BULLETIN OF STOMATOLOGY AND MAXILLOFACIAL SURGERY
Volume 21, Issue 7

DOI: 10.58240/1829006X-2025.21.7-68

REVIEW ARTICLE

# PERIODONTITIS AND UROLOGIC CANCERS: A NARRATIVE REVIEW OF POTENTIAL RISK CONNECTIONS

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***Received:*** *Jun 15. 2025;* ***Accepted:*** *Jul 19, 2025;* ***Published:*** *Aug 5,2025*

Urologic cancers, including prostate, bladder, and kidney cancers, represent a major global health burden with complex and multifactorial etiologies. An infectious inflammatory illness called periodontitis has been increasingly implicated in systemic conditions like cancer Mechanisms involving oxidative stress, disruption of the immune system, systemic inflammation, and body microbiome modulation are likely accountable for these correlations. We detail recently established correlations among urologic cancers and periodontitis, with emphasis upon systemic inflammation as an important common pathway, in this review. The correlation between oral diseases and cancer risk suggests multidisciplinary research potentially could define these correlations and provide focused therapies Clinically, periodontal evaluation inclusion in general cancer risk profiling potentially could deliver new directions in patient care prevention. A focus upon systemic as well as oral health holistically

***Keywords*:** Periodontitis, urologic cancers, systemic inflammation, immune dysregulation, prostate cancer, kidney cancer, bladder cancer, risk factors

## 1. INTRODUCTION

Urologic cancers, encompassing carcinomas of the prostate, upper urinary tract (urothelial carcinoma), bladder, kidneys, and testis, rank among the most prevalent ones globally.[1,2] Prostate cancer (PCa), in particular, has the highest incidence among men in industrialized nations and causes significant cancer deaths.[3,4] Bladder and kidney cancer are also among the most significant cancer statistics worldwide, indicating an emerging healthcare issue.[5,6] Numerous causes have been implicated in the higher incidence of these tumors, such as rising age, cigarette smoking, and hereditary genetic predisposition.[7-11] Chronic systemic inflammation has been found, more recently, as an added contributory factor.[12]

Truly, prolonged inflammation has been known to be integral not only in the onset of tumors, but even in the course of their evolution over time.[13] The chronic inflammatory conditions are known to create a permissive microenvironment for the tumor with the generation of reactive oxygen species, release of pro-inflammatory cytokines, along with induction of DNA damage.[13,14-16] These improvements could possibly drive the generation of genetic mutations, provide help in angiogenesis, along with inducing immune evasion by the tumour.[17-19] Systemic inflammation has also been associated with immune reaction suppression against tumours, thereby facilitating metastasis as well as compromising the effectiveness of therapeutic interventions.[13]

This highlights the broader role of inflammation, not just as a marker of cancer but as a modifiable factor in its treatment.

Periodontitis, an inflammatory chronic disease affecting primarily 15% the supporting tissues around the teeth, is common, with an appreciable percentage among populations around the globe. It's an illness with bacterial infection and immune reactions, potentially spreading its effects beyond the mouth.[20-22]

It has been correlated with systemic diseases like diabetes and cardiovascular disease, reflecting a general inflammatory and immunologic impact.[23] In response, enhanced prosthodontic rehabilitation has uncovered how systemic well-being is influenced by oral management procedures.[24,25] For example, telescopic overdenture rehabilitation in syndromic patients demonstrated the importance of functional oral restoration in improving over-all health outcomes.[26]

Recent studies indicate potential periodontitis-cancer associations, including those involving the urologic system. Systemic inflammation induced during periodontal diseases could provide a stimulus toward carcinogenesis, in part, by modifying pathways crucial in immune regulation as well as chronic tissue destruction.[27-31] Observational studies and genome-wide analyses have identified correlations, like higher risk for prostate and renal cancer among patients with periodontitis.[32] Nonetheless, these are still susceptible to the effects of confounding, including common environment and lifestyle effects. It attempts to integrate current evidence regarding likely connections between periodontitis and urologic cancers hoping to delineate the bidirectional interaction between oral health and systemic oncologic risk, leading the way toward directional future research and therapy.

It aims to synthesize existing evidence about future links between periodontitis and urologic cancers while working toward defining the bidirectional interaction between systemic oncologic risk and oral health, enabling directional future research investigation and intervention.

## 2. MATERIALS AND METHODS

We conducted a narrative review in accordance with this question's scope, an examination that takes into account the potential interrelationships existing between periodontitis and urologic cancers. The search was conducted with PubMed and Scopus, in addition to articles made available from inception until November 2024. The search strategy consisted of keywords relating to the subjects of urologic cancers and periodontitis, such as "periodontal disease, periodontitis," "gum disease," "prostate cancer," "bladder cancer," "urothelial cancer", "kidney cancer," "chronic inflammation," and "oral-systemic health." Boolean search markers (AND, OR) were employed in narrowing down the search. For instance, groupings such as ("periodontal disease" OR "gum disease") AND ("prostate cancer" OR "bladder cancer") were utilized in an attempt to narrow down the search.

## 3 RESULTS

### 2.1 Pathophysiological Links Between Periodontitis and Urologic Cancer

Periodontitis, an inflammatory chronic illness involving basically the ligament and alveolar bone, presents systemic manifestations besides oral diseases.[21] It causes an everlasting inflammatory process, where pro-inflammatory cytokines such as interleukin-6, tumour necrosis factor-alpha, and C-reactive protein are produced. These mediators are key players in systemic inflammatory diseases, recently rising as significant inducers of cancer onset.[33] Prostate, urothelial, and kidney cancer, among others, are urologic tumours that have been perpetually associated with systemic inflammatory diseases, including periodontal diseases. In the specific case of prostate cancer, elevated inflammatory cytokine levels and immune imbalances could promote the tumour's progress, besides drug resistance. In urothelial cancer, prolonged exposure to inflammatory mediators along with immune disruption could lead to urothelial conversion with recurrence.[2,10,34] In renal cell cancer, systemic pro-inflammatory profiling, an almost universal feature among patients with advanced periodontitis, has correlated with aggressive behavior with adverse prognosis.[35-38]

Interaction among periodontitis and cancer onset involves several mechanisms. Firstly, systemic dissemination of periodontal pathogens such as *Porphyromonas gingivalis* and *Fusobacterium nucleatum*, was determined to initiate chronic inflammation as well as immune deregulation. These pathogens release virulence factors with the capability of initiating DNA damage, cellular transformation, as well as inhibition of apoptosis. Moreover, the creation of reactive oxygen species (ROS) within periodontal tissues with inflammation elevates the level of oxidative stress with the capacity to initiate gene mutations as well as provide support for angiogenesis—a cancer onset signature.[13, 27, 39] Diagnostic imaging modalities like optical coherence tomography have been useful in the detection of early mucosal transformations, including autoimmune conditions like pemphigus vulgaris, highlighting the functional capability of advanced oral diagnostics in the detection of pre-cancerous/o inflammatory transformations.[40] Besides, systemic inflammation in tends to suppress anti-tumor immune responses.[32, 41]

The presence of elevated neutrophils and platelets, with low lymphocyte function, reflected by markers such as systemic immune-inflammation index (SII), implies an immunosuppressive tumour-favourable microenvironment conducive to tumour progress and metastasis. Inflammation also destroys homeostasis of the tumour microenvironment, facilitating immune evasion of the tumour as well as increased metastatic capacity.[33]

Recent research also suggests that the oral microbiome plays a role in cancer pathophysiology. Dysbiosis, with overgrowth of pathogenic organisms with losses of beneficial species, could impact systemic inflammatory pathways.[42] For example, activation of toll-like receptors could be induced by products of bacteria like lipopolysaccharides, thereby stimulating increased inflammation with an overall pro-carcinogenic microenvironment.[13, 27, 43] These research studies implicate the bidirectional periodontitis-systemic diseases interaction, in this example, with cancer, and emphasize the need to control chronic inflammatory conditions as a means to limit oncologic risk.

Furthermore, the functional evaluation of temporomandibular disorders among orthodontic patients showed that systemic inflammatory mechanisms could potentially interact with occlusal and musculoskeletal variables, hence reinforcing the importance of oral functional disorders in wider systemic illness frameworks. [44]

### 2.2 Evidence and Contradictions in the Association Between Periodontitis and Urologic Cancers

Periodontitis and urologic cancers have had an association that has been rising in interest, albeit the evidence remains patchy and sometimes contradictory. Key to this conundrum persists the hypothesis that systemic inflammation, common to both periodontitis and cancer, represents an underlying unifying hypothesis. Strong evidence acquired in this fashion, such as in the meta-analysis conducted by Wei et al. (2021), suggests an overt association between periodontal infection and risk of developing prostate cancer, with an estimated relative risk of 1.17 (95% CI: 1.07–1.27). These results also offer confirmation, once again, of the hypothesis that chronic inflammation plays an important role in determining a microenvironment supportive of tumors. All the same, this conclusion remains qualified due to the heterogeneity in the studies, as well as confounding variable adjustment.[45]

In contrast with the overall pattern, Li et al. (2024) employed Mendelian randomization methodology to evaluate causal associations at the gene level and discovered no considerable associations for the majority of urologic cancers, including prostate cancer. The inquiry, however, under their investigation indicated the prospective hereditary connection between periodontitis and renal cancer (OR = 1.287; 95% CI = 1.04–1.59). The discrepancy presents relevant questions about the extent to which hereditary propensities, rather than systemic inflammatory mechanisms, lie beneath these associations. Absence of reverse causality—urologic type cancers imposing upon periodontitis—one favors, moreover, the hypothesis that such an association, should it exist, likely mediates with common systemic pathways, as an alternative to direct action.[46] Providing an additional voice, Michaud et al. (2016) compared Health Professionals Follow-Up Study data, with the use of male never-smokers in an effort to control confounding secondary to tobacco use. They identified an overall moderate risk increase among people with periodontitis for cancer (HR = 1.13; 95% CI = 1.01–1.27), with stronger associations with smoking-related cancers such as bladder cancer. Extremity in periodontitis with extreme tooth loss presented with the strongest risks. These studies provide evidential support for the hypothesis that risk for cancer could be modified with the severity of periodontal disease but also cause concern regarding the difficulty in separating periodontal disease's effect with confounding lifestyle/health effects.[47] Overlaying this problem, Ma et al. (2020) provided meta-analysis that reinforced an important periodontitis-prostate cancer association (HR
= 1.20; 95% CI = 1.09–1.31) but offered inconclusive evidence for kidney cancer (HR = 1.30; 95% CI = 0.96–1.76). These differing conclusions among studies across kidney cancer could be brought about with definition heterogeneity among studies, interpopulation heterogeneity, or lack of adequate statistical power. Greater multicenter studies were also suggested by Ma et al. as one method to bridge these gaps.[48]

These distinctions probably both reflect methodologic distinctions as well as the multifactorial, difficult interaction between periodontitis and urologic cancers. Although the inflammatory hypothesis remains a reasonable unifying hypothesis, evidence drawn from epidemiologic as well as genetic studies impeaches its simplicity, contending instead that an understanding that interlaces genetic with environmental ones will prove fruitful in the future.[46] These studies collectively remind us that while the association between periodontitis and urologic cancers has some, mixed evidence in support, its nature—causal, associative, both, or neither— remains an open question, necessitating stringent, interdisciplinary research to shed light upon.

## 3.Future Directions in Research and Clinical Implications

In building upon existing evidence, future studies should consider the methodological variation and gaps identified in previous studies. A major priority area remains standardized definition both of periodontal disease severity and cancer outcome.

A great deal of the variation in results can be accounted for by differences in how these conditions are defined and expressed across studies. For example, standardized classification of advanced periodontitis and its systemic effects could narrow risk estimates and improve comparison across studies. In addition, examining the putative biological mechanisms underlying these associations should be given a research priority. Bacteria like *Porphyromonas gingivalis* and *Fusobacterium nucleatum,* both with the ability to induce systemic inflammation, could contribute to initiating tumors by modifying immune reactions or causing direct genomic damage. Worthy of investigation, therefore, are integrated studies incorporating genomics, microbiomics, and metabolomics.

Future developments in this area will also involve active cross-disciplinary practice between the urologist, oncologist, periodontist, and geneticist, providing an enriched paradigm both in research and practice. Virtual communication tools are also changing the manner in patients and caregivers are accessing information.[49] Investigations examining YouTube content searching for recurrent oral ulcerations in children demonstrated the influence of social media upon patient knowledge, with the importance of incorporating trustworthy online re-sources into prevention and educational plans for systemic disease management.[50] These collaborations could facilitate large-scale studies that combine epidemiologic data with biological understanding, along with an integrated description of the interplay between oral and systemic health. Clinically, the rationale remains firmly in support of periodontal evaluation inclusion as part of overall cancer risk evaluation, with special mention regarding population at risk.

Also, interventional studies that provide definitive information about whether periodontitis remediation decreases cancer risk, answering the question whether periodontitis remediation has practical value as an alterable risk factor, will be essential. Control of confounding variables, like smoking and social class, with higher- order statistical method or Mendelian randomization will be instrumental in determining causal associations. These new discoveries will not only furnish proof connecting periodontitis with urologic cancers but direct practical avenues toward prevention as well as treatment. cancer risk evaluation, with special mention regarding population at risk. Also, interventional studies that provide definitive information about whether periodontitis remediation decreases cancer risk, cancer risk evaluation, with special mention regarding population at risk.

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## 4.CONCLUSIONS

It brings into focus the close interplay between periodontitis and urologic cancers with systemic inflammation emerging as a likely shared pathway. While observational studies indicate strong associations, notably with prostate as well as with kidney cancer, the hereditary evidence suggests the association to be appreciably less linear with significant roles both for hereditary predisposition as well as with systemic mechanisms. The discrepancy evident among the existing studies underlines the need to devise standardized criteria and promote interdisciplinary research conjoining the clinical, molecular, as well as epidemiologic viewpoints.

From a clinical standpoint, periodontal health evaluation within cancer risk profiles could be helpful primarily among populations in the highest risk categories. Owing to its extant modifiability, periodontitis treatment could potentially create new avenues toward cancer prevention. In order to advance this area, it stands to reason that large-scale, multidisciplinary studies should be conducted, which could close current knowledge gaps but also give an in-depth account about the interrelationship between oral and body health.

## DECLARATIONS

**Funding:** None

**Acknowledgments:** None

**Conflicts of Interest:** None

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