УДК 616.211-002:616.98:578.834.11-06
DOI 10.24144/1998-6475.2026.71.52-57

FEATURES OF DYSOSMIA IN NASOPHARYNGITIS OF CORONAVIRUS (SARS-CoV-2) GENESIS

Haman Yu.V. *(ORCID ID: https://orcid.org/0000-0002-0475-3312)*
SHEI «Uzhhorod National University», Department of Surgical diseases, Medical Faculty, Uzhhorod

Abstract. *Introduction* The upper respiratory tract is affected in the early stage of COVID-19. Moreover, it has been studied that the cellular tropism of SARS-CoV-2 is limited to the nose ciliated epithelium and the oral cavity epithelium squamous. Initially, olfactory disorders, such as anosmia (complete loss of smell) or hyposmia (partial loss of smell), were not considered as specific symptoms.

The aim was to investigate and analyze information sources of thematic direction, which included pathological patterns of viral etiology and reactions of the upper respiratory tract mucosa in coronavirus infection.

Materials and methods. A systematic literature review was analised, including publications by the past ten years in the databases PubMed, Scopus and Web of Science, Cochrane Library, Embase, Clinical Trials.gov., UpToDate/DynaMed, National Library named by Vernadsky, professional Ukrainian journals (according to the requirements of the Supreme Attestation Commission, Ministry of Education and Science of Ukraine). The selection of articles was made according to keywords.

The results of the studies showed that the olfactory nasal cavity epithelium is an area of enhanced binding, new coronavirus replication and accumulation, which is carried out by the active expression of two "host" receptors (ACE2 receptor blockers and *TMPS2* inhibitors) and by numerously olfactory epithelium non-neuronal cells. The exact mechanism by which the virus leads to this symptom is still unclear. The proposed mechanism is that in "immunely balanced" individuals. The coronavirus damages the olfactory epithelium and causes acute local inflammation with edema and development of anosmia are which recovered after the acute phase. However, in individuals with a genetic predisposition to develop autoimmunity. The immune response to the any virus can create a parallel adequate autoimmune response, generating autoantibodies directed to olfactory receptors and related peptides (G-protein-coupled receptors), allowing the inflammatory process to access the central nervous system by the olfactory bulb. The inflammation is induced with the neurological symptom sprogress, including persistent anosmia, fatigue, brain fog, etc. The definitive clinical and pathophysiological of olfactory symptoms significance remains to be elucidated.

Conclusions. COVID-19 olfactory disorder in patients is an early symptom of the disease, usually severe with complete loss of olfactory function, but reversible in most cases. The clinical picture represents a specific lesion of the respiratory system, uncharacteristic for other respiratory viruses, with preservation of conditions for normal transnasal airflow in the presence or slight severity of edema, hyperemia of the nasal mucosa and/or rhinorrhea.

Key words: upper respiratory tract, nasopharyngeal mucosa, nasopharyngitis, dysosmia, SARS-CoV-2.

Особливості дизосмій при назофарингіті коронавірусного (SARS-CoV-2) **генезу**
Гаман Ю.В.

Резюме. *Вступ* Верхні дихальні шляхи уражаються на ранній стадії COVID-19. До того ж досліджено, що клітинний тропізм SARS-CoV-2 обмежений війчастим епітелієм носа та плоскоклітинним епітелієм ротової порожнини. Спочатку порушення нюху, такі як аносмія (повна втрата нюху) або гіпосмія (часткова втрата нюху), не вважалися специфічними симптомами,

проте деякі дослідження вказували на можливий зв'язок між досліджуваними розладами та COVID-19.

Мета дослідження. Дослідити та проаналізувати інформаційні джерела тематичного спрямування, які включали патологічні патерни вірусної етіології та реакції слизової оболонки верхніх відділів респіраторного тракту при коронавірусній інфекції

Матеріали та методи. Було проведено систематичний аналіз літератури, включаючи публікації за останні десять років у базах даних PubMed, Scopus та Web of Science, Cochrane Library, Embase, Clinical Trials. gov., UpToDate/DynaMed, Національної бібліотеки ім. Вернадського, у фахових українських журналах (за вимогами ВАК, МОН). Відбір статей здійснювався за ключовими словами.

Результати досліджень. Виявлено, що нюховий епітелій порожнини носа – ділянка посиленого зв'язування, реплікації та накопичення нового коронавірусу, що обумовлено активною експресією двох рецепторів «господаря» (протеаз АПФ2 і ТМПС2), численними нейрональними клітинами нюхового епітелію. Точний механізм, через який вірус призводить до цього симптому, досі незрозумілий. Запропо-

нований механізм полягає в тому, що у «імунітетно збалансованих» осіб коронавірус пошкоджує нюховий епітелій та викликає гостре місцеве запалення з набряком та розвитком аносмії, яке відновлюється після гострої фази. Однак у осіб із генетичною схильністю до розвитку аутоімунітету імунна відповідь на вірус може створити паралельну аутоімунну відповідь, створюючи аутоантитіла, спрямовані на нюхові рецептори та подібні пептиди (рецептори, пов'язані з G-білком), що дозволяє запальному процесу в центральній нервовій системі через нюхову цибулину, де індукується запалення з розвитком неврологічних симптомів, включаючи стійку аносмію, втому, туман у мозку тощо. Остаточну клінічну та патофізіологічну важливість нюхових симптомів ще належить з'ясувати [32].

Висновки. Розлад нюху у пацієнтів із COVID-19 є раннім симптомом захворювання, зазвичай важкого ступеня з повною втратою функції відчуття запахів, але здебільшого зворотний. Клінічна картина представляє особливе, нехарактерне для інших респіраторних вірусів ураження респіраторної системи із збереженням умов для нормального трансназального потоку повітря при присутності або незначної виразності набряку, гіперемії слизової носа та/або ринореї.

Ключові слова: верхні дихальні шляхи, слизова оболонка назофаренгіальної зони, назофарингіт, дизосмії, SARS-CoV-2.

Introduction

The upper respiratory tract is affected early in COVID-19. Moreover, it has been studied that the cellular tropism of SARS-CoV-2 is limited to the nose ciliated epithelium and the oral cavity squamous epithelium [1]. Initially, olfactory disorders, such as anosmia (complete loss of smell) or hyposmia (partial loss of smell), were not considered specific symptoms, however, some studies have indicated a possible link between the disorders under study and COVID-19. After the spread of the virus in Europe, it became apparent that anosmia and hyposmia are important criteria for diagnosing the disease. Therefore, the WHO recognized olfactory disorders as one of the main symptoms of COVID-19. At the same time, chemosensory insufficiency is most often an early, and sometimes the only manifestation of infection in carriers of the SARS-CoV-2 virus, who do not have other symptoms [2,3]. There has been increasing interest in the possible causes of early and specific chemosensory deviation in COVID-2019. We reviewed recent studies that have shown that the prevalence of symptoms such as olfactory and gustatory dysfunction in patients with COVID-19 is not the same in different populations. This is likely due to differences in the S-protein of several viral strains, or interpopulation differences in human proteins. These used by the virus for cell entry, which alters the infectious properties of the virus, respectively, the cellular and molecular mechanisms underlying virus-induced anosmia, especially highlighting new evidence of the key role of olfactory epithelium supporting cells of the [4].

The obtained data indicates brain damage in COVID-19, which interpreted the possibility of infection and pathways of SARS-CoV-2 to penetrated into the brain through the nasal cavity olfactory epithelium. The prospects of using symptoms of chemosensory dysfunction for rapid screening diagnostics of COVID-19 in the early stages of the disease were also analyzed [5]. Anosmia and hyposmia significantly affect the patients quality of life and have many etiological factors, including trauma, inflammatory conditions, in particular, chronic rhinosinusitis, neoplasms and viral infections (rhinovirus and SARS-CoV-2 too) [6].

Olfactory disorders are one of the main manifestations of COVID-19, but studies are still needed to clarify the mechanism associated with the development of anosmia and hyposmia caused by SARS-CoV-2 [3]. The main route of entry of SARS-CoV-2 into the body is the nasal mucosa. Conductive anosmia occurs due to nasal congestion, which is common with many viruses, and may be accompanied by symptoms of rhinorrhea and rhinitis. However, studies show that loss of smell in COVID-19 occurs in most cases independently of these symptoms[7]. Olfactory epithelial damage is the mechanism identified as the most likely cause of olfactory dysfunction caused by SARS-CoV-2, which may be exacerbated by central nervous system (CNS) damage [8,9]. The COVID-19 Access Accelerator has become an unexpected global effort to rapid development and expand diagnostics, treatments, and ensure equitable global access to COVID-19 vaccines, initiated by WHO and partners [10].

Since the WHO Director-General's declaration of a public health emergency of international concern in May 2023 and the emergency final phase. WHO has continued to lead the global response to COVID-19, semi-working with partners and governments, to transition by long-term, integrated management of COVID-19 and other coronavirus dangers. Current activities in this leading have included regular reporting, supporting ongoing research, updating recommendations and attitudes,

virus trends monitoring and vaccine formulations improving according to needing [2].

WHO has developed a strategical and operational plan about the help many countries address in COVID-19 as part of their current health systems, to conduct this transition. The plan assists the sustainable, evidence-based management of the significants coronavirus dangers, aligning COVID-19 responses with broader strategies for Respiratory tract disease control and public health stability. Strains of Omicron virus have a shorter incubation period than pre-Omicron virus strains, which now constitute the vast majority of virus strains circulating in humans [11,12].

The aim was to investigate and analyze information sources of thematic direction, which included pathological patterns of viral etiology and reactions of the upper respiratory tract mucosa in coronavirus infection.

Materials and methods

A systematic literature review was analised, including publications by the past ten years in the databases PubMed, Scopus and Web of Science, Cochrane Library, Embase, Clinical Trials.gov., UpToDate/DynaMed, National Library named by Vernadsky, professional Ukrainian journals (according to the requirements of the Supreme Attestation Commission, Ministry of Education and Science of Ukraine). The selection of articles was made according to keywords.

Results

The researchers sequenced the RNA in each cell, one cell at a time. (To put all the work into perspective, each patient's swab yielded an average of 562 cells.) for the getting a detailed picture of what's happening in the nasopharynx during coronavirus infection. The RNA data allowed the team to pinpoint which cells were present, which contained virus-derived RNA—a hallmark of infection—and which genes the cells were turning on and off in response [13,14].

It soon became clear that the nose and throat epithelial cells covering undergo significant changes in the presence of coronavirus infection. The cells generally became more diverse in type. There was an increasing in the level of secretory and goblet cells, which produced by mucus. At the same time, there was a striking loss of mature ciliated cells that line the airways, along with immature ciliated cells increasing in (which may have been trying to compensate) [15].

The researchers found SARS-CoV-2 RNA in a variety of cell types, including immature ciliated cells and secretory cells specific subtypes, squamous cells, and goblet cells. Infected cells, compared with uninfected "witness" cells, had more activated genes involved in the productive response to infection. The mucous membrane is a component of the nasal airways. In particular, the COVID-19 pandemic has demonstrated that the olfactory mucosa is an integral part of a heterogeneous barrier critical for upper respiratory tract immunity. However, insufficient knowledge about olfactory mucosal immunity hinders attempts to protect this tissue from infections and other diseases [16]. Upper respiratory tract infection caused by SARS-CoV-2 can lead to smell and taste loss, in prolonged COVID [20] and potential systemic spread of the virus [17]. Perhaps most importantly, this not only poses a risk to immunocompromised and unvaccinated individuals, but also creates an opportunity for the virus to evolve to evade the immune response. Experimental data indicate that nasal tissues are less protected from SARS-CoV-2 reinfection than the lungs [18]. Indeed, in models of airborne infectious disease, the nasal mucosa is typically unprotected even in the presence of systemic immunity [19]. Protection of the upper respiratory tract from infection should be a key correlate of protective responses, both against SARS-CoV-2 and against other respiratory infections. The nasal airway is a point of entry for many pathogens, and the establishment of protective immunity in this tissue is an important way to break the chain of infection transmission [20,21].

Many approaches have been proposed to establish local protective immunity, the effectiveness of which is currently limited by two considerations: what immune parameters are required to protect the nasal passages(?) and how can this tissue-specific immunity be generated? [22]. Among the complicating factors is that the nasal mucosa contains at least two different types of tissues that require protection, namely the olfactory mucosa (or olfactory epithelium) and the airway mucosa, each of which has unique immune features [23].

It is generally accepted that a tissue-specific mucosal immune response is required to protect the upper airways more than the lower airways [24].

Numerous hypotheses have been proposed as to what constitutes this mucosal response:

secretory IgA antibodies, resident T cells and mucosal cytokines are often mentioned [4]. Olfactory epithelial damage may also be exacerbated by an inflammatory response leading to cell death known as pyroptosis. The immune system is activated upon recognition of a pathogen, which causes to an increase in the secretion of proinflammatory cytokines and chemokines: interleukin (IL)-6, interferon gamma (IFN-γ), chemoattractant proteins from monocyte chemoattractant protein-1, and interferon-induced protein 10 (IP-10). These cytokines indicate a response more focused on the recruitment of monocytes and T lymphocytes. Chemosensory deficits are often the first, and sometimes the only, signs in asymptomatic carriers of SARS-CoV-2 [25].

The studies have demonstrated a possible correlation between anosmia and IL-6 levels in addition. IL-6 induces the expression of several acute phase proteins, including C-reactive protein, serum amyloid A, α1-antiquimotrypsin, haptoglobin, fibrinogen, and complement components. Therefore, patients with higher IL-6 levels have more severe olfactory disorders. High cytokine production may to triggered the death of olfactory neurons. Replacement of olfactory epithelial neurons by basal stem cells requires a longer recovery time, which explains cases of persistent anosmia [26]. Secreted immunoglobulins play a critical role in protecting the mucosal surfaces of the respiratory tract [27]. Testing for antibodies in the oral or nasal mucosa may be a low-cost and less invasive alternative to testing for antibodies in serum. Further studies are needed to studying and understanding of the duration of antibody detection in the naso-pharengial zone mucosa and how antibody concentrations change will have over time. SARS-CoV has the ability to infect the CNS via synapses, using olfactory nerve afferent fibers to reach the olfactory bulb, which increases the likeli hood that the virus will use this route of infection [27,28].

Comparisons of nasopharyngeal swabs from individuals with varying degrees of COVID-19 disease severity have demonstrated a violation of the early immune response. Epithelial cells showed increased activation of genes involved in antiviral responses in patients with mild to moderate COVID-19, particularly, genes stimulated by type I interferon are a very early alarm signal about the broader immune system activation [22,29,30].

In people who have developed severe COVID-19 requiring mechanical ventilation in the majority cases. Most notably, their epithelial cells had a significant muted response to interferon, despite the presence of large amounts of virus. At the same time, their smears showed increased numbers of immune cells,macrophages that promote inflammatory responses. “All people with severe COVID-19 had a suppressed response to interferon in epithelial cells early on, and they were never able to mount a defense,” says Ordovas-Montañez. “Having the right amount of interferon at the right time may be a crucial factor in fighting SARS-CoV-2 and other viruses” [31,32].

Conclusions

Olfactory disorder in patients with COVID-19 is an early significant symptom of the disease, usually severe duration with complete loss of olfactory function, but reversible in majority cases. The clinical picture represented a specific lesion of the respiratory tract, uncharacteristic for other respiratory viruses, with conditions safe-keeping for normal transnasal airflow or slight severity of edema, nasal mucosa hyperemia and/or rhinorrhea. The results of the studies presented that the nasal cavity olfactory epithelium is an area of increased conjunctiving, replication and accumulation of the new coronavirus, which is by to the active expression of two «host» receptors (ACE2 receptor blockers and *TMPS2* inhibitors) by olfactory epithelium numerous non-neuronal cells. [31,32]. The exact mechanism how virus leads to this symptom is still unclear. The proposed mechanism is that in “immunely balanced” individuals, the coronavirus damages the olfactory epithelium and causes acute local inflammation with edema and development of anosmia, which recovers after the acute phase. However, in individuals with a genetic predisposition to develop autoimmunity, the immune response to the virus can create a parallel autoimmune response, generating autoantibodies targeting olfactory receptors and related peptides (G-protein coupled receptors), which allows the inflammatory process to enter the central nervous system via the olfactory bulb, where inflammation is induced with the development of neurological symptoms, including persistent anosmia, fatigue, brain fog, etc. The clinical and pathophysiological significance definitive of olfactory symptoms remains to be elucidated [32].

REFERENCES

1. Lewis D. Is the coronavirus airborne? Experts can't agree. NatureNews. April 2, 2020. Access: https://www.nature.com/articles/d41586-020-00974-w
2. Coronavirus: WHO rethinking how Covid-19 spreads in air. BBC News. 8 July 2020. Access: https://www.bbc.com/news/world-53329946
3. Butowt R, Bilinska K, von Bartheld CS. Olfactory dysfunction in COVID-19: new insights into the underlying mechanisms. Trends Neurosci. 2023 Jan;46(1):75-90. doi: 10.1016/j.tins.2022.11.003. Epub 2022 Nov 16.PMID: 36470705
4. Agyeman AA, Lee Chin K, Landersdorfer CB, Liew D, Ofori-Asenso R. Smell and taste dysfunction in patients with COVID-19: a systematic review and meta-analysis. Mayo Clin Proc.2020; 95(8):1621–31. - PMC - PubMed
5. Butowt R, von Bartheld CS. Anosmia in COVID-19: Underlying Mechanisms and Assessment of an Olfactory Route to Brain Infection. Neuroscientist. 2021 Dec;27(6):582-603. doi: 10.1177/1073858420956905. Epub 2020 Sep 11. PMID: 32914699; PMCID: PMC7488171.
6. Mohamed R, Alsuhibani R, Almasaoud H et al.Assessment of anosmia/hyposmia in post-COVID-19 patients: a cross-sectional study in an eastern province of Saudi Arabia. Acta Biomed. 2023 Dec 5; 94(6):e2023259. doi: 10.23750/abm.v94i6.15130.PMID: 38054673
7. Shen J, Wu L, Wang P et al. Clinical characteristics and short-term recovery of hyposmia in hospitalized non-severe COVID-19 patients with Omicron variant in Shanghai, China. 2022 Nov 7;9:1038938. doi: 10.3389/fmed.2022.1038938. eCollection 2022.PMID: 36419783
8. Wu H, Sun Q, Yuan Set al. Receptors: Their Actions from Hypertension to Cognitive Impairment. Cardiovasc Toxicol. 2022 Apr; 22(4):311-325. doi: 10.1007/s12012-022-09730-0. Epub 2022 Feb 24.PMID: 35211833Free PMC article.Review.
9. Colizzi M, Peghin M, De Martino M et al. Mental health symptoms one year after acute COVID-19 infection: Prevalence and risk factors. 2023 Jan-Mar;16:38-46. doi: 10.1016/j.rpsm.2022.05.008. Epub 2022 Jun 17.PMID: 35755492
10. Cucinotta D, Vanelli M. WHO Declares COVID-19 a Pandemic. Acta Biomed. 2020 Mar 19;91(1):157-160. doi: 10.23750/abm.v91i1.9397. PMID: 32191675; PMCID: PMC7569573.
11. Xu X, Wu Y, Kummer AG et al Assessing changes in incubation period, serial interval, and generation time of SARS-CoV-2 variants of concern: a systematic review and meta-analysis. BMC Med. 2023; 21,: 374). https://doi.org/10.1186/s12916-023-03070-8
12. Shponka IS, Usova OM. The influence of external factors on the development of olfactory disorders in patients with the coronavirus disease 2019: an immunomorphological assessment Medicni perspektivi. 2024;29(1):4-9. https://doi.org/10.26641/2307-0404.2024.1.300438
13. Lu R, Zhao X, Li J et al. Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. Lancet. 2020. 395(10224): 565–574. DOI: https://doi.org/10.1016/s0140- 6736(20)30251-8
14. Komisarenko SV. Polyuvannya vchenykh na koronavirus SARS-COV-2 shcho vyklykaye COVID-19; naukovi stratehiyi podolannya pandemiyi [Scientists' hunt for the SARS-COV-2 coronavirus that causes COVID-19: scientific strategies for overcoming the pandemic]. Visn. Nac. Acad. Nauk Ukr. 2020; 8: 29-77. https://doi.org/10.15407/visn2020.08.029 (In Ukrainian)
15. Sungnak W, Huang N, Becavin C et al. SARS-CoV-2 entry factors are highly ex-pressed in nasal epithelial cells together with innate immune genes. Nat. Med. 2020. 26: 681–687. DOI: https://doi.org/10.1038/s41591-020-0868-6
16. Estimating the number of infections and the impact of non-pharmaceutical interventions on COVID-19 in 11 European countries. Access: https://www.imperial.ac.uk/media/imperial-college/medicine/mrc-gida/2020-03-30-COVID19-Report-13.pdf
17. Baig AM, Khaleeq A, Ali U, Syeda H. Evidence of the COVID-19 virus targeting the CNS: tissue distribution, host-virus interaction, and proposed neurotropic mechanisms. ACS Chem Neurosci. 2020;11(7):995–8. doi: 10.1021/acschemneuro.0c00122
18. Bilinska K, Jakubowska P, von Bartheld CS, Butowt R. Expression of the SARS-CoV-2 entry proteins ACE2 and TMPRSS2, in cells of the olfactory epithelium: identification of cell types and trends with age. ACS Chem Neurosci. 2020; 11(11):1555–62. doi: 10.1021/acschemneuro.0c00210 –
19. Hopkins C, Surda P, Kumar N. Presentation of new onset anosmia during the COVID-19 pandemic. Rhinology 2020;58(3):295–8. doi: 10.4193/Rhin20.116 - DOI - PubMed
20. Brann DH, Tsukahara T, Weinreb C et al. Non-neural expression of SARS-CoV-2 entry genes in the olfactory epithelium suggests mechanisms underlying anosmia in COVID-19 patients. Sci Adv. 2020;

6(31):eabc5801. doi: 10.1126/sciadv.abc5801 - DOI - PMC - PubMed
21. Baxter BD, Larson ED, Merle L et al. Transcriptional profiling reveals potential involvement of microvillous TRPM5-expressing cells in viral infection of the olfactory epithelium. bioRxiv [Preprint]. 2020 Dec 1:2020.05.14.096016. doi: 10.1101/2020.05.14.096016. Update in: BMC Genomics. 2021 Mar 30;22(1):224. doi: 10.1186/s12864-021-07528-y. PMID: 32511400; PMCID: PMC7263547.
22. Eliezer M, Hautefort C, Hamel AL et al. Sudden and Complete Olfactory Loss of Function as a Possible Symptom of COVID-19. JAMA Otolaryngol Head Neck Surg. 2020 Jul 1;146(7):674-675. doi: 10.1001/jamaoto.2020.0832. PMID: 32267483.
23. Horlenko OM, Hechko KA, Hechko MM et al, Cytomorphological characteristics of the Nasopharyngeal zone Mucosa in children with Covid-19.Wiadomości Lekarskie 2025;(9):1741-1745. DOI: https://doi.org/10.36740/WLek/212502
24. Zhang L, Jackson CB, Mou H et al. 0. The D614G mutation in the SARS-CoV-2 spike protein reduces S1 shedding and increases infectivity. Preprint. bioRxiv.
2020. doi: 10.1101/2020.06.12.148726
25. van Baarle D, Nawijn MC Variant-specific local tissue response to SARS-CoV-2 in the nasal mucosa. Nat Immunol. 2025 Feb; 26(2):152-154. doi: 10.1038/s41590-024-02067-6.
26. de Los Rios Kobara I, Jayewickreme R, Lee MJ et al. Interferon-mediated NK cell activation increases cytolytic activity against T follicular helper cells and limits antibody response to SARS-CoV-2. Stanford COVID-19 Biobank. Nat Immunol. 2025 Dec; 26(12):2201-2217. doi: 10.1038/s41590-025-02341-1. Epub 2025 Nov 21.
27. Bertlich M, Stihi C, Weiss BG et al. Characteristics of impaired chemosensory function in hospitalized COVID-19 Patients. Preprint SSRN. 2020. Access: doi: 10.2139/ssrn.3576889.
28. DosSantos MF, Devalle S, Aran V et al. Neuromechanisms of SARS-CoV-2: a review. Front Neuroanat. 2020; 14:37. doi: 10.3389/fnana.2020.00037
29 Horlenko OM, Hechko KA, Prylypko LB et al. Comprehensive approach to comparative characteristics of clinical and laboratory parameters of the study in children - in 6 months after covid-19 treatment and 6 months after Covid-19 vaccination. Wiadomości Lekarskie 2025;(4):838-844. DOI: https://doi.org/10.36740/WLek/203895
30. José Ordovás-Montañés, Carmen Gerlach, Pedro Moraes-Vieira et al. Navigating COVID-19: Starting a lab during the pandemic, Immunity.2021;Volume 54, Issue 10: 2169-2171. https://doi.org/10.1016/j.immuni.2021.09.020.
31. Walsh JML, Miao VN, Owings AH et al. Variants and vaccines impact nasal immunity over three waves of SARS-CoV-2. Nat Immunol. 2025 Feb;26(2):294-307. doi: 10.1038/s41590-024-02052-z.
32. Paula David, Anna Malkova, Chapter 23 - Anosmia in COVID-19 and post-COVID syndrome, Editor(s): Yehuda Shoenfeld, Arad Dotan, In Future of Autoimmunity Research, Autoimmunity, COVID-19, Post-COVID19 Syndrome and COVID-19 Vaccination, Academic Press. 2023: 487-494. https://doi.org/10.1016/B978-0-443-18566-3.00010-4.

Дата первинного надходження рукопису: 16.02.2026 р.
Дата прийняття рукопису до друку: 20.02.2026 р.
Дата публікації: 16.03.2026 р.