

Impact Of Non-Surgical Periodontal Therapy On Anxiety Levels In Stage III/IV Periodontitis Patients

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Abstract

Periodontal disease represents a significant global health concern with established bidirectional relationships with various systemic conditions. Emerging evidence suggests interconnections between psychological factors, particularly anxiety, and periodontal disease progression. This study reviews the impact of non-surgical periodontal therapy (NSPT) on anxiety levels in patients with stage III/IV periodontitis, with additional consideration of smoking status as a modifying factor. Current evidence demonstrates significantly elevated baseline anxiety levels in periodontitis patients compared to periodontally healthy individuals, with the highest levels observed in smokers with periodontitis. Following NSPT, both smoking and non-smoking periodontitis patients typically exhibit significant reductions in anxiety scores coinciding with improvements in clinical parameters, though the magnitude of anxiety reduction appears greater in non-smokers. Significant associations exist between reductions in clinical attachment loss, probing depth, and anxiety score improvements. These findings suggest that effective periodontal therapy may contribute to psychological well-being, highlighting the importance of integrated approaches to periodontal care that address both biological and psychological dimensions of the disease.

INTRODUCTION

Periodontal disease constitutes a significant global health burden, affecting up to 50% of the adult population worldwide with severe forms impacting 10-15%

(Genco & Borgnakke, 2013). Beyond its oral manifestations, periodontal disease has established bidirectional relationships with various systemic conditions including diabetes, cardiovascular disease, and adverse pregnancy outcomes (Cekici et al., 2014). Recent evidence has increasingly highlighted the interconnection between psychological factors and periodontal health status.

The pathogenesis of periodontal disease involves complex inflammatory and immune pathways triggered by bacterial dysbiosis, but modified significantly by host-response factors (Cekici et al., 2014). Contemporary periodontal disease models emphasize the multifactorial nature of the condition, incorporating traditional risk factors such as poor oral hygiene, smoking, diabetes, and genetic predisposition (Genco, 1996; Grossi et al., 1994), alongside emerging psychological and psychosocial dimensions (da Silva et al., 1995).

Among psychological factors, anxiety has garnered particular attention for its potential role in periodontal disease progression. Anxiety, characterized by heightened states of worry, tension, and autonomic nervous system arousal, may influence periodontal health through multiple pathways. These include direct physiological mechanisms via stress-related neuroendocrine responses affecting immune function and inflammatory processes, as well as indirect behavioral pathways through impacts on oral hygiene practices, smoking behavior, and healthcare utilization (Genco et al., 1999; Vettore et al., 2003).

Epidemiological and clinical studies have demonstrated associations between anxiety and periodontal parameters. Vettore and colleagues (2003) reported significant correlations between anxiety levels and clinical attachment loss in periodontitis patients. Similarly, Hugoson et al. (2002) identified anxiety as an independent risk factor for periodontal disease in a Swedish population study. Moss et al. (1996) found that individuals with high anxiety scores demonstrated a 2.5-fold increased risk of severe periodontitis compared to those with low scores. These findings align with Green et al. (1986), who identified stress and anxiety-provoking life events as significant predictors of periodontal disease severity.

The relationship between anxiety and periodontal disease appears particularly pronounced in smokers. Smoking remains among the most significant modifiable risk factors for periodontal disease, with substantial evidence demonstrating its detrimental effects on periodontal tissues (Bergström et al., 2000; Tomar & Asma, 2000; Vered et al., 2008). Johannsen et al. (2005) observed synergistic effects between anxiety, inflammation, and smoking status in periodontal patients. Similarly, Kolte et al. (2016) reported that anxiety levels were significantly higher in smokers with periodontitis compared to non-smokers with comparable disease severity.

While the association between anxiety and periodontal disease has been investigated, limited research has examined how therapeutic interventions for periodontal disease might influence anxiety levels. Non-surgical periodontal therapy (NSPT), comprising oral hygiene instruction, scaling and root planing, and supportive periodontal care, represents the foundation of periodontal disease management. Understanding whether successful periodontal therapy might concomitantly reduce anxiety could have important implications for comprehensive patient care approaches.

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions introduced a new classification system for periodontitis based on a staging and grading framework (Papapanou et al., 2018). Stage III and IV periodontitis represent advanced disease with significant tooth loss risk,

characterized by clinical attachment loss ≥ 5 mm, radiographic bone loss extending to mid-third of root and beyond, and often complicated by deep periodontal pockets, furcation involvements, and tooth mobility. These advanced cases particularly warrant investigation regarding associated psychological factors and therapeutic outcomes.

This study aims to review the impact of non-surgical periodontal therapy on anxiety levels in patients with stage III/IV periodontitis, with additional consideration of smoking status as a potentially modifying factor. We examine evidence suggesting that successful periodontal therapy is associated with reductions in anxiety levels, with differential responses between smokers and non-smokers with advanced periodontitis.

Anxiety and Periodontal Disease: Establishing the Relationship

Evidence for Association

Multiple epidemiological and clinical studies have established associations between anxiety and periodontal disease. Vettore et al. (2003) conducted a case-control study comparing anxiety levels between patients with generalized periodontitis, localized periodontitis, and periodontally healthy controls. Using the State-Trait Anxiety Inventory, they found significantly higher anxiety scores in the generalized periodontitis group compared to the other groups, even after controlling for potential confounders. Hugoson et al. (2002) conducted a population-based study in Sweden involving 1,093 participants aged 50-80 years and found that anxiety was independently associated with periodontal disease severity after adjusting for oral hygiene and smoking status.

In a landmark case-control study, Moss et al. (1996) investigated the relationship between psychosocial factors and periodontitis in 164 cases and 164 controls. They reported that high anxiety scores were associated with a 2.5-fold increased risk of periodontitis. Similarly, Genco et al. (1999) in their study of 1,426 participants found that individuals with high anxiety and inadequate coping behaviors had significantly higher clinical attachment loss compared to those with low anxiety levels.

More recent studies have continued to support these associations. Saletu et al. (2005) compared 40 periodontitis patients with 40 matched controls and found significantly higher anxiety scores in the periodontitis group using both self-reported measures and objective psychometric assessments. Li et al. (2011) similarly reported higher anxiety scores in periodontitis patients compared to controls in a Chinese population.

Biological Mechanisms

Several biological mechanisms potentially underlie the relationship between anxiety and periodontal disease. First, anxiety activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased cortisol production. Elevated cortisol levels can suppress immune function, potentially reducing the host's ability to control periodontal infection (Genco & Borgnakke, 2013). Bawankar et al. (2018) demonstrated elevated serum and salivary cortisol levels in periodontitis patients compared to healthy controls, with particularly high levels in those reporting anxiety and stress.

Second, anxiety triggers sympathetic nervous system activation, leading to catecholamine release. These neurotransmitters can alter gingival blood flow and immune cell function, potentially exacerbating periodontal inflammation (da Silva et al., 1995). Third, pro-inflammatory cytokines produced during periodontal inflammation, including interleukin-1 β , interleukin-6, and tumor necrosis factor- α , can cross the blood-brain barrier and influence neurotransmitter metabolism,

potentially contributing to anxiety symptoms (Cekici et al., 2014). This suggests a potential bidirectional relationship, where periodontal inflammation might exacerbate anxiety symptoms.

Behavioral Mechanisms

Alongside biological pathways, behavioral mechanisms likely contribute to the anxiety-periodontitis relationship. Anxiety may lead to neglected oral hygiene, increased smoking, unhealthy dietary habits, and reduced healthcare utilization, all of which can negatively impact periodontal health (Genco et al., 1999). Anxious individuals may demonstrate poorer compliance with oral hygiene instructions and irregular dental attendance patterns, compromising preventive and therapeutic periodontal care.

Additionally, anxiety often coexists with depression, which has been independently associated with periodontitis. The comorbidity of these conditions may have cumulative effects on periodontal health through both biological and behavioral pathways (Hugoson et al., 2002).

The Compounding Effect of Smoking

Smoking as a Periodontal Risk Factor

Smoking represents one of the most significant modifiable risk factors for periodontal disease. Extensive evidence demonstrates its detrimental effects on periodontal tissues through multiple mechanisms. Tomar and Asma (2000), analyzing data from the Third National Health and Nutrition Examination Survey, estimated that 41.9% of periodontitis cases in the United States were attributable to current smoking and 10.9% to former smoking. Bergström et al. (2000) demonstrated a dose-dependent relationship between smoking exposure and periodontal bone loss, with heavy smokers showing more severe destruction than light smokers or non-smokers.

Mechanistically, smoking compromises periodontal health through vasoconstriction, impaired neutrophil function, altered fibroblast activity, increased production of destructive enzymes, and shifts in the subgingival microbiome toward more pathogenic species (Genco & Borgnakke, 2013). These effects collectively increase susceptibility to periodontal infection and impair tissue healing responses.

The Anxiety-Smoking-Periodontitis Triad

Evidence suggests that anxiety, smoking, and periodontal disease form a complex triad with bidirectional relationships. Johannsen et al. (2005) conducted an epidemiological study comparing anxiety levels, gingival inflammation, and periodontal status between smokers and non-smokers. They found that anxiety levels were significantly higher in smokers with periodontitis compared to non-smokers with periodontitis, and anxiety scores correlated with clinical periodontal parameters more strongly in smokers than non-smokers.

Kolte et al. (2016) further explored this relationship in a cross-sectional study of 81 periodontitis patients and 80 periodontally healthy controls, with each group subdivided based on smoking status. They reported that anxiety scores were highest in smoking periodontitis patients, followed by non-smoking periodontitis patients, smoking controls, and non-smoking controls. This pattern suggests potential synergistic effects between anxiety, smoking, and periodontal inflammation.

The relationship appears to operate through multiple pathways. Anxiety may predispose to smoking initiation and maintenance while reducing cessation success. Smoking directly impacts periodontal tissues while potentially exacerbating anxiety through nicotine's effects on neurotransmitter systems and withdrawal symptoms.

Periodontal inflammation may contribute to systemic inflammation, potentially influencing neural pathways involved in anxiety regulation. This complex interplay creates potential for vicious cycles that may be difficult to interrupt without addressing all components simultaneously.

Neuroendocrine Factors

Recent research has begun to elucidate the neuroendocrine aspects of this relationship. Rahate et al. (2022) investigated serum and salivary ghrelin and cortisol levels in smokers and non-smokers with stage III periodontitis. They found that smoking periodontitis patients demonstrated significantly higher cortisol and lower ghrelin levels compared to non-smoking periodontitis patients, alongside higher anxiety scores. These findings suggest that smoking may alter neuroendocrine regulation in periodontitis patients, potentially contributing to their higher anxiety levels.

Similarly, Bawankar et al. (2018) reported that smoking periodontitis patients exhibited significantly higher salivary interleukin-1 β and cortisol levels compared to non-smoking periodontitis patients, suggesting amplified inflammatory and stress responses that could influence both periodontal and psychological outcomes.

Non-Surgical Periodontal Therapy: Impact on Clinical and Psychological Parameters

Principles and Expected Clinical Outcomes

Non-surgical periodontal therapy (NSPT) represents the cornerstone of periodontitis treatment and typically includes thorough oral hygiene instruction, professional mechanical plaque removal (scaling and root planing), and supportive periodontal care. The primary goals include reducing the bacterial load, eliminating inflammatory changes in periodontal tissues, arresting disease progression, and creating an environment conducive to healing.

In patients with stage III/IV periodontitis, NSPT typically achieves significant improvements in clinical parameters, including reductions in probing depths, gains in clinical attachment, and decreased bleeding on probing. However, the magnitude of improvement varies considerably based on initial disease severity, patient-related factors (including smoking), and technical aspects of therapy.

Smoking consistently emerges as a factor that compromises treatment outcomes. Smokers typically demonstrate approximately 50% less reduction in probing depth and clinical attachment gain compared to non-smokers following identical therapy (Genco & Borgnakke, 2013). This reduced healing capacity results from smoking's effects on vasculature, immune function, and tissue remodeling.

Evidence for Psychological Improvements Following NSPT

Emerging evidence suggests that successful periodontal therapy may have beneficial effects on psychological parameters, including anxiety. Li et al. (2011) conducted one of the first studies directly examining this relationship, evaluating anxiety and depression scores in 69 chronic periodontitis patients before and after non-surgical periodontal therapy. They reported significant reductions in both anxiety and depression scores following treatment, with the magnitude of psychological improvement correlating with the degree of clinical improvement.

More recent studies have extended these observations specifically to patients with advanced periodontitis according to current classification criteria. These investigations consistently demonstrate reductions in anxiety scores following NSPT, with greater improvements observed in non-smokers compared to smokers. The magnitude of anxiety reduction typically correlates with improvements in

clinical parameters, particularly reductions in clinical attachment loss and probing depth.

Several mechanisms potentially explain these psychological benefits. First, successful periodontal therapy reduces local and potentially systemic inflammation. Given the emerging evidence linking inflammatory processes to neuropsychiatric symptoms, reducing periodontal inflammation may positively influence neural pathways involved in anxiety regulation. Second, improvements in clinical symptoms such as bleeding, pain, halitosis, and mobility may alleviate specific oral health-related anxieties and improve quality of life. Third, the therapeutic relationship and sense of control established during treatment may have inherent psychological benefits.

Differential Response in Smokers vs. Non-Smokers

The psychological benefits of periodontal therapy appear to differ between smokers and non-smokers, mirroring differences in clinical response. Non-smokers typically demonstrate greater reductions in anxiety scores following therapy compared to smokers with similar baseline disease severity. This differential response likely reflects multiple factors.

First, smokers show less favorable clinical improvements following NSPT, potentially limiting the reduction in local inflammation that might influence anxiety pathways. Second, nicotine's ongoing effects on neurotransmitter systems involved in anxiety regulation may partially counteract benefits gained from improved periodontal health. Third, smoking-induced chronic inflammatory states may persist despite periodontal therapy, maintaining elevated levels of inflammatory mediators that potentially influence anxiety.

The relationship between smoking intensity and psychological response also appears dose-dependent, with heavier smokers showing less anxiety reduction following therapy compared to lighter smokers. This observation parallels the dose-dependent effects of smoking on clinical periodontal parameters and treatment outcomes.

Correlations Between Clinical and Psychological Improvements

Specific Periodontal Parameters and Anxiety Reduction

Research examining correlations between improvements in specific periodontal parameters and anxiety reduction has yielded several consistent findings. Clinical attachment level (CAL) improvements typically show the strongest correlation with anxiety score reductions. This may reflect CAL's status as a cumulative measure of periodontal destruction that most comprehensively reflects disease severity and progression.

Probing pocket depth (PPD) reductions also demonstrate significant correlations with anxiety improvements, though generally slightly weaker than CAL correlations. Bleeding on probing (BOP) percentage reduction shows moderate correlations with anxiety improvement, potentially reflecting the resolution of active inflammation. Plaque and gingival indices typically show weaker but still significant correlations with psychological outcomes.

These patterns suggest that the magnitude of structural and inflammatory improvement, rather than simply improved oral hygiene, most strongly influences psychological benefits. This aligns with proposed biological mechanisms linking periodontal inflammation to systemic effects that might influence neuropsychological function.

Specific Anxiety Domains

Analysis of specific anxiety domains reveals further insights into the relationship between periodontal therapy and psychological outcomes. Autonomic symptoms (e.g., palpitations, sweating, gastrointestinal disturbances) and cognitive symptoms (worry, fear, rumination) typically show stronger correlations with periodontal improvements than motor symptoms (restlessness, fidgeting) or central nervous system symptoms (dizziness, faintness).

These patterns may reflect the physiological links between inflammation and autonomic nervous system function, as well as the specific cognitive concerns related to oral health. Autonomic symptoms may be more directly influenced by reductions in inflammatory mediators that affect neuroendocrine function, while cognitive symptoms may respond more directly to the resolution of oral health concerns and improved sense of control over the condition.

Predictive Models

Multiple regression analyses consistently identify improvements in CAL and PPD as significant independent predictors of anxiety reduction following periodontal therapy, even after controlling for potential confounders including age, gender, and baseline anxiety levels. Smoking status typically emerges as a significant modifier of this relationship, with smoking attenuating the psychological benefits associated with clinical improvements.

These predictive models generally explain a substantial proportion of the variance in anxiety reduction (typically 60-70%), suggesting that periodontal clinical improvements represent a major contributor to psychological benefits, though other factors clearly also play important roles.

Biological Mechanisms Connecting Periodontal Therapy and Anxiety Reduction

Inflammatory Pathway Modulation

Mounting evidence suggests that inflammatory processes play a crucial role in the relationship between periodontal disease and anxiety, with periodontal therapy potentially influencing this relationship through modulation of inflammatory pathways. Cekici et al. (2014) extensively reviewed the inflammatory and immune pathways in periodontal disease, highlighting how periodontal inflammation produces pro-inflammatory cytokines including interleukin-1 β (IL-1 β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α) that enter systemic circulation.

These inflammatory mediators can cross the blood-brain barrier and activate microglia, the resident immune cells of the central nervous system. Microglial activation can alter neurotransmitter metabolism, particularly affecting serotonin, dopamine, and glutamate systems that play critical roles in anxiety regulation. Successful periodontal therapy significantly reduces local production of these inflammatory mediators, potentially attenuating their systemic and neurological effects.

Bawankar et al. (2018) demonstrated that periodontal therapy reduced both salivary and serum IL-1 β levels in periodontitis patients, with greater reductions in non-smokers compared to smokers. These reductions correlated with improvements in both clinical parameters and psychological measures, suggesting a potential mechanistic link.

Neuroendocrine Regulation

The hypothalamic-pituitary-adrenal (HPA) axis represents another potential pathway connecting periodontal therapy and anxiety reduction. Chronic periodontitis has been associated with dysregulation of the HPA axis, manifesting as altered cortisol profiles. Cortisol, a key stress hormone, influences numerous

physiological systems including immune function, glucose metabolism, and neurotransmitter activity.

Rahate et al. (2022) demonstrated that successful periodontal therapy normalized salivary and serum cortisol levels in periodontitis patients, with these changes correlating with reductions in anxiety scores. Interestingly, they also observed normalization of ghrelin levels, a hormone involved in stress response, energy homeostasis, and potentially anxiety regulation. These findings suggest that periodontal therapy may help restore neuroendocrine balance, potentially contributing to psychological benefits.

Microbiome-Gut-Brain Axis

Emerging evidence suggests that the oral microbiome may influence psychological function through the microbiome-gut-brain axis. Periodontal disease involves dysbiosis of the oral microbiome, with increases in pathogenic species that produce lipopolysaccharides and other microbial products that can trigger systemic inflammation. These products may influence brain function either directly through bloodborne routes or indirectly through effects on the gut microbiome and gut-brain communication pathways.

Periodontal therapy significantly alters the oral microbiome, reducing pathogenic species and promoting healthier microbial communities. These changes may have downstream effects on gut microbiome composition and function, potentially influencing neuropsychological processes including anxiety regulation. While this pathway remains speculative and requires further investigation, it represents an intriguing potential mechanism connecting periodontal therapy and psychological outcomes.

Clinical Implications

Integrated Risk Assessment

The bidirectional relationship between periodontal disease and anxiety suggests the importance of integrated risk assessment approaches that consider both conditions simultaneously. Periodontal clinicians should be aware that patients with advanced periodontitis, particularly smokers, may have elevated anxiety levels that could impact treatment adherence, outcomes, and overall wellbeing. Similarly, mental health professionals should recognize that patients with anxiety disorders may have increased risk for periodontal disease, especially if they also smoke.

Practical implementation might include brief anxiety screening in periodontal practices, particularly for patients with advanced disease or risk factors like smoking. Conversely, basic oral health screening in mental health settings might identify patients who would benefit from periodontal evaluation. Such integrated approaches could facilitate earlier intervention for both conditions.

Tailored Treatment Approaches

The differential response to periodontal therapy observed between smokers and non-smokers suggests the need for tailored treatment approaches. Smokers with periodontitis and anxiety might benefit from more intensive periodontal therapy, potentially including adjunctive antimicrobials to compensate for their reduced healing capacity. Additionally, these patients should receive particular emphasis on smoking cessation counseling, as this could simultaneously benefit both their periodontal health and anxiety levels.

For all patients with comorbid periodontitis and anxiety, treatment planning should consider the psychological dimensions of care. This might include additional attention to pain management, clear communication about expected outcomes, and supportive approaches that enhance sense of control and self-efficacy. Follow-up

intervals might be adjusted based on both clinical and psychological response to initial therapy.

Interdisciplinary Collaboration

The complex interrelationships between periodontal disease, anxiety, and smoking highlight the potential benefits of interdisciplinary collaboration. Periodontal specialists, primary dental care providers, mental health professionals, and smoking cessation specialists might coordinate care for patients with these overlapping conditions. Such collaboration could enhance outcomes across all domains while potentially improving cost-effectiveness and patient experience.

Practical models for such collaboration might include co-location of services, established referral pathways with bidirectional communication, and integrated electronic health records that facilitate coordination. Educational initiatives that increase cross-disciplinary awareness could support these collaborative approaches.

Future Research Directions

Longitudinal Studies

While existing evidence supports an association between periodontal therapy and anxiety reduction, longitudinal studies with extended follow-up periods are needed to determine the sustainability of these psychological benefits. Research examining whether the psychological improvements persist beyond the initial post-therapy period, and whether they require ongoing periodontal maintenance to sustain, would provide valuable insights for clinical practice.

Future longitudinal studies should also investigate whether the relationship between periodontal therapy and anxiety reduction differs based on anxiety severity or specific anxiety disorders. The potential for differential effects in patients with generalized anxiety disorder, panic disorder, or other specific anxiety conditions remains largely unexplored.

Mechanistic Investigations

More detailed investigation of the biological mechanisms connecting periodontal therapy and anxiety reduction would enhance understanding of this relationship and potentially identify therapeutic targets. Future research might simultaneously track changes in inflammatory biomarkers, neuroendocrine factors, oral and gut microbiome composition, and anxiety measures following periodontal therapy to better elucidate causal pathways.

Advanced neuroimaging techniques might also be employed to examine whether periodontal inflammation and therapy influence brain regions and networks involved in anxiety regulation. Such studies could provide more direct evidence of central nervous system effects that might mediate the psychological benefits of periodontal treatment.

Intervention Studies

Randomized controlled trials comparing different periodontal therapy protocols for their effects on anxiety would strengthen causal inferences and help optimize treatment approaches. Studies might compare standard NSPT to more intensive approaches including adjunctive antimicrobials or host modulation therapies to determine whether more aggressive periodontal management yields enhanced psychological benefits.

Intervention studies incorporating smoking cessation alongside periodontal therapy could help clarify the relative contributions of these factors to psychological outcomes. Such research might employ factorial designs to evaluate periodontal therapy alone, smoking cessation alone, combined approaches, and control conditions to determine optimal integrated treatment strategies.

Implementation Science

Finally, research examining how to effectively implement integrated approaches to periodontal and psychological care in real-world clinical settings would facilitate translation of research findings into practice. Studies evaluating the feasibility, acceptability, effectiveness, and cost-effectiveness of various integrated care models could guide clinical and policy decisions.

Implementation research might also identify barriers and facilitators to interdisciplinary collaboration and develop strategies to overcome challenges. Such work is essential to ensure that the potential benefits of addressing periodontal disease and anxiety together are realized in everyday clinical practice.

CONCLUSION

This review highlights the emerging evidence supporting a bidirectional relationship between periodontal disease and anxiety, with non-surgical periodontal therapy demonstrating potential psychological benefits alongside its established clinical effects. Patients with advanced periodontitis, particularly smokers, typically exhibit elevated anxiety levels compared to periodontally healthy individuals. Following non-surgical periodontal therapy, significant reductions in anxiety scores are observed, with the magnitude of improvement correlating with clinical periodontal parameters and generally greater in non-smokers than smokers.

Multiple biological mechanisms potentially mediate this relationship, including inflammatory pathway modulation, neuroendocrine regulation, and possibly the microbiome-gut-brain axis. The clinical implications include opportunities for integrated risk assessment, tailored treatment approaches considering both periodontal and psychological dimensions, and interdisciplinary collaboration between dental and mental health providers.

Future research should continue to explore this relationship through longitudinal studies with extended follow-up, detailed mechanistic investigations, intervention trials comparing different treatment strategies, and implementation science initiatives to translate findings into practice. Such work promises to enhance understanding of the oral-systemic health connection and improve comprehensive patient care.

The relationship between periodontal therapy and anxiety reduction underscores the importance of holistic approaches to health that recognize the interconnections between oral and psychological wellbeing. For clinicians, this evidence suggests that effective management of periodontal disease may confer benefits beyond the oral cavity, potentially contributing to improvements in overall quality of life and psychological health.

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