doi: 10.17116/terarkh20168834-12
4. Lötvall J., Akdis C.A., Bacharier L.B., et al. Asthma endotypes: a new approach to classification of disease entities within the asthma syndrome // *J Allergy Clin Immunol.* 2011. Vol. 127, N 2. P. 355–360. doi: 10.1016/j.jaci.2010.11.037
5. Sag C., Ozden F.O., Acikgoz G., Anlar F.Y. The effects of combination treatment with a long-acting beta2-agonist and a corticosteroid on salivary flow rate, secretory immunoglobulin A, and oral health in children and adolescents with moderate asthma: a 1-month, single-blind clinical study // *Clin Ther.* 2007. Vol. 29, N 10. P. 2236–2242. doi: 10.1016/j.clinthera.2007.10.014
6. Ramsey C.D., Gold D.R., Litonjua A.A., et al. Respiratory illnesses in early life and asthma and atopy in childhood // *J Allergy Clin Immunol.* 2007. Vol. 119, N 1. P. 150–156. doi: 10.1016/j.jaci.2006.09.012
7. Al-Ramahi N., Al-Najjar M.A.A., Jabaley A., et al. Variations in the respiratory microbiota amongst asthmatic and non-asthmatic

- subjects in Jordan // *Saudi J Biol Sci.* 2022. Vol. 29, N 10. P. 103406. doi: 10.1016/j.sjbs.2022.103406
8. Charlson E.S., Bittinger K., Haas A.R., et al. Topographical continuity of bacterial populations in the healthy human respiratory tract // *Am J Respir Crit Care Med.* 2011. Vol. 184, N 8. P. 957–963. doi: 10.1164/rccm.201104-0655OC
9. Innes J.A., Reid P.T. Respiratory diseases. In: Boon N.A., Colledge N.R., Walker B.R., Hunter J.A., editors. *Davidson's principles and practice of medicine.* 20th ed. Churchill Livingstone: Elsevier, 2006. P. 670–678.
10. Peng X., Cheng L., You Y., et al. Oral microbiota in human systematic diseases // *Int J Oral Sci.* 2022. Vol. 14, N 1. P. 14. doi: 10.1038/s41368-022-00163-7
11. Tuganbaev, T., Yoshida K., Honda K. The effects of oral microbiota on health // *Science.* 2022. Vol. 376, N 6596. P. 934–936. doi: 10.1126/science.abn1890
12. Belibasakis G.N., Bostanci N., Marsh P.D., Zaura E. Applications of the oral microbiome in personalized dentistry // *Arch Oral Biol.* 2019. Vol. 104. P. 7–12. doi: 10.1016/j.archoralbio.2019.05.023
13. Zhu J., Chu W., Luo J., et al. Dental materials for oral microbiota dysbiosis: an update // *Front Cell Infect Microbiol.* 2022. Vol. 12. P. 900918. doi: 10.3389/fcimb.2022.900918
14. Wade W.G., Prosdocimi E.M. Profiling of oral bacterial communities // *J Dent Res.* 2020. Vol. 99, N 6. P. 621–629. doi: 10.1177/0022034520914594
15. Bourdin A., Gras D., Vachier I., Chanez P. Upper airway x 1: allergic rhinitis and asthma: united disease through epithelial cells // *Thorax.* 2009. Vol. 64, N 11. P. 999–1004. doi: 10.1136/thx.2008.112862
16. Erb-Downward J.R., Thompson D.L., Han M.K., et al. Analysis of the lung microbiome in the “healthy” smoker and in COPD // *PLoS One.* 2011. Vol. 6, N 2. P. e16384. doi: 10.1371/journal.pone.0016384
17. Garzoni C., Brugger S.D., Qi W., et al. Microbial communities in the respiratory tract of patients with interstitial lung disease // *Thorax.* 2013. Vol. 68, N 12. P. 1150–1156. doi: 10.1136/thoraxjnl-2012-202917
18. Noverr M.C., Huffnagle G.B. The ‘microflora hypothesis’ of allergic diseases // *Clin Exp Allergy.* 2005. Vol. 35, N 12. P. 1511–1520. doi: 10.1111/j.1365-2222.2005.02379.x
19. Reddel H.K., FitzGerald J.M., Bateman E.D., et al. GINA 2019: a fundamental change in asthma management: treatment of asthma with short-acting bronchodilators alone is no longer recommended for adults and adolescents // *Eur Respir J.* 2019. Vol. 53, N 6. P. 1901046. doi: 10.1183/13993003.01046-2019
20. Song X., Greiner-Tollersrud O.K., Zhou H. Oral microbiota variation: a risk factor for development and poor prognosis of esophageal cancer // *Dig Dis Sci.* 2022. Vol. 67, N 8. P. 3543–3556. doi: 10.1007/s10620-021-07245-2
21. Samec T., Jan J. Developmental defects of enamel among Slovenian asthmatic children // *Eur J Paediatr Dent.* 2022. Vol. 23, N 2. P. 121–124. doi: 10.23804/ejpd.2022.23.02.14
22. Ersin N.K., Gülen F., Eronat N., et al. Oral and dental manifestations of young asthmatics related to medication, severity and duration of condition // *Pediatr Int.* 2006. Vol. 48, N 6. P. 549–554. doi: 10.1111/j.1442-200X.2006.02281.x
23. Reddy D.K., Hegde A.M., Munshi A.K. Dental caries status of children with bronchial asthma // *J Clin Pediatr Dent.* 2003. Vol. 27, N 3. P. 293–295.
24. Chuang C.Y., Sun H.L., Ku M.S. Allergic rhinitis, rather than asthma, is a risk factor for dental caries // *Clin Otolaryngol.* 2018. Vol. 43, N 1. P. 131–136. doi: 10.1111/coa.12912
25. Stensson M., Wendt L.K., Koch G., et al. Oral health in preschool children with asthma // *Int J Paediatr Dent.* 2008. Vol. 18, N 4. P. 243–250. doi: 10.1111/j.1365-263X.2008.00921.x
26. Stensson M., Wendt L.K., Koch G., et al. Oral health in young adults with long-term, controlled asthma // *Acta Odontol Scand.* 2011. Vol. 69, N 3. P. 158–164. doi: 10.3109/00016357.2010.547516
27. Mehtonen I.T., Rantala A.K., Hugg T.T., et al. Dental caries is associated with lower respiratory tract infections: a population-based cohort study // *Respir Med.* 2019. Vol. 158. P. 1–5. doi: 10.1016/j.rmed.2019.09.002
28. Alavaikko S., Jaakkola M.S., Tjäderhane L., Jaakkola J.J. Asthma and caries: a systematic review and meta-analysis // *Am J Epidemiol.* 2011. Vol. 174, N 6. P. 631–641. doi: 10.1093/aje/kwr129
29. Shashikiran N.D., Reddy V.V., Raju P.K. Effect of antiasthmatic medication on dental disease: dental caries and periodontal disease // *J Indian Soc Pedod Prev Dent.* 2007. Vol. 25, N 2. P. 65–68. doi: 10.4103/0970-4388.33450
30. Ryberg M., Möller C., Ericson T. Effect of beta 2-adrenoceptor agonists on saliva proteins and dental caries in asthmatic children // *J Dent Res.* 1987. Vol. 66, N 8. P. 1404–1406. doi: 10.1177/00220345870660082401
31. Gani F., Caminati M., Bellavia F., et al. Oral health in asthmatic patients: a review: asthma and its therapy may impact on oral health // *Clin Mol Allergy.* 2020. Vol. 18, N 1. P. 22. doi: 10.1186/s12948-020-00137-2
32. de Almeida Pdel V., Grégio A.M., Machado M.A., et al. Saliva composition and functions: a comprehensive review // *J Contemp Dent Pract.* 2008. Vol. 9, N 3. P. 72–80. doi: 10.5005/jcdp-9-3-72
33. Johansson I., Ericson T. Saliva composition and caries development during protein deficiency and beta-receptor stimulation or inhibition // *J Oral Pathol.* 1987. Vol. 16, N 3. P. 145–149. doi: 10.1111/j.1600-0714.1987.tb01482.x
34. Kargul B., Tanboga I., Ergeneli S., et al. Inhaler medicament effects on saliva and plaque pH in asthmatic children // *J Clin Pediatr Dent.* 1998. Vol. 22, N 2. P. 137–140.
35. Ryberg M., Möller C., Ericson T. Saliva composition and caries development in asthmatic patients treated with beta 2-adrenoceptor agonists: a 4-year follow-up study // *Scand J Dent Res.* 1991. Vol. 99, N 3. P. 212–218. doi: 10.1111/j.1600-0722.1991.tb01887.x
36. Yalçın S.S., Emiralioğlu N., Yalçın S. Evaluation of blood and tooth element status in asthma cases: a preliminary case-control study // *BMC Pulm Med.* 2021. Vol. 21, N 1. P. 201. doi: 10.1186/s12890-021-01565-9
37. Fathima R., Shenoy R., Jodalli P.S., et al. Evaluation of salivary parameters and oral health status among asthmatic and nonasthmatic adult patients visiting a tertiary care hospital // *Cureus.* 2019. Vol. 11, N 10. P. e5957. doi: 10.7759/cureus.5957
38. Mehta A., Sequeira P.S., Sahoo R.C., Kaur G. Is bronchial asthma a risk factor for gingival diseases? A control study // *NY State Dent J.* 2009. Vol. 75, N 1. P. 44–46.

39. Ferreira M.K.M., Ferreira R.O., Castro M.M.L., et al. Is there an association between asthma and periodontal disease among adults? Systematic review and meta-analysis // *Life Sci.* 2019. Vol. 223. P. 74–87. doi: 10.1016/j.lfs.2019.03.005
40. Irwin R.S., Richardson N.D. Side effects with inhaled corticosteroids: the physician's perception // *Chest.* 2006. Vol. 130(Suppl. 1). P. 41S–53S. doi: 10.1378/chest.130.1_suppl.41S
41. Mortimer K.J., Harrison T.W., Tattersfield A.E. Effects of inhaled corticosteroids on bone // *Ann Allergy Asthma Immunol.* 2005. Vol. 94, N 1. P. 15–79. doi: 10.1016/S1081-1206(10)61280-X
42. Heffler E., Madeira L.N.G., Ferrando M., et al. Inhaled corticosteroids safety and adverse effects in patients with asthma // *J Allergy Clin Immunol Pract.* 2018. Vol. 6, N 3. P. 776–781. doi: 10.1016/j.jaip.2018.01.025
43. Shen T.C., Chang P.Y., Lin C.L., et al. Risk of periodontal disease in patients with asthma: a nationwide population-based retrospective cohort study // *J Periodontol.* 2017. Vol. 88, N 8. P. 723–730. doi: 10.1902/jop.2017.160414
44. Fukushima C., Matsuse H., Saeki S., et al. Salivary IgA and oral candidiasis in asthmatic patients treated with inhaled corticosteroid // *J Asthma.* 2005. Vol. 42, N 7. P. 601–604. doi: 10.1080/02770900500216259
45. Fukushima C., Matsuse H., Tomari S., et al. Oral candidiasis associated with inhaled corticosteroid use: comparison of fluticasone and beclomethasone // *Ann Allergy Asthma Immunol.* 2003. Vol. 90, N 6. P. 646–651. doi: 10.1016/S1081-1206(10)61870-4
46. Pandya D., Puttanna A., Balagopal V. Systemic effects of inhaled corticosteroids: an overview // *Open Respir Med J.* 2014. Vol. 8. P. 59–65. doi: 10.2174/1874306401408010059
47. Hanania N.A., Chapman K.R., Sturtridge W.C., et al. Dose-related decrease in bone density among asthmatic patients treated with inhaled corticosteroids // *J Allergy Clin Immunol.* 1995. Vol. 96(5 Pt 1). P. 571–579. doi: 10.1016/s0091-6749(95)70254-7
48. Wactawski-Wende J. Periodontal diseases and osteoporosis: association and mechanisms // *Ann Periodontol.* 2001. Vol. 6, N 1. P. 197–208. doi: 10.1902/annals.2001.6.1.197
49. Han E.R., Choi I.S., Kim H.K., et al. Inhaled corticosteroid-related tooth problems in asthmatics // *J Asthma.* 2009. Vol. 46, N 2. P. 160–164. doi: 10.1080/02770900802553102
50. Choi H., Bae K.H., Lee J.W. Association between age at asthma diagnosis and tooth loss // *Acta Odontol Scand.* 2018. Vol. 76, N 7. P. 466–472. doi: 10.1080/00016357.2018.1436723
51. Roland N.J., Bhalla R.K., Earis J. The local side effects of inhaled corticosteroids: current understanding and review of the literature // *Chest.* 2004. Vol. 126, N 1. P. 213–219. doi: 10.1378/chest.126.1.213
52. Kurt E., Yildirim H., Kiraz N., et al. Oropharyngeal candidiasis with dry-powdered fluticasone propionate: 500 microg/day versus 200 microg/day // *Allergol Immunopathol (Madr).* 2008. Vol. 36, N 1. P. 17–20. doi: 10.1157/13115666
53. Ellepola A.N., Samaranayake L.P. Inhalational and topical steroids, and oral candidosis: a mini review // *Oral Dis.* 2001. Vol. 7, N 4. P. 211–216. doi: 10.1034/j.1601-0825.2001.70402.x
54. Tootla R., Toumba K.J., Duggal M.S. An evaluation of the acidogenic potential of asthma inhalers // *Arch Oral Biol.* 2004. Vol. 49, N 4. P. 275–283. doi: 10.1016/j.archoralbio.2003.11.006
55. Torres S.R., Peixoto C.B., Caldas D.M., et al. Relationship between salivary flow rates and Candida counts in subjects with xerostomia // *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2002. Vol. 93, N 2. P. 149–154. doi: 10.1067/moe.2002.119738
56. Samaranayake L.P., Hughes A., Weetman D.A., MacFarlane T.W. Growth and acid production of Candida species in human saliva supplemented with glucose // *J Oral Pathol.* 1986. Vol. 15, N 5. P. 251–254. doi: 10.1111/j.1600-0714.1986.tb00617.x
57. Selroos O., Löfroos A.B., Pietinalho A., Riska H. Asthma control and steroid doses 5 years after early or delayed introduction of inhaled corticosteroids in asthma: a real-life study // *Respir Med.* 2004. Vol. 98, N 3. P. 254–262. doi: 10.1016/j.rmed.2003.10.007
58. Salzman G.A., Pyszczyński D.R. Oropharyngeal candidiasis in patients treated with beclomethasone dipropionate delivered by metered-dose inhaler alone and with Aerochamber // *J Allergy Clin Immunol.* 1988. Vol. 81, N 2. P. 424–428. doi: 10.1016/0091-6749(88)90911-6
59. Lyu X., Zhao C., Yan Z.M., Hua H. Efficacy of nystatin for the treatment of oral candidiasis: a systematic review and meta-analysis // *Drug Des Devel Ther.* 2016. Vol. 10. P. 1161–1171. doi: 10.2147/DDDT.S100795
60. McDerra E.J., Pollard M.A., Curzon M.E. The dental status of asthmatic British school children // *Pediatr Dent.* 1998. Vol. 20, N 4. P. 281–287.
61. Kenny D.J., Somaya P. Sugar load of oral liquid medications on chronically ill children // *J Can Dent Assoc.* 1989. Vol. 55, N 1. P. 43–46.
62. Bjerkeborn K., Dahlöf G., Hedlin G., et al. Effect of disease severity and pharmacotherapy of asthma on oral health in asthmatic children // *Scand J Dent Res.* 1987. Vol. 95, N 2. P. 159–164. doi: 10.1111/j.1600-0722.1987.tb01824.x
63. Eloit A.K., Vanobbergen J.N., De Baets F., Martens L.C. Oral health and habits in children with asthma related to severity and duration of condition // *Eur J Paediatr Dent.* 2004. Vol. 5, N 4. P. 210–215.
64. Hyypä T.M., Koivikko A., Paunio K.U. Studies on periodontal conditions in asthmatic children // *Acta Odontol Scand.* 1979. Vol. 37, N 1. P. 15–20. doi: 10.3109/00016357909004680
65. Knight L., Fletcher J. Growth of Candida albicans in saliva: stimulation by glucose associated with antibiotics, corticosteroids, and diabetes mellitus // *J Infect Dis.* 1971. Vol. 123, N 4. P. 371–377. doi: 10.1093/infdis/123.4.371

AUTHORS' INFO

* **Angela S. Brago**, MD, Cand. Sci. (Medicine), Associate Professor;
address: 6 Mikluho-Maklaja street, 117198 Moscow, Russia;
ORCID: 0000-0001-8947-4357;
eLibrary SPIN: 2437-8867;
e-mail: anzhela_bogdan@mail.ru

Svetlana N. Razumova, MD, Dr. Sci. (Medicine), Professor;
ORCID: 0000-0002-9533-9204;
eLibrary SPIN: 6771-8507;
e-mail: razumova_sv@mail.ru

Nikolai V. Sturov, MD, Cand. Sci. (Medicine), Associate Professor;
ORCID: 0000-0002-3138-8410;
eLibrary SPIN: 2805-9823;
e-mail: sturov-nv@rudn.ru

Natalia A. Latysh;
ORCID: 0009-0001-0631-9819;
e-mail: dr.latysh1978@mail.ru

ОБ АВТОРАХ

* **Браго Анжела Станиславовна**, канд. мед. наук, доцент;
адрес: Россия, 117198, Москва, ул. Миклухо-Маклая, д. 6;
ORCID: 0000-0001-8947-4357;
eLibrary SPIN: 2437-8867;
e-mail: anzhela_bogdan@mail.ru

Разумова Светлана Николаевна, д-р мед. наук, профессор;
ORCID: 0000-0002-9533-9204;
eLibrary SPIN: 6771-8507;
e-mail: razumova_sv@mail.ru

Стуров Николай Владимирович, канд. мед. наук, доцент;
ORCID: 0000-0002-3138-8410;
eLibrary SPIN: 2805-9823;
e-mail: sturov-nv@rudn.ru

Латыш Наталья Алексеевна;
ORCID: 0009-0001-0631-9819;
e-mail: dr.latysh1978@mail.ru

* Corresponding author / Автор, ответственный за переписку