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The Role of Oral Microbiota and Mycobiota in the Development of Inflammatory Processes in Patients with Autism

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Abstract

The article addresses the relevant issue of the role of the oral microbiota and mycobiota in the development of inflammatory processes in patients with autism spectrum disorders (ASDs). The study aims to determine the significance of oral microbial and fungal communities in the pathogenesis of inflammatory conditions in patients with ASDs.

The analysis of current scientific sources demonstrated that the oral microbiota in children with ASDs is characterized by structural and functional alterations associated with the maintenance of inflammatory processes. The most significant changes were identified in saliva and dental plaque and included reduced bacterial diversity, shifts in the taxonomic profile, an increased proportion of opportunistic microorganisms, and a decreased abundance of commensal taxa. Culture-based studies revealed that the level of *Streptococcus mutans* above 10^5 colony-forming units (CFU) was detected in 54.5% of children with ASDs compared with 43.1% in the control group, while *Lactobacillus* spp. above 10^5 CFU was found in 47.7% and 49.0%, respectively. A reduction in local mucosal immunity indicators, particularly salivary IgA, was also observed, suggesting weakened protective mechanisms in the oral cavity.

At the same time, data on the oral mycobiota, especially *Candida* species, remain limited and insufficiently represented in quantitative studies. The findings indicate that dysbiotic changes in the oral microbiota and mycobiota may contribute to inflammatory processes in patients with autism and represent a promising direction for further research.

Keywords: Autism Spectrum Disorders (ASDs); Oral Microbiota; Oral Mycobiota; Inflammatory Processes; Oral Dysbiosis; Candida; Saliva; Mucosal Immunity; Children.

1. Introduction

Despite the active research into the microbiome–immune system–brain axis, most of the studies on autism focused mainly on the intestinal microbiota, while the microbiota, and especially the oral mycobiota, remain understudied. This imbalance is significant, as the oral cavity not only reflects the state of mucosal immunity, but can also act as a source of prolonged antigenic stimulation, maintaining local dysbiosis and increasing systemic inflammation. Currently available data indicate that fungal communities may participate in the pathophysiological processes associated with autism, but this information has not yet been sufficiently integrated into the concept of the role of the oral cavity as a separate microbial niche [1,2]. At the same time, recent reviews indicate that the oral mycobiota in children has significant importance for health and disease, and therefore its analysis may deepen the understanding of the mechanisms of the inflammatory phenotype in patients with ASDs [3]. In this regard, it remains relevant to summarize current data on the

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role of the microbiota and oral mycobiota in the development of inflammatory processes in patients with autism, as well as to determine their potential as diagnostic and pathogenetically significant markers [2–4].

ASDs are complex neurodevelopmental disorders whose pathogenesis cannot be explained by genetic or neurobiological mechanisms alone. In recent years, increasing attention has been paid to the role of microorganisms in the development of neuropsychological and behavioural disorders, as microbial agents are able to influence the immune response, chronic systemic inflammation, barrier properties of mucous membranes, and neuroimmune interactions [4]. Against this background, the fungal component of the microbiome is of particular importance, as *Candida* and other representatives of the mycobiota are considered as potential participants in pathological processes associated with immune activation, metabolic disorders, and changes in behavioural reactions in children with autism [1]. Current reviews also show that the mycobiota and antifungal antibodies are gradually being considered not only as concomitant markers, but also as promising diagnostic and prognostic targets in chronic immunoinflammatory diseases [2]. This expands the idea of the possible contribution of the fungal component to the formation of the inflammatory background in patients with autism.

The oral cavity deserves special attention in this context, as it is one of the most active microbial biotopes of the body, where bacteria, fungi, epithelium, salivary defence factors and local immunity constantly interact. Even though the main scientific interest was focused for a long time mainly on the bacterial composition of the oral microbiome, recent studies emphasise the independent importance of the oral mycobiota, particularly in childhood. It is noted that the children' oral microbiota is a dynamic system associated not only with the state of the oral tissues, but also with broader systemic consequences for the body [3]. That is why the oral microbiota and mycobiota should be considered as a potentially important link in the development and maintenance of inflammatory processes in patients with autism [4,5].

In recent years, ASD has increasingly been viewed not only as a neurodevelopmental or genetic condition, but also in terms of immunoinflammatory, metabolic, and microbiome-related mechanisms. Current studies emphasize the role of neuroinflammation, barrier dysfunction, oxidative stress, cytokine imbalance, and innate immune activation in autism pathogenesis [6–11]. These data support the involvement of microbial factors in maintaining a systemic inflammatory state that influences CNS functioning and behavioural manifestations. This has led to the development of the microbiome–immune system–brain axis concept [12–15].

Research on the gut microbiota remains the most developed direction, showing dysbiosis-associated alterations in microbial composition, intestinal barrier integrity, immune activation, and metabolic and neurotransmitter changes [16–19]. These processes are linked to behavioural disturbances and chronic inflammation in ASD [5,6,20,21]. However, this focus has overshadowed other mucosal ecosystems, particularly the oral cavity, which represents a highly reactive microbial environment involving bacteria, fungi, biofilms, and local immune factors [19,22–24].

Recent attention has also been directed to the fungal microbiome. Mycobiota is now considered an active participant in immune modulation, inflammatory maintenance, and barrier regulation [1,2]. A. Herman and A. P. Herman [1] discuss the potential role of *Candida* in ASD pathophysiology, while E. Diez-Martin et al. [2] highlight the diagnostic relevance of mycobiota and antifungal antibodies in chronic inflammatory conditions.

Accordingly, emerging studies on the oral microbiome in ASD are particularly significant. Children with ASD demonstrate alterations in salivary and dental plaque microbiota, reduced diversity, shifts in taxonomic composition, and changes in local immunity, including salivary IgA levels [25–29]. Z. Xiang and Y. Liu [3] describe the oral microbiome as a dynamic system involved in both health maintenance and disease development. Overall, these findings suggest that the oral cavity may act not only as a site of microbial imbalance but also as a contributor to chronic inflammation, immune dysregulation, and systemic antigenic stimulation in ASD.

Despite the significant increase in the number of studies on the role of the microbiome in the pathogenesis of ASD, most available studies focus mainly on the intestinal microbiota, the gut-brain axis, barrier dysfunction, and systemic neuroinflammation [16,22,23,30]. In contrast, the microbiota and the oral cavity mycobiota have been studied much less fully, although this biotope is one of the most active mucosal ecosystems of the body, where bacteria, fungi, the epithelial barrier, saliva and local immune mechanisms constantly interact [2–4]. Existing studies only outline individual changes in the oral microbiome in ASD, including shifts in the composition of saliva, dental plaque, tongue microbiome, and mucosal defence indices [25,28,31], but these data remain fragmentary, methodologically heterogeneous, and insufficiently integrated into the overall concept of the development of inflammatory processes in patients with autism.

Several fundamental questions remain unresolved. It has not been established which changes in the microbiota and oral mycobiota are pathogenetically significant for maintaining local and systemic inflammation in patients with ASD, and which only accompany the main pathological process. The evidence base regarding the oral mycobiota in patients with autism is particularly limited: available sources lack direct quantitative data on the colonization of fungal communities, particularly *Candida spp.,* in the oral cavity of children with ASD, while most conclusions are extrapolated from the intestinal biotope [1–3]. That is why the issue of the role of the oral microbiota and mycobiota as a separate pathogenetically significant link in the development of inflammatory processes in patients with ASDs requires further systemic generalization.

The novelty of this study is the comprehensive analysis of the role of the oral microbiota and mycobiota in the development of inflammatory processes in patients with ASDs, considering their impact on immune mechanisms, metabolic pathways, and neurotransmitter systems.

The aim of the study is to determine the role of the and microbiota oral mycobiota in the development of inflammatory processes in patients with autism.

2. Methods

The obtained results summarize current scientific data on the participation of the oral microbiota and mycobiota in the pathogenesis of neuroinflammatory processes in patients with autism and can serve as a theoretical background for further research in the field of neuroimmunology, microbiology, and psychoneurology. Expanding knowledge about the mechanisms of interaction between the oral microbiota and the central nervous system opens prospects for the development of new diagnostic approaches and therapeutic strategies in the treatment of ASDs.

The study was conducted using the method of systematic analysis of scientific literature on the role of oral microbiota and mycobiota in the development of inflammatory processes in patients with ASDs. The search for scientific sources was carried out in the international scientometric databases PubMed, Scopus, Web of Science, and Google Scholar. The following keywords and their combinations in English were used to search the literature: “autism spectrum disorder”, “mycobiota”, “neuroinflammation”, “Candida”, “Saccharomyces”, “Malassezia”, “microbial metabolites”, “neurotransmitters”. The analysis included scientific publications for 2020–2026, which contained the results of experimental, clinical, and review studies on the interaction of the oral microbiota with the immune and nervous systems in patients with autism.

3. Results

Analysis of the current literature has shown that in ASD, inflammatory processes should be considered not only from the perspective of the intestinal microbiome but also considering the state of the microbiota and mycobiota of the oral cavity. The oral cavity is one of the most active mucosal ecosystems of the body, in which bacterial and fungal communities, the epithelial barrier, saliva, local protective factors and immune mechanisms constantly interact [1,2,26,27]. A summary of the available data shows in Table 1 that in children with ASD, the oral microbiome has structural and functional features that may be associated with both local dental changes and the maintenance of a systemic inflammatory background [3,28,29,31].

Table 1 Main changes in the oral microbiota and mycobiota in patients with ASDs according to the literature

Habitat	Main changes detected	Potential clinical significance
Saliva	High levels of *S. mutans* and *Lactobacillus spp.* in some children	Maintenance of dysbiosis, cariogenic potential
Saliva, dental plaque	Changes in the composition of the oral microbiota, reduced diversity	Potential diagnostic marker of ASD
Tongue	Less pronounced changes in the microbiome	Heterogeneity of oral niches
Saliva	Decrease in secretory IgA	Weakening of local mucosal defence
Oral cavity	Importance of the oral microbiota in children	Potential involvement in inflammation

Source: developed by the authors based on [3,25,26,28,31].

Data analysis shows that microbial changes in patients with ASDs are not isolated but systemically organized and cover different biotopes of the oral cavity [25,26]. Most importantly, the identified changes concern not only the composition of microbial communities, but also the functional state of local mucosal immunity, which creates conditions for maintaining a chronic inflammatory environment. The totality of the above results indicates a violation of the colonization balance, weakening of the protective mechanisms of the mucosa, and a probable increase in intermicrobial interactions within oral biofilms [3,28,31]. This gives grounds to consider the oral cavity not only as a local focus of dysbiosis, but as a potentially active link in the formation of a systemic immunoinflammatory response in patients with autism.

One of the few studies that specifically reported quantitative culture values for children with autism is the study of F. Tulumbacı et al. [25]. The authors examined 95 children, 44 of whom had ASD and 51 were included in the control group. It was found that the level of *Streptococcus mutans* above 105 CFU was detected in 54.5% of children with autism versus 43.1% in the group without ASD, while the level of Lactobacillus spp. above 105 CFU was determined in 47.7% and 49.0% of children, respectively. Although the authors did not find a statistically significant difference between the groups, increased microbial loads in a significant proportion of children with ASD is clinically significant, as it confirms the need to take into account the oral microbial component in the assessment of the dental and general inflammatory status of these patients [25] (Table 2).

Table 2 Quantitative indicators of CFU of oral microorganisms in children with ASDs

Microorganism	Biomaterial	RAS group	Control group	Threshold value
Streptococcus mutans	Saliva	54.5% of children had a level $>10^5$ CFU	43.1% of children had a level $>10^5$ CFU	$>10^5$ CFU
Lactobacillus spp.	Saliva	47.7% of children had a level $>10^5$ CFU	49.0% of children had a level $>10^5$ CFU	$>10^5$ CFU

Source: developed by the authors based on [1,2,25].

The above cultural indicators do not exhaust the changes in the oral microbiome in patients with autism. Sequencing studies show much deeper shifts in the structure of bacterial communities. Based on the analysis of 111 oral samples, Y. Qiao et al. [26] demonstrated that the salivary and dental microbiota of children with ASD differ significantly from that of healthy children. The authors recorded a decrease in bacterial diversity, especially in dental plaque, an increase in the relative abundance of Haemophilus in saliva and *Streptococcus* in dental plaque, as well as a decrease in the number of commensal genera *Prevotella*, *Selenomonas, Actinomyces, Porphyromonas,* and *Fusobacterium*. It is important that the diagnostic model for saliva built on key microbial markers achieved an accuracy of 96.3%, which emphasizes the high potential of the oral biotope as a source of non-invasive diagnostic indicators [26].

Similar results are reported by S. Hicks et al. [27], who studied the active salivary microbiome in children aged 2–6 years. The authors found that 12 taxa differed between developmental groups, and 28 taxa allowed to distinguish children with ASD with and without gastrointestinal disorders. This is especially important for the pathogenetic model of this article, as it indicates that dysbiotic changes in autism are not limited to the intestinal biotope, but also extend to the oropharyngeal area [26,27]. Therefore, the oral cavity may act not just as an anatomical "entrance" to the gastrointestinal tract, but as a separate functionally active link in the "oral microbiome – mucosal immunity – inflammation" system [3,4,27].

However, not all parts of the oral cavity show the same severity of microbial changes. A. Abdulhaq et al. [31] showed that the tongue microbiome of children with ASD did not have statistically significant differences in species richness and diversity compared to neurotypical children, although its structure was dominated by *Prevotella, Streptococcus, Leptotrichia, Veillonella, Haemophilus,* and *Rothia*, which together accounted for more than 60% of the average microbiome. This result is of fundamental importance, as it indicates the heterogeneity of the oral microbiome: the most pronounced changes in autism may not be associated with all oral niches equally, but primarily with saliva and dental plaque, where biofilm formation, microbial interactions and contacts with local defence factors are more active [26,27].

An important addition to the bacterial block is the data on mucosal immunity in the oral cavity. W. Gong et al. [28] demonstrated that salivary IgA levels are significantly reduced in children with ASD and have diagnostic value for autism. The authors also showed that ASD-associated streptococci can reduce PIGR levels in salivary gland cells, which creates a mechanistic link between bacterial shifts and a weakening of local immune defence. This significantly strengthens the concept that the oral cavity in ASD is not only a site of altered microbial composition, but also an

environment with impaired mucosal immune homeostasis, capable of supporting chronic local and, probably, systemic inflammation [2,3,28].

It should be noted that new studies from 2025 further strengthen the importance of the oral cavity in the pathogenetic ideas about ASD. S. Meng et al. [29] showed that both in saliva and in dental plaque of children with autism, α-diversity is generally lower, and β-diversity is significantly different from controls. At the genus level, *Streptococcus* and *Porphyromonas* dominated, and LEfSe analysis revealed a number of taxa that were significantly associated with the ASD profile in children without caries, with a significance level of $p<0.001$. These results are methodologically important because they show that for the correct interpretation of the oral microbiome in autism, the influence of caries should be considered separately, and oral biofilms may contain markers that are more specific for ASD itself, and not only for concomitant dental pathology [29].

Regarding the oral mycobiota, the available data are much more modest. A. Herman and A. P. Herman [1] consider the possible involvement of Candida in the pathophysiology of autism, but the main body of data they analysed concerns the intestinal rather than the oral biotope. E. Diez-Martin et al. [2] emphasize that the mycobiota and antifungal antibodies have diagnostic and prognostic potential in diseases with a chronic immunoinflammatory component. Z. Xiang and Y. Liu [3] showed that the microbiome and mycobiome are a dynamic system associated with both the normal functioning of the oral mucosa and pathological conditions in childhood. However, quantitative data on the oral fungal communities in children with ASD are almost absent in the available sources. This should be regarded as a gap that requires separate cultural and molecular studies [1–3].

So, the results of the literature analysis give grounds to draw several fundamental conclusions. The oral cavity in patients with ASD is not a neutral biotope but demonstrates both quantitative cultural changes of individual microorganisms and significant structural rearrangements of bacterial communities [25,26,29,31]. Oral dysbiosis is combined with changes in local mucosal immunity, with a decrease in secretory IgA in saliva, which creates conditions for maintaining a chronic inflammatory environment [28]. The evidence base regarding oral mycobiota in patients ASD is still being formed, but there are already sufficient grounds to consider the fungal component of the oral cavity as a promising link in the development of inflammatory processes in patients with ASD.

4. Conclusion

The analysis of the current literature indicates that the oral microbiota and mycobiota are potentially significant participants in the development of inflammatory processes in patients with ASD. The oral cavity should be regarded not only as a site of microbial colonization but also as an active mucosal environment involved in shaping local and systemic immunoinflammatory responses.

The reviewed studies demonstrate structural and functional alterations of the oral microbiome in children with ASD, manifested by changes in microbial composition in saliva, dental plaque, and oral niches, as well as by impaired local mucosal immunity. These findings suggest a relationship between oral dysbiosis and mechanisms sustaining a chronic inflammatory state in autism. Among quantitatively studied microorganisms, *Streptococcus mutans* and *Lactobacillus* spp. remain the most frequently investigated, although available cultural data do not fully capture the complexity of microbial alterations.

The oral microbiota and mycobiota may be considered important modulators of neuroimmune interactions in ASD, highlighting their potential relevance for further research into the pathophysiology of these disorders.

Further studies should focus on a more detailed characterization of bacterial and fungal components of the oral ecosystem using combined culture-based and molecular approaches, including the analysis of saliva, dental plaque, biofilms, and local immune markers.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest.

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References

[1] Herman A, Herman AP. Could Candida overgrowth be involved in the pathophysiology of autism? *Journal of Clinical Medicine*. 2022;11(2):442. https://doi.org/10.3390/jcm11020442

[2] Diez-Martin E, Hernandez-Suarez L, Astigarraga E, Ramirez-Garcia A, Barreda-Gómez G. Mycobiota and antifungal antibodies as emerging targets for the diagnosis and prognosis of human diseases. *Journal of Fungi (Basel)*. 2025;11(4):296. https://doi.org/10.3390/jof11040296

[3] Xiang Z, Liu Y. The pediatric oral mycobiome: a comprehensive review of its role in health and disease. *Frontiers in Cellular and Infection Microbiology*. 2026;16:1711789. https://doi.org/10.3389/fcimb.2026.1711789

[4] Bransfield RC, Mao C, Greenberg R. Microbes and mental illness: past, present, and future. *Healthcare (Basel)*. 2023;12(1):83. https://doi.org/10.3390/healthcare12010083

[5] Sivamaruthi BS, Kesika P, Chaiyasut C, Ragu Varman D. Gut dysbiosis in neurodevelopmental disorders: linking microbiota signatures to cognitive rigidity in autism spectrum disorder. *Frontiers in Microbiology*. 2026;17:1760635. https://doi.org/10.3389/fmicb.2026.1760635

[6] Zarimeidani F, Rahmati R, Mostafavi M, et al. Gut microbiota and autism spectrum disorder: a neuroinflammatory mediated mechanism of pathogenesis? *Inflammation*. 2025;48(2):501–519. https://doi.org/10.1007/s10753-024-02061-y

[7] Priego-González L, Pardo PP, Redegeld F. The role of mast cells in autism spectrum disorder. *Neuroscience and Biobehavioral Reviews*. 2025;176:106263. https://doi.org/10.1016/j.neubiorev.2025.106263

[8] Kearns R. Gut-brain axis and neuroinflammation: the role of gut permeability and the kynurenine pathway in neurological disorders. *Cellular and Molecular Neurobiology*. 2024;44(1):64. https://doi.org/10.1007/s10571-024-01496-z

[9] Zhang R, Ding N, Feng X, Liao W. The gut microbiome, immune modulation, and cognitive decline: insights on the gut-brain axis. *Frontiers in Immunology*. 2025;16:1529958. https://doi.org/10.3389/fimmu.2025.1529958

[10] Długosz A, Wróblewski M, Błaszak B, Szulc J. The role of nutrition, oxidative stress, and trace elements in the pathophysiology of autism spectrum disorders. *International Journal of Molecular Sciences*. 2025;26(2):808. https://doi.org/10.3390/ijms26020808

[11] Doenyas C. Potential role of epigenetics and redox signaling in the gut-brain communication and the case of autism spectrum disorder. *Cellular and Molecular Neurobiology*. 2022;42(2):483–487. https://doi.org/10.1007/s10571-021-01167-3

[12] Młynarska E, Barszcz E, Budny E, et al. The gut-brain-microbiota connection and its role in autism spectrum disorders. *Nutrients*. 2025;17(7):1135. https://doi.org/10.3390/nu17071135

[13] Petropoulos A, Stavropoulou E, Tsigalou C, Bezirtzoglou E. Microbiota gut-brain axis and autism spectrum disorder: mechanisms and therapeutic perspectives. *Nutrients*. 2025;17(18):2984. https://doi.org/10.3390/nu17182984

[14] Hetta HF, Alanazi FE, Alqifari SF, et al. The gut-brain axis in autism: inflammatory mechanisms, molecular insights, and emerging microbiome-based therapies. *Molecular Neurobiology*. 2025;63(1):211. https://doi.org/10.1007/s12035-025-05321-6

[15] Krynicka P, Cortegoso Valdivia P, Morawski M, et al. Microbiota-driven strategies for managing IBD-associated risks: from infections to mental health. *Pharmaceuticals*. 2026;19(1):118. https://doi.org/10.3390/ph19010118

[16] Yadav A, Tadas M, Kale M, et al. Gut microbiota and behavioral ontogeny in autism spectrum disorder: a pathway to therapeutic innovations. *Physiology & Behavior*. 2025;299:114989. https://doi.org/10.1016/j.physbeh.2025.114989

[17] Dargenio VN, Dargenio C, Castellaneta S, et al. Intestinal barrier dysfunction and microbiota-gut-brain axis: possible implications in ASD. *Nutrients*. 2023;15(7):1620. https://doi.org/10.3390/nu15071620

[18] Lewandowska-Pietruszka Z, Figlerowicz M, Mazur-Melewska K. Microbiota in autism spectrum disorder: a systematic review. *International Journal of Molecular Sciences*. 2023;24(23):16660. https://doi.org/10.3390/ijms242316660

[19] Chen Y, Xu J, Chen Y. Regulation of neurotransmitters by the gut microbiota and effects on cognition in neurological disorders. *Nutrients*. 2021;13(6):2099. https://doi.org/10.3390/nu13062099

[20] Hu T, Dong Y, He C, Zhao M, He Q. The gut microbiota and oxidative stress in autism spectrum disorders. *Oxidative Medicine and Cellular Longevity*. 2020;2020:8396708. https://doi.org/10.1155/2020/8396708

[21] Cimmino F, Petrella L, Cristiano C, et al. Autism spectrum disorders and nutritional interventions: dimethylglycine and B-vitamins effects on behavior, inflammation, microbiota and mitochondria. *Biomedicine & Pharmacotherapy*. 2025;191:118477. https://doi.org/10.1016/j.biopha.2025.118477

[22] Davoody S, Halimi H, Zali A, et al. Double-edged sword effect of Sutterella in neurological disorders. *Neurobiology of Disease*. 2025;214:107032. https://doi.org/10.1016/j.nbd.2025.107032

[23] Rudzki L, Stone TW, Maes M, et al. Gut microbiota-derived vitamins in psychiatric health and disease. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*. 2021;107:110240. https://doi.org/10.1016/j.pnpbp.2020.110240

[24] Shrivastav K, Pandey M, Gor H, et al. Gut virome plays an extended role with bacteriome in neurological health and disease. *Journal of the Neurological Sciences*. 2026;481:125754. https://doi.org/10.1016/j.jns.2026.125754

[25] Tulumbacı F, Korkut E, Özer H. Comparative evaluation of oral health status in children with ASD. *Journal of Pediatric Infectious Diseases*. 2020;15(5):223–227. https://doi.org/10.1055/s-0040-1709659

[26] Qiao Y, Wu M, Feng Y, et al. Alterations of oral microbiota distinguish children with autism spectrum disorders. *Scientific Reports*. 2018;8:1597. https://doi.org/10.1038/s41598-018-19982-y

[27] Hicks SD, Uhlig R, Afshari P. Oral microbiome activity in children with autism spectrum disorder. *Autism Research*. 2018;11(9):1286–1299. https://doi.org/10.1002/aur.1984

[28] Gong W, Qiao Y, Li B. The alteration of salivary immunoglobulin A in autism spectrum disorders. *Frontiers in Psychiatry*. 2021;12:669193. https://doi.org/10.3389/fpsyt.2021.669193

[29] Meng S, Zhang Y, Liu X. Caries influences the composition of oral microorganisms in male children with ASD. *Microbial Pathogenesis*. 2025;207:107904. https://doi.org/10.1016/j.micpath.2025.107904

[30] Alharthi A, Alhazmi S, Alburae N, Bahieldin A. The human gut microbiome as a potential factor in autism spectrum disorder. *International Journal of Molecular Sciences*. 2022;23(3):1363. https://doi.org/10.3390/ijms23031363

[31] Abdulhaq A, Khodor S, Shatat IF. Tongue microbiome in children with autism spectrum disorder. *Journal of Oral Microbiology*. 2021;13(1):1936434. https://doi.org/10.1080/20002297.2021.1936434