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Review Article

Mechanistic Pathways Linking Depression and Anxiety as Risk Factors for Progression of Periodontal Disease: Role of Cortisol, Inflammation and Health Behaviours

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ABSTRACT

Both periodontitis and common mental disorders are highly prevalent, chronic and mutually reinforcing. There is growing evidence that depression and anxiety are risk indicators for the onset, severity and treatment resistance of periodontal disease, independent of shared confounders such as smoking and socioeconomic status. This narrative review summarizes current mechanistic evidence linking depressive and anxiety disorders with periodontal breakdown through three interrelated pathways: (i) neuroendocrine dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and resultant hypercortisolaemia; (ii) systemic and local proinflammatory cytokine dysregulation, involving interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α); and (iii) maladaptive health behaviours, including poor oral hygiene compliance, tobacco use, and reduced engagement with dental care. These pathways are not independent with chronic psychological stress leading to prolonged glucocorticoid exposure which paradoxically does not suppress, and may potentiate, local periodontal inflammation. Behavioural disengagement adds to biological susceptibility. Clinical evidence also supports that nonsurgical periodontal therapy is less effective under psychological stress. The recognition of depression and anxiety as modifiable, biologically active risk indicators has implications for periodontal risk assessment, interdisciplinary care between dentistry and mental health services, and the design of future intervention trials targeting the stress-inflammation-behavior axis.

Introduction

Periodontitis is a chronic, multifactorial, dysbiosis-driven inflammatory disease of the tooth-supporting apparatus that remains a major public health problem and one of the most prevalent non-communicable diseases worldwide . Severe forms of periodontitis affect several hundred million adults worldwide with substantial cross-national variation associated with socioeconomic development . Nationally representative surveys with full-mouth periodontal examination protocols show that periodontitis of any severity affects >40% of adults and that the greatest risk is among older individuals, men, and cigarette smokers [28]. It progresses through the cumulative destruction of the periodontal ligament and alveolar bone, and if left untreated, results in tooth loss, with major consequences for masticatory function, nutrition, systemic health and quality of life [2]. The disease is formally classified and staged by severity, extent, and rate of progression under the 2017 World Workshop framework [3,4] . Mental health disorders are explicitly mentioned in current risk-factor models for periodontitis and peri-implantitis as a patient-related risk factor together with smoking,

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diabetes and socioeconomic status to be considered in the clinical context. [23]

Depression and anxiety disorders are the two largest contributors to the global burden of mental illness. In 2020, there were an estimated 246 million people in the world with an anxiety disorder and 193 million with a major depressive disorder, numbers that were greatly inflated by the COVID-19 pandemic. [5] Mental disorders account for almost 5% of all disability-adjusted life-years worldwide over the lifespan, with depressive and anxiety disorders contributing the most to this burden. [6]

The co-occurrence of these two disease groups is not a coincidence. There is growing clinical, epidemiological and biomarker evidence that depression and anxiety are true risk indicators for periodontal disease onset, severity and poor response to treatment, beyond the well established confounding effects of smoking, poor oral hygiene and low socioeconomic status.[7] Systematic reviews of biomarker studies consistently, though heterogeneously, associate psychological distress with salivary or serum markers of stress and inflammation in patients with periodontitis.[8] Narrative synthesis has indicated that chronic stress-related disease and periodontal/peri-implant disease share overlapping psychobiological pathways, including impaired wound healing. [9]

Three broad, interactive mechanistic pathways have been proposed to explain this relationship: neuroendocrine dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and its principal effector hormone, cortisol; dysregulated systemic and local inflammatory signaling; and behavioral pathways by which mental disorders erode oral self-care and increase exposure to periodontal risk factors such as tobacco use . This review synthesises current evidence for each pathway and considers their interaction to accelerate periodontal tissue destruction.

SEARCH STRATEGY

A structured narrative literature search was performed in PubMed/MEDLINE and Scopus-indexed sources (via Consensus academic search, which aggregates PubMed, Scopus and Semantic Scholar records) for articles published in English language on depression, anxiety, chronic psychological stress, HPA axis, cortisol, pro-inflammatory cytokines, health behaviour and periodontal or peri-implant disease. Systematic reviews, meta-analyses and Periodontology 2000 thematic volumes were prioritised, supplemented by primary biomarker and cohort studies where these provided mechanistic detail not available in synthesis form. All citations were cross-checked with the original PubMed/journal entries before inclusion.

EPIDEMIOLOGICAL OVERVIEW: BIDIRECTIONAL ASSOCIATION

Cross-sectional and cohort studies consistently find that the prevalence and severity of periodontal disease are greater in those with depression and anxiety compared to controls, although the strength of the association varies depending on the diagnostic method, population and control for confounders. Kisely and colleagues performed a systematic review and meta-analysis of 26 studies including more than 330,000 subjects.[20] .In this meta-analysis, people with depression, anxiety or dental phobia had significantly greater tooth loss and

dental decay than the general population, and the relationship for periodontal disease specifically was more variable, although it was strengthened for panic disorder.[20] Another large German case-control study comparing patients with and without periodontitis found that across a broad panel of psychosocial variables dental anxiety and general anxiety, depressive symptomatology and lower socioeconomic status were all significantly elevated in the periodontitis group with dental anxiety and socioeconomic position being the strongest independent predictors. [24]

The studies have tried to establish the effect of psychological state on the results of periodontal treatment, and not just compare prevalence. A systematic review of longitudinal intervention studies found that psychological stress was associated with poorer response to non-surgical periodontal therapy (NSPT) in the majority of studies included, with stressed patients demonstrating greater residual probing depth and bleeding on probing after treatment. [25] A prospective study of patients with generalised stage III/IV periodontitis also found that increased perceived stress and avoidant coping strategies were associated with significantly worse post-treatment bleeding scores and probing depths at three-month re-evaluation.[26] These findings indicate that the depression/anxiety-periodontitis association is not simply a shared risk exposure, but an active biological influence on disease progression and healing ability.

MECHANISTIC PATHWAY ONE: NEUROENDOCRINE DYSREGULATION – THE HPA AXIS AND CORTISOL

The hypothalamic–pituitary–adrenal (HPA) axis is the main neuroendocrine mediator of the physiological stress response. Acute stress causes the hypothalamus to secrete corticotropin-releasing hormone, which stimulates the pituitary to release adrenocorticotropic hormone, which stimulates the adrenal gland to secrete cortisol.[14] Cortisol is normally secreted in a pulsatile, circadian rhythm, which is essential to the regulation of immune and metabolic function; in chronic depression and anxiety, persistent or dysregulated glucocorticoid signalling disrupts this rhythm and has been implicated in a number of stress-related disease conditions.[13]

Hyperactivity of the HPA axis is a robust and replicable finding in depressive disorders, specifically, with elevated basal cortisol, a blunted cortisol suppression on a dexamethasone challenge, and altered diurnal cortisol slopes. Chronic activation of this axis interacts with inflammatory and oxidative stress pathways to generate structural and functional changes in stress-regulatory brain regions and further perpetuate HPA dysregulation. [13]

This neuroendocrine dysregulation has direct biological relevance in the periodontal tissues. Cortisol and other glucocorticoids are immunomodulatory; they have anti-inflammatory, immunosuppressive effects at physiological concentrations but at times of chronic elevation this regulatory capacity is lost, leading to what has been called glucocorticoid receptor resistance, in which target tissues do not respond correctly to

cortisol's anti-inflammatory signal but are still exposed to its immunosuppressive and catabolic effects in other locations. Repeated measures of salivary and serum cortisol concentrations have been performed in periodontitis populations, and a systematic review of 26 biomarker studies concluded that psychological stress and depression are positively, though heterogeneously, associated with cortisol, dehydroepiandrosterone (DHEA) and chromogranin A levels, and that this hormonal profile is qualitatively associated with periodontal disease severity.[8] A related biopsychosocial framework suggests that adverse psychosocial exposure triggers activation of the HPA axis and the release of cortisol, which contributes to a pro-inflammatory immune state, evidenced by hyperactive neutrophils implicated in the destruction of periodontal tissue, with this relationship being amplified among individuals of lower socioeconomic status and higher financial stress .

A parallel, less studied mechanism is the direct effect of stress hormones on the subgingival microbiome. Catecholamines released by the sympathetic adrenal medullary arm of the stress response can influence bacterial growth, biofilm formation, and virulence factor expression in several pathogenic species, and emerging work on periodontal pathogens suggests that noradrenaline can potentiate pro-inflammatory cytokine responses to bacterial lipopolysaccharide providing a plausible neuro-microbial route by which sympathetic activation compounds local tissue inflammation. [7]

The concept of allostatic load, the cumulative physiological 'wear and tear 'that results from repeated or chronic activation of stress response systems, provides an integrative framework for these observations. Allostatic load models aggregate primary mediators (cortisol, catecholamines, DHEA) and secondary indices of cardiovascular, metabolic and immune strain into a composite index instead of using a single biomarker. Reviews applying this framework to periodontal disease argue that it is a more biologically coherent risk indicator than perceived stress scales alone, though the majority of supporting studies are still cross-sectional.[10] Consistent with this model, work characterizing oral immune profiles in the context of socioeconomic adversity has identified a 'stressed-out 'oral immune phenotype, in which chronic exposure to psychosocial stressors is associated with hyperactive neutrophil function and an augmented inflammatory response to periodontal bacterial challenge.[11]

MECHANISTIC PATHWAY 2: SYSTEMIC AND LOCAL INFLAMMATORY DYSREGULATION

Depression is linked to a chronic, low-grade systemic inflammatory state, regardless of neuroendocrine effects. A landmark meta-analysis of cytokine concentrations in major depression combining data from 24 studies noted significantly increased circulating concentrations of the pro-inflammatory cytokines interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α) in depressed subjects compared with non-depressed controls.[15] Later reviews of the cytokine

hypothesis of depression have confirmed this pattern multiple times across IL-1β, TNF-α, IL-6 and C-reactive protein, suggesting that activation of the innate immune inflammatory response system is a central aspect of the pathophysiology of depression, with plausible bidirectional signaling between peripheral cytokines and central neurotransmitter and neuroplasticity pathways. [16]

These same cytokines are main mediators of destruction of periodontal tissue. Periodontitis is essentially a disease of dysregulated host-microbial interaction, in which bacterial biofilm accumulation triggers a local innate and adaptive immune response; IL-1β, TNF-α and IL-6 drive osteoclastogenesis, matrix metalloproteinase activation and connective tissue breakdown at the periodontal pocket 7 . As these same mediators are systemically elevated in depression, a plausible convergence exists. A person with depression enters any periodontal bacterial challenge with a pre-sensitised pro-inflammatory systemic immune background that may amplify the local tissue response to a given microbial load. Likewise, in population-based studies of inflammatory ageing markers, depression in addition to smoking and periodontal disease itself has been identified as an independent correlate of elevated IL-6 in older adults, and IL-6 has emerged as the marker most robustly associated with incident disease and disability across multiple organ systems.[17] This underscores the fact that this cytokine functions as a shared final common pathway linking psychiatric and periodontal pathology rather than a phenomenon specific to either condition alone. Reviews of modifiable risk-factors for periodontitis explicitly position psychological stress and depression alongside smoking, obesity and metabolic disease as contributors to the systemic inflammatory burden that accelerates age-related periodontal breakdown, reinforcing that these pathways operate within, rather than separate to, the broader chronic-disease risk architecture.[19]

Counter-regulatory anti-inflammatory signalling adds another level of complexity. Interleukin-10 (IL-10), a major immunoregulatory cytokine, acts on and regulates the HPA axis, increasing corticotropin-releasing hormone and adrenocorticotropic hormone release and decreasing adrenal cortisol secretion. It has been shown to contribute to maintaining homeostasis and reducing alveolar bone loss in periodontal tissue. Emerging models suggest that chronic stress may interfere with this IL-10 mediated regulatory loop, tipping the local cytokine balance in the periodontal tissue even more towards net tissue destruction.[14]

A third, more speculative but biologically plausible pathway is neuroinflammatory cross-talk between periodontal pathogens and the central nervous system. The association between periodontitis and neuropsychiatric disorders such as major depression has been reviewed, suggesting that periodontal pathogens and their inflammatory products can enter the systemic circulation and in susceptible individuals contribute to neuroinflammation, suggesting that the association between periodontal and psychiatric disease may be

reciprocal rather than unidirectional, with chronic systemic inflammation of periodontal origin plausibly contributing to, as well as being a consequence of, depressive symptomatology.[18]

MECHANISTIC PATHWAY 3: HEALTH BEHAVIOUR MEDIATION

In addition to the direct neuroendocrine and immunological mechanisms, depression and anxiety have a major effect on periodontal health via behavioural pathways. Core symptoms of depressive illness such as anhedonia, fatigue, psychomotor slowing and lowered motivation for self-care directly undermine adherence to daily oral hygiene practices such as toothbrushing and interdental cleaning . Anxiety disorders, in particular dental anxiety, are strongly linked to avoidance of professional dental care and delayed presentation for treatment.[7,24]

One of the most important behavioral mediators is tobacco use. Smoking is one of the strongest established modifiable risk factors for periodontitis, impairing neutrophil function, gingival microvascular perfusion and wound healing, and promoting a dysbiotic subgingival microbiome. Detailed reviews of the effects of tobacco on periodontal health report consistent associations between smoking status and increased probing depth, attachment loss and reduced response to periodontal therapy.[21] Tobacco use is considerably more prevalent in those with depressive, anxiety and psychotic-spectrum disorders than in the general population and this behavioural clustering has been proposed to be a major contributor to the poorer oral health profile seen in psychiatric populations more generally.[7]

Alcohol misuse, diet change and disengagement from preventative health services follow a similar pattern. In a comparative study of patients with depression or attention-deficit/hyperactivity disorder, psychiatric patients reported significantly poorer oral health-related quality of life than mentally healthy controls. The authors noted a paradoxical or contradictory relationship between self-reported oral hygiene behaviours and outcome scores, which they interpreted as reflecting altered self-perception and self-monitoring of oral health status in psychiatric patients rather than straightforward behavioural neglect alone.[29]

It is worth noting that in practice, behavioural and biological pathways cannot be separated. Suboptimal plaque control interacts with an already pro-inflammatory, hypercortisolaemic host response to produce a disease trajectory that is both more likely to develop and more resistant to standard non-surgical intervention. This pattern is directly reflected in the finding that patients using avoidant coping strategies under psychosocial stress show significantly poorer plaque scores alongside poorer clinical healing outcomes following periodontal therapy.[26] Therefore, formal behavioural change frameworks tailored to dental practice, focusing on motivation, self-efficacy and habit formation in oral hygiene, have been proposed as an integral rather than adjunctive part of

periodontal prevention and therapy in psychologically vulnerable populations.[22]

BRIDGING THE PATHWAYS: A CONVERGENT MODEL

The three pathways described above are best thought of not as independent, competing explanations but as interacting arms of a single psychobiological system. The HPA axis is activated by the chronic psychological stress associated with depression and anxiety, which results in sustained glucocorticoid exposure that is inadequate to control local periodontal inflammation because of glucocorticoid receptor resistance, but permits the elevation of systemic pro-inflammatory cytokines (IL-6, TNF-α) characteristic of depressive illness itself. This neuro-endocrine-immune dysregulation together decreases the threshold for destructive responses to the periodontal pathogens in the tissue. At the same time, depressive and anxiety symptomatology erodes the behavioral buffers of oral hygiene, tobacco avoidance and regular professional care that would otherwise limit bacterial challenge, so that a biologically sensitized periodontium is also more frequently and heavily exposed to its principal etiologic trigger . This convergence has clinical implications, as treatment-outcome studies have demonstrated that psychosocial stress and avoidant coping predict poorer response to nonsurgical periodontal therapy even when baseline severity of disease is comparable across groups.25,26 suggesting mechanistic pathways operate throughout the disease course rather than at initiation only.

IMPLICATIONS FOR CLINICAL AND RESEARCH

The recognition of depression and anxiety as biologically active and modifiable risk indicators for periodontal disease has several practical implications. First, periodontal risk assessment protocols could justifiably include brief validated screening for depressive and anxiety symptoms in addition to established behavioural risk factors, such as smoking, particularly in patients presenting with disease severity disproportionate to plaque control or with poor response to conventional therapy.[7,10] Second, closer interdisciplinary collaboration between dental and mental health providers may improve outcomes for both conditions. Adjunctive stress-reduction or coping-skills interventions delivered in conjunction with standard non-surgical therapy may benefit periodontal treatment planning for patients with diagnosed mood or anxiety disorders, an approach supported, if not yet definitively proven, by intervention studies linking coping style to treatment response.[26] Third, the large methodological heterogeneity across the biomarker literature, differing assay methods, sampling times, and psychological instruments points to a clear need for standardised, longitudinal biomarker protocols (combining cortisol, IL-6, TNF-α, and validated depression/anxiety instruments) capable of establishing temporal and dose-response relationships rather than the cross-sectional associations that currently predominate. [8]

LIMITATIONS OF THE EVIDENCE BASE

Several limitations temper causal interpretation of the literature reviewed. The majority of primary studies are cross-sectional in nature, and so no firm conclusions can be drawn regarding the direction of causality between psychological state and periodontal status. A bidirectional and multivariable Mendelian randomisation analysis of major depressive disorder, anxiety and stress-related disorders in relation to dental caries and periodontitis found limited and inconsistent evidence of a causal single-variable association, with no significant relationship surviving adjustment for smoking status and educational attainment in multivariable analysis . This highlights the fact that in some, but not all, cases, observational associations may be due to shared upstream determinants (e.g., socioeconomic adversity) rather than direct causal pathways . The definition and measurement of exposure (perceived stress scales versus clinical diagnostic interviews for depression/anxiety) and outcome (self-reported versus full mouth clinical periodontal examination) vary substantially between studies, limiting comparability and pooling. Finally, most mechanistic biomarker studies are underpowered and do not adjust comprehensively for the behavioural confounders (smoking, oral hygiene, healthcare utilisation) that plausibly mediate a substantial portion of the observed association, making it difficult to isolate the independent contribution of neuroendocrine and inflammatory pathways from behavioural pathways in currently published data.

CONCLUSIONS

There is growing recognition that depression and anxiety are not merely coincidental comorbidities but are genuine, biologically based risk indicators for the progression of periodontal disease. Converging evidence implicates three interacting mechanistic pathways: (a) dysregulation of the HPA axis and prolonged exposure to cortisol, (b) elevation of systemic and local pro-inflammatory cytokines, and (c) behavioural erosion of oral self-care and preventive engagement. These pathways work together to reduce the threshold for destructive inflammatory responses of the periodontal tissue, and to impair healing after conventional therapy. The evidence base is still largely cross-sectional and mechanistically incomplete, however, there is enough consistency to warrant the inclusion of psychological screening in periodontal risk assessment and to encourage closer integration of dental and mental health care for affected patients. Future longitudinal and intervention studies using standardised psychological and biomarker measures are needed to clarify causal direction and to determine whether targeted psychological or stress-reduction interventions can measurably improve periodontal treatment outcomes.

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