

INTERACTIONS BETWEEN THE ORAL MICROBIOME AND THE UROGENITAL TRACT: IMPLICATIONS FOR SYSTEMIC HEALTH AND INFLAMMATORY DISEASES

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Abstract

The human microbiome plays a fundamental role in maintaining systemic health, with growing recognition of cross-communication between distant microbial ecosystems. This review explores the bidirectional relationship between the oral microbiome and the urogenital tract, emphasizing how dysbiosis in the oral cavity, particularly in the context of periodontal disease, may influence urogenital health through systemic inflammation, microbial dissemination, and immune modulation. We examine the composition and functions of both the oral and urogenital microbiomes, as well as their susceptibility to internal and external perturbations such as antibiotic therapy, hormonal shifts, and lifestyle factors. Multiple biological mechanisms, including hematogenous spread, cytokine-mediated inflammation, and microbial signaling through the gut-bladder axis, are discussed as possible routes of interaction. Clinical and epidemiological evidence supports associations between poor oral health and increased risk of lower urinary tract symptoms, prostatitis, and recurrent urinary tract infections. Therapeutic implications include the integration of oral care in urological management, microbiome-targeted therapies such as probiotics, and a multidisciplinary approach to chronic inflammatory conditions. Future research should focus on causal validation and the development of personalized interventions addressing microbial interconnectivity across systems.

Keywords: Oral microbiome, urogenital tract, periodontal disease, systemic inflammation, dysbiosis

Introduction

The human body harbors trillions of microorganisms forming complex and dynamic communities collectively known as the microbiome. Among these, the oral and urogenital microbiomes have historically been studied in isolation, yet growing evidence suggests that cross-talk between these microbial ecosystems may influence systemic health and contribute to the pathogenesis of various inflammatory disorders. The oral cavity is the gateway to the gastrointestinal and respiratory tracts and hosts over 700 species of bacteria,

fungi, viruses, and protozoa. It plays a critical role in maintaining local and systemic immune homeostasis, while dysbiosis in the oral microbiome has been linked to conditions such as periodontal disease, cardiovascular pathology, diabetes mellitus, and adverse pregnancy outcomes [1-3].

Similarly, the urogenital tract possesses its specialized microbiome, particularly in women, where vaginal microbiota dominated by *Lactobacillus* species provide protection against

pathogens and maintain a healthy mucosal barrier. Disruption of this balance has been associated with bacterial vaginosis, urinary tract infections (UTIs), reproductive complications, and increased susceptibility to sexually transmitted infections (STIs). In males, although less studied, the urethra and prostate also host distinct microbial communities that may influence urological health [1-4].

A growing body of literature suggests that chronic oral infections such as periodontitis may contribute to systemic inflammatory burden and increase the risk of urogenital complications. The proposed mechanisms include hematogenous dissemination of oral pathogens or their endotoxins, translocation of inflammatory mediators, and modulation of the host's immune response. For instance, periodontal pathogens like *Porphyromonas gingivalis* and *Fusobacterium nucleatum* have been detected in distant anatomical sites, including the amniotic fluid, placenta, and even the urinary tract, suggesting their potential to migrate and colonize extraoral environments [2-4].

Furthermore, systemic inflammation driven by oral dysbiosis may exacerbate existing urological conditions such as interstitial cystitis, chronic prostatitis, or overactive bladder syndrome. These interactions are not merely coincidental but reflect a broader systemic response where local microbial imbalances trigger a cascade of immune and metabolic alterations with distant consequences. It is important to note that the oral microbiome also shares functional and compositional similarities with the gut microbiome, which in turn has established bidirectional links with the urogenital system via the so-called gut-bladder axis. Therefore, the oral

microbiota may indirectly influence the urogenital microbiome through shared immunological and metabolic pathways [1,3].

The clinical relevance of these observations is underscored by epidemiological studies associating poor oral health with increased incidence of urinary tract infections, particularly in vulnerable populations such as the elderly, pregnant women, and individuals with diabetes or immune dysfunction. Moreover, the widespread use of systemic antibiotics in the management of both dental and urological infections can disrupt microbial homeostasis across multiple mucosal surfaces, potentially leading to resistant infections and recurrent inflammatory states. This highlights the importance of adopting a more integrated approach to microbiome research and clinical care [2-5].

The purpose of this review is to synthesize current evidence on the interactions between the oral microbiome and the urogenital tract, focusing on mechanistic pathways, clinical implications, and future therapeutic opportunities. By exploring the oral-urogenital axis through a multidisciplinary lens, we aim to promote awareness of these underrecognized links and stimulate further research into microbiome-targeted strategies for disease prevention and systemic health optimization.

The oral microbiome: composition, functions, and dysbiosis

The oral microbiome is one of the most diverse and complex microbial ecosystems in the human body, second only to the gut. It comprises over 700 identified microbial species, including bacteria, fungi, archaea, protozoa, and viruses. These

microorganisms colonize various niches within the oral cavity, such as the tongue, cheeks, hard and soft palate, teeth surfaces, gingival sulcus, and saliva. Core bacterial

genera include *Streptococcus*, *Actinomyces*, *Veillonella*, *Fusobacterium*, *Prevotella*, and *Neisseria*, which coexist in a balanced, symbiotic relationship with the host [2,4-6].

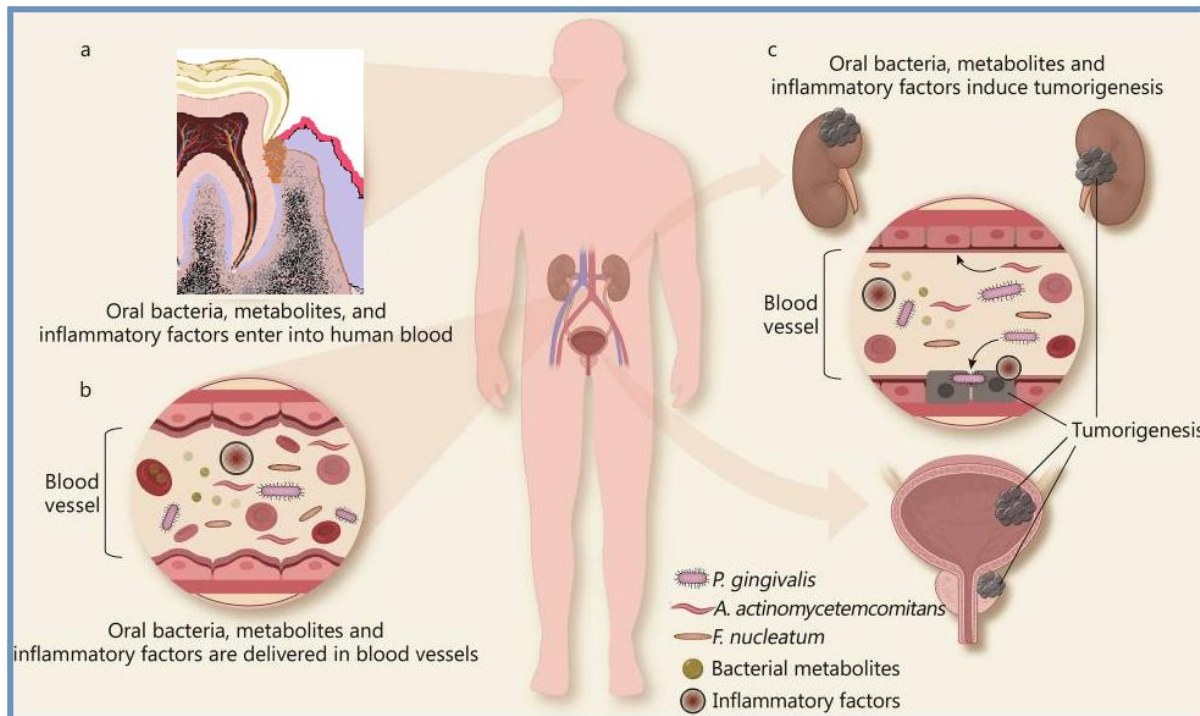


Figure 1. Oral bacteria (a), their metabolic byproducts, and inflammatory mediators may enter systemic circulation (b) and reach the urogenital tract via the bloodstream (c). Once localized, bacterial components such as lipopolysaccharides (LPS), FadA adhesin, or gingipains can contribute to carcinogenic processes in the bladder, prostate, and kidneys. Chronic exposure to these agents may induce epithelial transformation, promote cellular proliferation, enhance tissue invasion, and stimulate tumor-associated angiogenesis. Inflammatory species like *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, and *Aggregatibacter actinomycetemcomitans* are frequently implicated in this systemic pro-tumorigenic cascade [5,6]

The oral microbiome plays critical roles in maintaining local and systemic health. Locally, it contributes to nutrient metabolism, regulates pH, and provides colonization resistance against exogenous pathogens through microbial competition and the production of antimicrobial compounds. It also modulates the host's immune responses by interacting with oral epithelial cells and immune mediators, helping maintain a state of immunological tolerance and mucosal integrity. Beyond the

oral cavity, microbial metabolites and inflammatory signals can influence distant organs via systemic circulation, suggesting the oral microbiome's relevance in systemic homeostasis [2-6].

However, disruption of this delicate microbial equilibrium, referred to as dysbiosis, can lead to the development of oral diseases, including dental caries, gingivitis, and periodontitis. Dysbiosis is typically characterized by a shift from beneficial commensal species to more

pathogenic, proteolytic, and inflammatory organisms like *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola*, collectively known as the “red complex.” These pathogens trigger a sustained inflammatory response, causing tissue destruction, alveolar bone resorption, and potential entry of bacteria and toxins into systemic circulation [4-6].

Table 1 summarizes key components of the oral microbiome, highlighting their roles in maintaining oral and systemic health. It outlines ecological niches, physiological functions, triggers of dysbiosis, and consequences, both local and systemic, emphasizing the oral cavity's influence beyond dental disease [3-9]

Table 1. Oral microbiome

Category	Function/role	Examples
Main microbial genera	Dominant taxa in healthy oral microbiota	Streptococcus, Actinomyces, Veillonella, Fusobacterium
Habitats in the oral cavity	Primary ecological niches colonized by microbes	Tongue, gingival sulcus, teeth surfaces, cheeks, and saliva
Physiological functions	Beneficial physiological and immunological roles	pH regulation, nutrient metabolism, and immune modulation
Homeostasis markers	Indicators of microbial and host equilibrium	Low inflammation, balanced biofilm, stable microbiota
Triggers of dysbiosis	Factors that disrupt microbial balance	Smoking, diabetes, poor hygiene, antibiotics, and xerostomia
Pathogenic shift (dysbiosis)	Microbial composition changes associated with disease	<i>P. gingivalis</i> , <i>T. forsythia</i> , <i>T. denticola</i> (Red complex)
Local consequences	Clinical manifestations in the oral cavity	Gingivitis, periodontitis, caries, and mucosal lesions
Systemic impact	Impact beyond the oral cavity via inflammation or dissemination	Elevated IL-6, TNF- α ; bacteremia, systemic inflammation

Oral dysbiosis has been increasingly implicated in a range of extraoral pathologies. It contributes to systemic inflammation through elevated levels of C-reactive protein, interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α). These inflammatory mediators, once in the bloodstream, can reach remote organs including the cardiovascular system, placenta, kidneys, and urogenital tract. Additionally, bacteremia following routine oral procedures or daily activities such as tooth brushing can result in microbial dissemination to extraoral sites [5-9].

Understanding the composition and function of the oral microbiome is crucial

for identifying how its dysregulation may contribute to urogenital inflammation and pathology. As research advances, the oral cavity is no longer viewed solely as a site of localized disease, but as a potential source of systemic microbial influence with far-reaching health consequences [6-9].

The urogenital microbiome: characteristics and susceptibility to external influences

The urogenital microbiome, particularly in females, plays a vital role in maintaining reproductive and urinary tract health. Unlike the oral or gut microbiomes, the urogenital tract, especially the vaginal

environment, is typically characterized by low microbial diversity, with dominance of *Lactobacillus* species such as *L. crispatus*, *L. jensenii*, and *L. gasseri*. These bacteria contribute to host defense through the production of lactic acid, hydrogen peroxide, and bacteriocins, which maintain a low vaginal pH (<4.5) and inhibit pathogen overgrowth. This protective ecosystem is closely regulated by host factors, including estrogen levels, mucosal immunity, and metabolic status [7-9].

In males, the urogenital microbiome is less well-characterized but includes distinct microbial populations along the urethra, penis, and prostate. While not as extensively colonized as the female counterpart, the male urogenital tract can harbor commensals such as *Corynebacterium*, *Staphylococcus*, and *Streptococcus* species, as well as occasional uropathogens, particularly in cases of prostatitis or urethritis [8-10].

The urogenital microbiome is highly sensitive to external and internal influences. Antibiotic therapy, particularly broad-spectrum agents, can lead to rapid disruption of the protective microbial balance. This dysbiosis may result in overgrowth of anaerobic bacteria associated with bacterial vaginosis, or in colonization by uropathogens like *Escherichia coli*, *Gardnerella vaginalis*, and *Ureaplasma spp.*. Hormonal fluctuations during menstruation, pregnancy, and menopause also impact microbial composition and increase susceptibility to infection [8-11].

Moreover, sexual activity introduces foreign microorganisms and can alter the vaginal microbiome, potentially promoting conditions such as recurrent urinary tract infections (rUTIs). Hygiene practices, contraceptive use, and even systemic

factors like diabetes or immune suppression further modulate the urogenital microbial environment. Recent studies have also linked gut microbiome composition to urogenital health via the so-called gut-bladder and gut-vagina axes, with bacterial translocation and systemic inflammation acting as mediators [11-14].

Emerging research indicates that urogenital dysbiosis may not only contribute to local infections and inflammation but also influence systemic disease risk. For example, alterations in vaginal microbiota have been associated with adverse pregnancy outcomes, pelvic inflammatory disease, and even increased risk of sexually transmitted infections and certain cancers [10,12,13].

Biological mechanisms of oral-urogenital interaction

The interaction between the oral and urogenital microbiomes is increasingly recognized as part of a broader network of systemic microbial and immunological communication. Several biological mechanisms have been proposed to explain how disruptions in the oral microbiome, particularly through periodontitis and oral dysbiosis, can influence urogenital health and contribute to local and systemic inflammatory disease [13-15].

One central mechanism is hematogenous dissemination. During episodes of bacteremia, which may occur even during daily oral activities such as chewing or brushing, oral pathogens or their byproducts (e.g., lipopolysaccharides) can enter systemic circulation. This allows migration to distant sites, including the kidneys, bladder, prostate, and reproductive organs. For instance, *Fusobacterium nucleatum* and *Porphyromonas gingivalis*

have been detected in extraoral tissues and are known to evade immune surveillance, promoting chronic inflammation [13-16].

Another important mechanism involves systemic inflammation and immune activation. Chronic periodontitis contributes to elevated levels of circulating proinflammatory cytokines such as IL-6, IL-1 β , and TNF- α . These molecules can increase vascular permeability and promote inflammatory processes in remote organs, including the urogenital tract. This “inflammatory spillover” is thought to sensitize tissues, making them more susceptible to infection or immune-mediated damage. It is particularly relevant in conditions like interstitial cystitis, prostatitis, and recurrent urinary tract infections [14-16].

The microbial translocation theory further suggests that oral bacteria or their metabolic byproducts may alter mucosal immunity in distant sites, potentially influencing the integrity of urogenital epithelial barriers. This can impair local microbial homeostasis and promote colonization by opportunistic or pathogenic organisms [14-16].

An emerging area of interest is the oral-gut-urogenital axis, which considers that disturbances in the oral microbiome may also influence the gut microbiota, which in turn can modulate urogenital health. The gut microbiome acts as an immunological hub, and its interaction with the bladder and reproductive tract through metabolic and inflammatory signaling may mediate cross-site microbial effects [14-17].

Additionally, shared immunological pathways, including toll-like receptor signaling, neutrophil activation, and T-cell polarization, may underlie parallel immune responses in the oral and urogenital

mucosae. Dysregulation in one mucosal site may thus propagate similar immune dysfunction in another, especially in genetically or metabolically predisposed individuals [15-17].

Collectively, these mechanisms underscore the oral cavity's potential as a source of systemic microbial influence. Understanding these biological pathways highlights the importance of oral health in maintaining systemic and urogenital integrity and suggests potential therapeutic targets across interconnected microbial ecosystems [15-18].

Periodontal disease and its association with urological pathologies

Periodontal disease (PD), a chronic inflammatory condition resulting from microbial dysbiosis in the oral cavity, has traditionally been associated with localized destruction of soft and hard tissues surrounding the teeth. However, mounting evidence links PD with a wide spectrum of systemic conditions, including urological disorders. This association is likely mediated by systemic inflammation, immune modulation, and the dissemination of pathogenic oral bacteria or their toxins to distant anatomical sites [17-19].

One of the most studied connections is between PD and lower urinary tract symptoms (LUTS). Clinical studies have shown a higher prevalence and severity of LUTS, such as urinary urgency, frequency, and weak stream, in individuals with advanced periodontitis. These symptoms are often attributed to chronic inflammation affecting the bladder or prostate, suggesting a shared inflammatory burden driven by circulating cytokines like IL-6 and TNF- α originating from oral infections [9,16,18].

Similarly, emerging data suggest that chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS) may also be linked to poor oral health. The systemic inflammatory milieu generated by PD can exacerbate localized inflammation in the prostate, potentially influencing pain, urinary dysfunction, and quality of life. Oral pathogens like *Fusobacterium nucleatum* and *P. gingivalis* have been detected in prostatic secretions and may directly colonize urogenital tissues [16-18].

Furthermore, PD has been indirectly associated with recurrent urinary tract infections (rUTIs). Chronic oral inflammation may compromise mucosal immunity and contribute to dysbiosis in the vaginal or urinary microbiomes, thereby reducing colonization resistance against uropathogens [14,17-19].

These findings underscore the importance of considering oral health as a modifiable risk factor in urology. While causality has not been definitively established, the consistent correlations observed in epidemiological and clinical studies warrant further investigation. Integrating periodontal screening and management into comprehensive urological care protocols could represent a novel preventive strategy for mitigating systemic inflammatory burden and reducing the risk of chronic urological conditions [17-19].

Therapeutic implications and future research directions

The emerging evidence linking the oral microbiome and periodontal disease with urogenital pathologies offers promising avenues for interdisciplinary prevention and treatment strategies. Understanding these microbial and immunological connections provides

clinicians and researchers with a rationale to explore oral health as both a biomarker and modifiable risk factor in managing systemic inflammatory and infectious urogenital conditions [18-20].

One immediate implication is the integration of oral health assessment into routine urological care, especially in patients with chronic lower urinary tract symptoms, recurrent urinary tract infections (rUTIs), or prostatitis. Identifying and managing periodontal inflammation may help reduce systemic inflammatory load and potentially alleviate associated urological symptoms. Similarly, dentists should be aware of the extraoral consequences of periodontitis, including potential urological impacts, to enhance patient education and interdisciplinary referrals [18-21].

Probiotics and prebiotics have shown promise in modulating both oral and urogenital microbiomes. Administering specific strains of *Lactobacillus* or *Bifidobacterium* may help restore microbial balance, enhance mucosal immunity, and reduce pathogen colonization. Oral probiotic lozenges or vaginal suppositories are being explored as adjuncts in managing bacterial vaginosis, rUTIs, and periodontitis. Although clinical data are still limited, these biologically based interventions are gaining traction as low-risk, host-friendly options [15,18-21].

Recent advances in materials science and regenerative medicine offer promising directions for microbiome-targeted therapies in systemic conditions involving oral and urogenital inflammation. Developments in hydrogel technology, bioactive essential oils, and biodegradable polymers have expanded the therapeutic arsenal for tissue repair and antimicrobial

delivery. Innovations in diagnostic tools, including AI-enhanced imaging and CBCT, can improve early detection of inflammatory foci. Furthermore, 3D bioprinting and the integration of gold-based biomaterials demonstrate translational potential in prosthetic and surgical applications. Emerging phytotherapeutics and selective antimicrobial agents may support microbiome modulation without disrupting mucosal equilibrium. A deeper understanding of microbial interactions and systemic pathways is necessary to design minimally invasive, personalized strategies that restore homeostasis and prevent cross-site inflammation [22-33].

Targeted antimicrobial therapy also warrants reconsideration. Overuse of systemic antibiotics for dental or urological conditions can inadvertently disturb the microbiome in both niches. There is a growing need for narrow-spectrum, locally acting antimicrobials that minimize collateral damage to commensals and reduce the risk of resistance development. Photodynamic therapy, antimicrobial peptides, and enzymatic biofilm disruptors are emerging as alternatives to traditional antibiotics [18,34,35].

From a research perspective, longitudinal studies are essential to determine causal links and assess temporal dynamics between changes in the oral microbiome and urogenital disease progression. Advanced sequencing technologies, such as metagenomics and metabolomics, can help characterize microbial communication networks and identify key functional pathways involved in cross-site inflammation. Additionally,

mechanistic studies using animal models and in vitro organoid systems could elucidate the exact biological routes, hematogenous, lymphatic, or neural, by which oral dysbiosis affects distant mucosal sites [34,35].

Conclusion

The oral microbiome plays a critical and often underestimated role in systemic health. Its interaction with the urogenital tract reflects a complex network of microbial and immune communications that extend beyond local infection or inflammation. Accumulating evidence indicates that oral dysbiosis, particularly in the context of periodontal disease, may contribute to urological conditions such as LUTS, prostatitis, and recurrent UTIs through systemic inflammation, microbial dissemination, and immune modulation.

Recognizing the oral-urogenital axis opens new perspectives for interdisciplinary prevention, early diagnosis, and integrated treatment strategies. Maintaining oral health may not only preserve dentition and quality of life but also reduce the risk of chronic inflammatory processes elsewhere in the body. As microbiome science evolves, clinical approaches must expand to consider the interconnectedness of mucosal ecosystems.

Future research should focus on longitudinal, mechanistic, and interventional studies to confirm causality and identify therapeutic targets. Ultimately, oral health should be viewed as a foundational element in comprehensive, systemic healthcare, impacting not just smiles but organs far beyond the mouth.

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