

# Enigmas of the Oral Mycobiome: A Narrative Review of the Delicate Dance Between Commensal and Pathogenic Fungi

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**Background:** The oral cavity harbors diverse microbial communities essential for oral and systemic health. Dysbiosis, or disruption of this balance, contributes to disease. The objective of this review was to elucidate the complexity and dualistic nature of the oral mycobiome, emphasizing the interplay between commensal and pathogenic fungi.

**Methods:** A comprehensive review of current literature was conducted, focusing on the oral mycobiome—particularly the interplay between commensal and pathogenic fungi. Studies were selected based on relevance and quality, with key findings synthesized to provide a focused overview.

**Results:** Recent technological advances have revealed the oral microbiome's complexity, extending beyond bacteria to include fungi and viruses. *Candida* species, especially *C. albicans*, dominate the healthy oral mycobiome, but their shift to pathogenicity is influenced by bacterial interactions, immunity, and host factors. Non-*Candida* fungi, such as *Malassezia* and *Cladosporium*, are consistently present in health, indicating broader ecological roles. Cross-kingdom interactions between fungi and bacteria foster biofilm formation, increasing resistance to therapy and contributing to oral diseases like caries and periodontitis. Fungal dysbiosis is associated with systemic conditions, including diabetes and HIV, with oral mycobiome shifts serving as both biomarkers and modulators of disease. Next-generation sequencing has highlighted inter-individual variability, shaped by genetics, environment, and diet. The oral mycobiome also modulates immune responses, influencing susceptibility to infection.

**Conclusion** This review synthesizes current knowledge on the oral mycobiome, emphasizing the specific roles and interactions of commensal and pathogenic fungi, factors shaping their balance, and their emerging clinical significance

**Keywords:** Oral Mycobiome. Commensal, Pathogen, Fungi

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## INTRODUCTION

The oral cavity contains a complex community of microbial species.<sup>1-3</sup> Indigenous microbes interact and co-evolve with each other and with the host, adapting to dynamic and often rapidly changing conditions while maintaining a relatively stable overall composition.<sup>4</sup> These microbes also perform important community-level functions, such as providing colonization resistance.<sup>5</sup>

Recent research has focused mainly on bacteria, often overlooking fungi due to their low abundance, difficulties in sample preparation, and limited reference databases. Nevertheless, various symbiotic fungi—collectively referred to as the mycobiota—have been isolated from the oral cavity. These fungal organisms are significantly larger than bacteria and possess specialized metabolic gene clusters that enable them to respond to specific ecological pressures.<sup>6</sup> The most commonly reported human-associated fungal genera include *Candida*, *Saccharomyces*, *Malassezia*, *Aspergillus*, *Fusarium*, and *Cladosporium*.<sup>7</sup> The predominant oral fungi are *Candida*, *Aspergillus*, *Cladosporium*, *Saccharomyces*, *Fusarium*, *Penicillium*, *Pichia*, and *Cryptococcus*.<sup>8</sup>

The mycobiota can have a significant impact on health, both supporting host immunity and contributing to disease during oral dysbiosis. These effects depend on the interactions between microbes and their human host.<sup>9</sup> Understanding these interactions requires examining how mycobiota affects host health in different states, the factors that shape mycobiota diversity, and the current limitations in mycobiota research. Advancing research in the mycobiome field is essential. This review aimed to:

- ✧ To elucidate the complexity and dualistic nature of the oral mycobiome, emphasizing the interplay between commensal and pathogenic fungi.
- ✧ To assess the significance of the oral mycobiome in human health and disease.
- ✧ To identify current research gaps and highlight the need for further investigation in this emerging field

## METHODS

The information collected and the procedures followed in writing this article was done according to SANRA (Scale for the Assessment of Narrative Review Articles), and are summarized in Table A.1.A comprehensive literature search was conducted across multiple databases, including PubMed, Scopus, Web of Science, and Embase, to identify

relevant studies pertaining to the oral mycobiome and the interplay between commensal and pathogenic fungi. The search strategy incorporated a combination of keywords and Medical Subject Headings (MeSH) terms such as “oral mycobiome,” “fungal microbiota,” “Oral fungi AND commensals,” “Oral fungi AND pathogens,” “Oral fungi OR commensals”, and “Oral fungi OR pathogens”. A total of 2,540 records were identified, and after removing duplicates, a total of 1,234 records were identified: 420 from PubMed, 320 from Scopus, 280 from Web of Science, and 214 from Embase. Titles and abstracts were independently screened by two reviewers, resulting in the exclusion of 1,000 records that did not meet the inclusion criteria. The full texts of the remaining 234 articles were assessed for eligibility. Following this step, 60 studies were deemed relevant and included in the final narrative review.

Any disagreements between reviewers during the selection process were resolved through discussion or consultation with a third reviewer, ensuring a rigorous and unbiased selection of studies.

The Inclusion Criteria included:

### Study Focus:

- ◆ Studies investigating the composition, diversity, or dynamics of the oral mycobiome.
- ◆ Research addressing the role of commensal and/or pathogenic fungi in the oral cavity.
- ◆ Articles discussing mechanisms, interactions, or recommendations related to balancing commensal and pathogenic fungi in the oral environment.
- ◆ Studies examining the clinical implications of the oral mycobiome (e.g., oral candidiasis, fungal dysbiosis, oral health/disease).

### Study Type:

- ◆ Original research articles (e.g., observational studies, clinical trials, laboratory studies).
- ◆ Systematic reviews, meta-analyses, and narrative reviews relevant to the oral mycobiome.
- ◆ Guidelines or expert recommendations related to the management of oral fungi.

### Population:

- ◆ Human studies (adults or children).
- ◆ In vitro or animal studies if directly relevant to mechanisms or interventions in the oral mycobiome.

### Language:

- ◆ Publications in English.

### Publication Date:

Published within the last 10 years.

**The Exclusion Criteria included:**

**Irrelevant Focus:**

- ◆ Studies focused solely on non-oral mycobiomes (e.g., gut, skin) without clear relevance to the oral environment.
- ◆ Articles addressing only bacterial components of the oral microbiome without reference to fungi.

**Article Type:**

- ◆ Editorials, letters, conference abstracts, case reports, or commentaries without original data or substantive review content.

**Population:**

- ◆ Studies exclusively involving non-human subjects unless directly applicable to oral mycobiome mechanisms relevant to human health

**Language:**

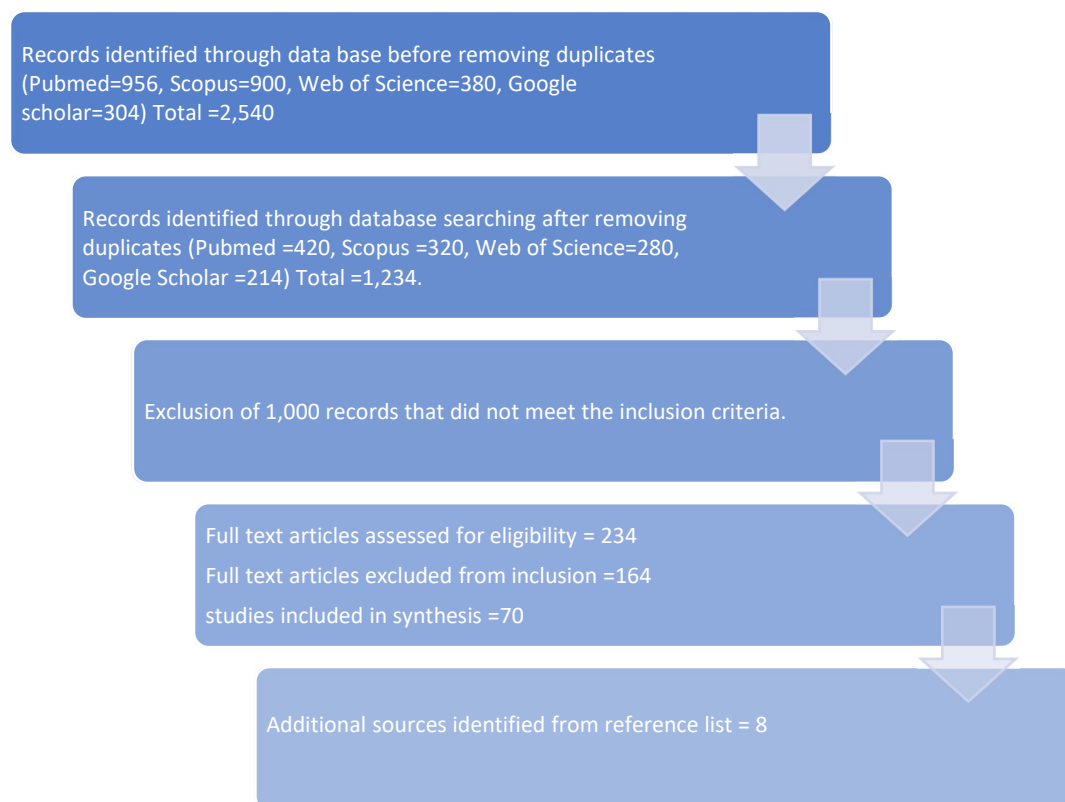
- ◆ Non-English publications.

**Publication Date:**

Publications older than 10 years, unless cited as foundational or highly relevant.

The search dates was between September 2024 to January 2026.

Figure A.1



**DISCUSSION**

**The complexity and Dualistic nature of Oral Mycobiome**

The oral mycobiome refers to the community of fungi present on mammalian oral mucosal surfaces, coexisting with other 2 major components of the oral cavity -the oral bacteriome (bacterial), and the oral virome or oral virobiome (viral).<sup>9</sup> It was previously considered a minor component of the oral microbiota due to limited research. However, with

the development of new sequencing testing and molecular identification methods, it has become a subject of increased investigation, indicating significant expansion in this field.<sup>10</sup> Among healthy individuals, the oral mycobiota is considered the most diverse and complex of all the body's mycobiomes.<sup>11</sup>

There are varying reports on the proportion of the oral mycobiome. Some studies report that fungi comprise 0.1% of the total gut microbial

population,<sup>12</sup> while others indicate that fungi constitute only 0.004% of the overall oral microorganisms, comprising approximately 100 species.

Common genera found include *Aspergillus*, *Aureobasidium*, *Candida*, *Cladosporium*, *Cryptococcus*, *Fusarium*, *Gibberella*, *Penicillium*, *Rhodotorula*, *Saccharomycetales*, and *Schizophyllum*.<sup>13</sup> Another study, which used interrogation of ITS1/ITS2 sequences, identified 85 fungal genera, of which 11 were unculturable. The number of fungal genera varied among individuals, ranging from 5 to 39.<sup>14</sup> The core fungal genera reported were: *Candida* (75%); *Cladosporium* (60%); *Aureobasidium* (50%); *Aspergillus* (35%); *Fusarium* (30%), and; *Cryptococcus* (20%). The highest prevalence was *Candida* species – *Candida albicans*, *Candida parapsilosis* and *Candida dubliniensis*.<sup>14</sup> The implication of this report is that the oral mycobiome, although minor in quantity, may play a significant role in oral health and disease due to its diversity and the presence of both common and rare genera. The high prevalence of *Candida* species, particularly *Candida albicans*, underscores its potential importance in both commensalism and pathogenicity within the oral environment. Furthermore, individual variability in mycobiome composition suggests that host factors, environment, and methodology influence the detected fungal community, making it a dynamic and individualized component of the oral microbiome.

Recent advancements in high-throughput NGS technology over the past decade have dramatically transformed the way the oral mycobiome have been studied. Moving beyond traditional, culture-based methodologies, researchers now utilize amplicon-based technologies that target fungal-specific housekeeping genes. This approach enables the identification of both cultivable and non-cultivable fungal species in a wide variety of environmental samples. These housekeeping genes are located within the fungal ribosomal RNA gene cluster (rRNA), which includes the 18S rRNA, 5.8S rRNA, and 28S rRNA genes, as well as the internal transcribed spacer regions (ITS1 and ITS2).<sup>15</sup> Sequencing uncovers a much greater fungal diversity than culture, including unculturable or previously unrecognized taxa. Both Sequencing and cultural method findings consistently identify *Candida* as the most prevalent genus, but sequencing reveals additional core genera (e.g., *Cladosporium*,

*Aureobasidium*) and individual variability. The clinical implication of this is that, relying solely on culture may underestimate the complexity of the oral mycobiome and overlook potential roles of minority or unculturable fungi in health and disease. Sequencing provides a more holistic understanding but should be interpreted alongside culture data for clinical relevance.

The oral fungi have been reported mainly in supragingival plaque and oral rinses.<sup>16</sup> Another study identified a broad presence of oral fungi in saliva samples, with two well-demarcated genus-level community types: *Malassezia* and *Candida*.<sup>17</sup> In addition to colonizing the mucosa, *Candida* species have been found on all tooth surfaces, including enamel, dentin, and cementum.<sup>18,19</sup> Multiple *Candida* species have been isolated from dentin and root caries in both children and adults, with reported prevalence rates of 66%–97% in children and 31%–56% in adults.<sup>19,20</sup> This raises the question of whether the oral mycobiome is also present elsewhere on denture plaque or other prosthetic devices worn in the mouth.

At the heart of this complexity lies the dualistic nature of the oral mycobiome. On the one hand, commensal fungi, such as *Candida* species, often exist harmlessly within the oral cavity, contributing to the ecological balance and possibly even conferring protective effects against pathogenic invaders.<sup>21</sup> These commensal fungi can interact with other members of the microbiome to suppress the overgrowth of potentially harmful organisms, modulate immune responses, and support mucosal health. A study suggested that apart from *Candida* being a human commensal, it might also have environmental reservoir.<sup>22</sup>

On the other hand, the same commensal fungi can become opportunistic pathogens under certain conditions, such as immunosuppression, antibiotic use, or changes in the oral environment. Pathogenic fungi can disrupt oral homeostasis, leading to diseases such as oral candidiasis and contributing to systemic health complications.<sup>23</sup>

The transition is influenced by a multitude of factors, including microbial interactions, host immunity, and environmental changes.<sup>21,23</sup> Fungal colonization of the mouth begins at birth, with sources including both maternal (vertical transmission) and environmental (horizontal transmission) factors.<sup>24,25</sup> Factors influencing mycobiome development include gestational age, birth weight, delivery method, and, later in life, diet.<sup>23,24</sup> In adults, the

composition of the oral mycobiome is further shaped by diet, body weight,<sup>26</sup> gender, age, antibiotic and antifungal use,<sup>27</sup> lifestyle, oral hygiene, and geography.<sup>26,28</sup>

Understanding the interplay between commensal and pathogenic fungi is essential for elucidating the mechanisms that govern oral health and disease. This dualistic relationship underscores the necessity of maintaining a balanced oral mycobiome, as disturbances can shift the equilibrium toward disease. Future research aimed at deciphering these complex interactions holds the potential to pave the way for novel therapeutic strategies targeting the oral mycobiome, ultimately improving both oral and systemic health outcomes.

In Africa, *Candida albicans* is the most common cause of oral fungal infection, accounting for 48% to 87% of cases. Recently, there has been an emergence of infections caused by non-*albicans* *Candida* species,<sup>29,30</sup> such as *C. parapsilosis*, *C. glabrata*, *C. tropicalis*, *C. dubliniensis*, *C. krusei*, and *C. guilliermondii*.<sup>31</sup>

It is important to note that the diagnosis of oral candidiasis is often based solely on clinical presentation, without identification of the causative agent. However, empirical management is frequently inadequate, and suboptimal treatment can result in antifungal resistance, persistent symptoms, and life-threatening dissemination, significantly impacting quality of life.<sup>29</sup>

Table 1: The Nigerian experience of oral fungal isolation

| Study/Author                         | Study Title                                                                                                                               | Oral Fungi Isolates Obtained                                                                                                           | Disease Condition |
|--------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------|-------------------|
| Ekwealor et al., 2023 <sup>32</sup>  | Prevalence and antifungal susceptibility pattern of oral candidiasis among HIV-infected patients in a Mission Hospital, southeast Nigeria | <i>Candida albicans</i>                                                                                                                | Oral Candidiasis  |
| Osaigbovo et al., 2017 <sup>33</sup> | Fluconazole Resistance among Oral Candida Isolates from People Living with HIV/AIDS in a Nigerian Tertiary Hospital                       | <i>Candida albicans</i> , <i>C. parapsilosis</i> , <i>C. glabrata</i> , <i>C. tropicalis</i> , <i>C. krusei</i> , <i>C. famata</i>     | Oral Candidiasis  |
| Oladejo & Abioye 2023 <sup>34</sup>  | Identification and Characterization of Candida Species Isolated from Clinical Specimens in Illorin, Kwara State.                          | <i>Candida albicans</i> , <i>C. tropicalis</i> , <i>C. glabrata</i> , <i>C. tropicalis</i> , <i>C. krusei</i> , <i>C. dubliniensis</i> | Oral Thrush       |
| Lawal et al., 2025 <sup>35</sup>     | Prevalence and Associated Risk Factors of Oropharyngeal Candidiasis among Sero-positive HIV Patients in Southwestern Nigeria              | <i>Candida albicans</i> , <i>C. parapsilosis</i>                                                                                       | Oral Candidiasis  |

Significance of the Oral Mycobiome in Human Health and Disease

The oral mycobiome, residing in the oral cavity, plays a significant role in maintaining human health and influencing disease processes. Although bacteria have traditionally received most attention in oral microbiome studies, recent advances have highlighted the importance of fungi, particularly species such as *Candida*, *Aspergillus*, and *Malassezia*.<sup>10, 36,37</sup> Fungal cells are reported to be approximately ten times wider and one hundred times larger in volume than bacterial cells.<sup>38</sup> Furthermore, fungi have larger genomes, greater genetic variability, and more pronounced morphological diversity compared to bacteria, as well as unique metabolic and immunoregulatory

functions. The healthy human oral cavity harbors a diverse fungal community, primarily composed of species from the Ascomycota and Basidiomycota, as well as other phyla.<sup>39,40</sup> The oral mycobiome is generally stable due to its interactions with bacteria, the host immune system, and the oral mucosa. However, it can undergo dysbiosis when influenced by certain factors, leading to potential imbalances and health implications.<sup>41</sup> A balanced mycobiome helps in preventing colonization by pathogenic microbes, modulating immune responses, and participating in nutrient metabolism.<sup>42</sup> The oral mycobiome engages in complex interdomain interactions with bacterial communities as well as intradomain interactions among different fungal species within the oral cavity. This was extensively

reviewed by Mishra et al. (2021).<sup>43</sup> These dynamic relationships help maintain microbial balance and prevent the overgrowth of pathogenic microorganisms. By competing for space and nutrients, producing antimicrobial compounds, and modulating the local immune response, the oral mycobiome plays a crucial role in protecting the oral environment from pathogenic colonization and maintaining overall oral health.

### Modulation of the Immune Response

The immune system interacts closely with the mycobiome. This interaction is essential for maintaining health, supporting immune tolerance, and regulating the composition of microbial communities.<sup>44</sup>

### Interactions Between Oral Bacteria and Fungi

Microbial communities in the oral cavity do not exist as isolated entities; rather, they dynamically shape and are shaped by their microenvironment through a network of inter- and intra-species interactions. These interactions, which can be physical, chemical, or metabolic, collectively generate diverse micro-niches that underpin oral ecosystem function and resilience.<sup>45</sup>

Physical interactions, such as co-aggregation and co-adhesion, between bacterial and fungal cells are not merely passive associations, but actively facilitate communication and genetic exchange.<sup>46,47</sup> For example, the formation of membranous structures by *Streptococcus* species and *C. albicans* within dental plaque exemplifies how physical proximity facilitates metabolic cooperation and signaling. Such spatial organization creates a scaffold for nutrient sharing, competitive exclusion, and collective defense mechanisms.<sup>48,49</sup>

Metabolic interdependence further structures the oral microbial community. Bacteria frequently engage in both competition and cooperation, vying for shared nutrients or capitalizing on metabolic byproducts produced by neighboring species.<sup>50,51</sup> Quorum sensing emerges as a central regulatory mechanism, enabling bacteria to synchronize gene expression and coordinate behaviors, such as biofilm formation or antagonism of fungal growth. For instance, *Streptococcus mutans* can suppress *Candida albicans* germ tube formation via quorum-sensing molecules, while *Actinobacillus actinomycetemcomitans* deploys autoinducer-2 (AI-2) to inhibit *Candida* biofilm development—

demonstrating the nuanced, species-specific nature of these cross-kingdom interactions.<sup>52,53</sup>

The oral mycobiome's contributions extend beyond nutrient metabolism, encompassing immune modulation, digestion, nutrient absorption, and even neurotransmitter production. The distinctive characteristics of fungi—including their larger size, genetic diversity, and unique metabolic and immunoregulatory capacities—position them as specialized actors within the oral ecosystem.<sup>44</sup> Importantly, the stability and diversity of the oral mycobiome, maintained through ongoing crosstalk with bacterial communities and the host, are essential for oral homeostasis. Disruption of this finely tuned balance—dysbiosis—not only predisposes to oral diseases but may have systemic implications. Thus, synthesizing these lines of evidence highlights the indispensable and multifaceted roles of fungi in oral health and underscores the importance of targeting fungal-bacterial interactions in strategies to prevent and treat oral disease.<sup>54-56</sup>

### Participating in nutrient metabolism

The oral mycobiome plays a crucial role in maintaining overall oral and systemic health by contributing to nutrient metabolism and supporting the immune system.<sup>42,57</sup> It exists in close symbiosis with the host, engaging in essential processes such as digestion, nutrient absorption, immune regulation, and even the production of neurotransmitters.<sup>58</sup> Through these interactions, the oral mycobiome not only aids in breaking down complex dietary components but also helps modulate immune responses, creating a balanced environment that is vital for health.<sup>59</sup>

Within the oral cavity, bacteria and fungi form complex communities where metabolic interactions are common. Bacteria frequently rely on one another for essential nutrients: some species compete directly for the same resources, while others engage in cooperative relationships where the metabolic byproducts of one organism serve as nutrients for another. This dynamic interplay creates a constantly shifting balance between competition and cooperation within the microbial ecosystem.<sup>60</sup>

A key mechanism that regulates community behavior is quorum sensing—a sophisticated form of cell-to-cell communication. Through quorum sensing, bacteria can coordinate their activities based on population density, enabling collective behaviors that enhance survival or defense. For

example, *Streptococcus mutans* can secrete quorum-sensing molecules that inhibit the germ tube formation of *Candida albicans*, a process critical to the pathogenicity of this fungus. Similarly, *Actinobacillus actinomycetemcomitans* produces autoinducer-2 (AI-2), a signaling molecule that can suppress the formation of *Candida* biofilms, though the specific mechanisms of inhibition may differ between microbial species.<sup>61,62</sup>

Overall, these intricate interactions among oral microbes underscore the complexity of the oral ecosystem, where both competitive and cooperative behaviors shape the community's composition and, ultimately, influence host health.

The importance and implications of these insights are substantial for both scientific understanding and clinical practice:

**Holistic View of Oral Health:** Recognizing that oral health is maintained by a dynamic, interdependent community of bacteria and fungi—rather than by individual species—shifts the focus from targeting single pathogens to managing the balance and interactions within the entire microbiome.

**Targeted Therapeutic Strategies:** By understanding the specific ways in which bacteria and fungi communicate, cooperate, or compete, researchers and clinicians can develop targeted interventions. For example, therapies might aim to disrupt harmful signaling pathways (such as those that promote fungal overgrowth), support beneficial microbial interactions, or restore balance after dysbiosis.

**Disease Prevention and Diagnosis:** The synthesis highlights those disruptions in microbial balance (dysbiosis) can lead to oral and possibly systemic diseases. Monitoring the composition and activity of both bacterial and fungal communities may improve early detection of disease risk and enable more precise preventive strategies.

**Personalized Medicine:** The distinct roles and interactions of fungal species suggest that individual differences in the oral mycobiome could influence susceptibility to disease and response to treatment. This opens avenues for personalized oral healthcare based on a person's unique microbiome profile.

**Broadening Research Horizons:** Appreciating the complexity of oral microbial ecosystems encourages exploration beyond bacteria to include fungi and their cross-kingdom interactions. This broader perspective can lead to new discoveries about oral-systemic health links, novel biomarkers, and innovative therapeutic targets.

## Commensal Fungi: Pathogenic Potential and Dysbiosis

Fungal species such as *Candida*, which typically exist as harmless commensals in the oral cavity, can shift to a dysbiotic state under certain conditions—immune suppression, antibiotic use, disrupted host barriers, or changes in microbial community composition.<sup>63</sup> This transition is often associated with increased pathogenicity and clinical infection. The report of transition from commensalism to pathogenicity in the oral cavity is a result of adherence of fungi to the epithelial cells by factors such as Als3, Hwp1, and Iff4 to establish persistent colonization. Biofilms are reported to be formed, having complex structure of different morphological form (yeast - hyphae) with further cell proliferation and filamentation occurring. At this phase, the cells develop elongated protrusions that grow into filamentous hyphal forms. Hyphae formation marks the onset of biofilm development. During the maturation phase, an extracellular polysaccharide matrix accumulates. The final phase is characterized by the dispersion of non-adherent cells, enabling the initiation of new biofilms, and facilitating tissue dissemination.<sup>64</sup>

The extracellular polysaccharide matrix is composed of extracellular polymers and extracellular DNA, both of which are integral to maintaining biofilm structure. Extracellular DNA also plays a crucial role in anchoring the biofilm to the substrate. A key component of the extracellular matrix is  $\beta$ -1,3-glucans, which substantially contribute to the biofilm's resistance to antifungal drugs by limiting drug access to target cells. *C. albicans* cells within biofilms release greater amounts of  $\beta$ -1,3-glucans into the extracellular matrix compared to planktonic cells.<sup>65,66,67</sup> There are suggestion of variability in species in the biofilms of different individual, and also intrapersonal variability in the course of the day.<sup>68</sup> However, in a state of dysbiosis, both fungal and bacterial biodiversity can be compromised.<sup>58</sup> The interplay between the bacteriome and mycobiome remains critical under these circumstances. For instance, frequent carbohydrate intake combined with poor oral hygiene may result in acidification of the oral niche due to sugar fermentation by bacteria. This drop in pH imposes selective pressure, enabling certain microbes to thrive while others diminish.<sup>43</sup> Similarly, exposure to antibiotics or immunodeficient states can reduce biodiversity, favoring the overgrowth and proliferation of organisms such as *Candida*.<sup>69</sup> Such conditions may result in oral

candidiasis or thrush.<sup>70</sup> Notably, many newborns who have not yet developed sufficient microbial diversity are prone to thrush, further highlighting the importance of a diverse microbiome in maintaining immune competence and controlling opportunistic organisms.

During dysbiosis, fungi may face selective pressure from antifungal agents, leading to the emergence and enrichment of resistant strains.<sup>71,72</sup> *Candida albicans* and non-*albicans* species (e.g., *Candida glabrata*, *Candida auris*) are notable for developing resistance to azoles, echinocandins, and polyenes.<sup>73</sup>

**The Mechanisms of Resistance can be genetic mutations:** Alterations in drug target sites (e.g., ERG11 for azoles, FKS1 for echinocandins), **efflux pumps:** Upregulation of transporter proteins (e.g., CDR1, MDR1) that remove antifungal drugs from the cell, **biofilm formation:** Biofilms confer inherent resistance due to reduced drug penetration and altered metabolic states, or **horizontal gene transfer:** Exchange of resistance determinants may occur, especially in mixed microbial communities.<sup>74-76</sup>

**The Trends and Clinical Implications of these are:**

- **Increasing prevalence:** Surveillance data indicate rising rates of antifungal resistance, particularly among non-*albicans Candida* and emerging pathogens like *C. auris*.
- **Therapeutic challenges:** Resistant strains complicate treatment, requiring alternative or combination therapies and raising the risk of treatment failure.
- **Microbiome disruption:** Broad-spectrum antifungal use can further disrupt microbial balance, promoting expansion of resistant fungi.

**The Prevention and Management will include:**

- **Antifungal stewardship:** Prudent use of antifungals is critical to slow resistance emergence.
- **Diagnostic advances:** Rapid identification and susceptibility testing help tailor therapy.
- **Restoration of microbiota:** Strategies to restore commensal balance (e.g., probiotics, microbiome therapies) are under investigation.

Fungal dysbiosis can lead to infection and invasion, particularly in individuals exposed to antineoplastic or immunosuppressive drugs, broad-spectrum antibiotics, poor oral hygiene, prosthetic devices, poor diet, lack of clean water, or grafts.<sup>71</sup> Changes in the mycobiome have also been associated with,

periodontal disease, dental caries, oral lichen planus, systemic conditions such as diabetes, HIV/AIDS, and certain cancers.<sup>77,78</sup>

### Quality of Evidence

The references provide a comprehensive and up-to-date overview of oral and gut microbiota research, covering bacterial and fungal communities, biofilm formation, immunity, disease associations, and antimicrobial resistance. They are sourced from a wide range of reputable journals and include both foundational studies and the latest publications, ensuring scientific rigor and relevance. The bibliography reflects a global and interdisciplinary perspective, integrating research from microbiology, immunology, dentistry, and medicine. It balances review articles and original research, addresses health and disease states, and covers specific mechanisms and taxa. Modern methodologies and clinical relevance further enhance the utility and credibility of the references.

### Limitations of the included studies

The majority were observational in nature, rather than systematic reviews, meta-analyses, or narrative reviews. Observational studies cannot establish causality, are subject to confounding variables, selection bias, recall bias, reporting bias, and have limited control over extraneous factors.

### Future Perspectives

The outlook for balancing the commensal and pathogenic oral mycobiome is both promising and multifaceted. Key areas for future exploration include:

1. **Enhanced Understanding of the Mycobiome:** Continued research will likely deepen our knowledge of the specific roles that various fungi play in oral health and disease. Understanding how commensal fungi prevent pathogen colonization will be particularly important.
2. **Targeted Therapies:** Advances in biotechnology may lead to the development of antifungal treatments that selectively eliminate harmful fungi while preserving beneficial species, thereby helping to maintain a balanced mycobiome.
3. **Microbiome Modulation:** New strategies, such as the use of fungal-specific prebiotics and probiotics, could emerge to promote the growth of beneficial fungi and inhibit pathogenic ones.
4. **Personalized Approaches:** As understanding of individual microbiomes grows, personalized

treatments and prevention strategies can be developed. Tailoring oral care routines to an individual's unique mycobiome composition may improve outcomes.

5. Integration with Oral Health Practices: Future dental practices might incorporate mycobiome assessments into routine check-ups, enabling early detection of imbalances and timely interventions.
6. Systematic Research: More comprehensive studies on the interactions between the oral mycobiome and other components of the oral microbiome (such as bacteria and viruses) will be necessary to fully appreciate their collective impact on oral health.
7. Public Awareness and Education: Increasing public understanding of the oral mycobiome's importance may encourage better oral hygiene and healthier lifestyle choices.

## CONCLUSION

The oral mycobiome is a complex and dynamic community, embodying a dualistic nature in which commensal fungi support health while pathogenic fungi can promote disease. This review aims to elucidate this complexity, highlighting the interplay between beneficial and harmful fungi and emphasizing their significance for oral and systemic health. Despite recent advances, critical research gaps remain—including the mechanisms driving microbial balance, factors influencing transitions from commensalism to pathogenicity, and the precise role of the mycobiome in disease. Addressing these gaps will require innovative research strategies and comprehensive integration of mycobiome analysis with broader microbiome studies. Ultimately, a deeper understanding of the oral mycobiome will inform targeted interventions to restore microbial homeostasis and improve health outcomes.

## Source of Support

Nil.

## Conflict of Interest

None declared

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