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RELATIONSHIP BETWEEN PERIODONTAL DISEASES AND MENTAL HEALTH: A NARRATIVE REVIEW

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Abstract

Introduction. Periodontal diseases are prevalent oral diseases affecting the global population. These diseases arise from infections and inflammation of the gums and supporting bones, significantly impacting oral health. The established link between periodontal and systemic diseases underscores their importance as a public health concern. Chronic stress and depression can result in general dysregulation of the immune system, of both cellular and humoral pathways, which may contribute to pathogenic infection and concomitant periodontal tissue destruction.
Aim. This work aims to analyze the literature data of scientific research on this topic to assess the pathogenetic mechanisms of the stress response to the condition of periodontal tissues. The evidence is consistent with the hypothesis that stress can modify the host defense and progression of periodontal infections in patients susceptible to periodontitis.
Materials and methods. The authors conducted a review of contemporary literature regarding the connection between psycho-emotional stress, periodontal diseases, and mental health. It was utilized 4 databases to find articles published from 2010 to November 2024. Initially, it was identified 1712 records, and after screening and applying specific inclusion and exclusion criteria, 98 articles were selected for full-text review. Ultimately, 56 records were excluded after reading full-text, and the process is visually summarized in a PRISMA flowchart.
Results. Periodontal diseases are primarily caused by bacteria in dental plaque, with key pathogens like *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* leading to severe tissue destruction. The disease progresses through a complex interplay between microbial activity and the host's inflammatory response, with dysbiosis contributing to its cyclic nature. Chronic stress is a significant risk factor, creating a bidirectional relationship with periodontal disease and potentially exacerbating its progression.
Conclusions. Understanding this bidirectional relationship highlights the importance of comprehensive health approaches integrating periodontal care with mental health support. Improving oral health contributes to better physical outcomes and enhances mental well-being, emphasizing the need for interdisciplinary strategies in patient care. Future studies must clarify the complex interactions of chronic stress in periodontal diseases.

Keywords: **mental health, periodontal diseases, psycho-emotional stress, chronic stress**

INTRODUCTION

Periodontal diseases are a significant global health concern, affecting approximately 3.5 billion individuals worldwide. This prevalence, as reported by the global burden of disease estimates within the World Bank, the World Health Organisation and the Institute for Health Metrics and Evaluation in 2019, underscores the urgent need for research and intervention in this field [1].

Periodontitis, the leading cause of tooth loss in both developing and developed nations, is a matter of concern. Its global prevalence, affecting 5-15% of the general adult population, highlights the need for effective prevention and treatment strategies [2].

Severe periodontitis, which may result in tooth loss, is found in 5-20% of adult populations worldwide [3]. Children and adolescents can have any

of the several forms of periodontal diseases, such as necrotizing diseases, periodontitis, and periodontitis, as a manifestation of systemic diseases [4, 5].

Periodontitis is a dysbiotic biofilm-initiated chronic inflammatory disease of the tooth-supporting apparatus, which is mediated by a dysregulated host response leading to a progressive loss of connective attachment and supporting alveolar bone [6].

Chronic periodontitis is a complex disease of infectious origin characterized by chronic inflammation and destruction of the supporting structures of the teeth, which is attributed to the presence of bacteria in the dental plaque [7]. This problem's significance is determined by the long chronic course of the inflammatory process, negative influence on the patient's organism, and decreased life quality.

Stress markers, including cortisol and catecholamines, influence the growth of periodontal bacteria and the expression of their virulence factors. Chronic low-grade inflammation, often associated with stress and certain mental health conditions, may play a role in the connection between periodontal disease and these disorders and vice versa [8].

AIM

This study aims to analyze the scientific literature on the pathogenetic mechanisms of stress responses affecting periodontal tissues.

MATERIALS AND METHODS

The authors analyzed, compared, and summarized information from modern scientific literature on the investigated problem. A literature search was conducted using MEDLINE/PubMed, the Cochrane Oral Health Group specialist trials register, Scopus, and Google Scholar. An additional hand search was conducted in the reference lists of the retrieved articles. A combination of search terms related to psycho-emotional stress, periodontal diseases, and mental health were used. Studies were eligible for inclusion if they involved human adults, included psycho-emotional factors as an exposure measure, outcome measures of periodontal conditions. Only English-language articles were considered.

RESULTS

A narrative review style [9] was used, employing a comprehensive literature search for this review article. The search considered works published from 2010 until November 2024 using the abovementioned keywords, as we want to include only up-to-date information. The inclusion criteria are as follows: (i) full-text journal articles written in English; (ii) books and book chapters written in English. The exclusion criteria are as follows: (i) case reports (clinical trials), (ii) conference papers, (iii) materials published earlier than 2010, (iv) randomized controlled studies, and (v) editorials.

(1) The search was carried out in MEDLINE (PubMed), the Cochrane Oral Health Group specialist trials register, Scopus, and Google Scholar, using the keywords «stress», «endocrine system», «cortisol,» and «periodontal disease» in various combinations. In total, 1712 records were found. After the removal of duplicates, 893 articles were left and screened by title and abstract.

(2) Co-authors analyzed 893 records for compliance with the inclusion and exclusion criteria. Scientific papers were distributed equally among the authors for screening. Additional documents (7) were identified by hand-searching the reference lists and included in the manuscript due to the importance of the information discussed. In total, 795 records were deleted, i.e., 98 records remained.

(3) All selected records were distributed among all authors for reading the full-text articles and preparing the manuscript. At this stage, fifty-six (56) records were excluded after reviewing the full text for eligibility. The procedure is shown in Figure 1 in the PRISMA flowchart [9].

DISCUSSION

The etiology and pathogenesis of periodontal disease

The etiological agent of periodontal diseases is bacteria. Dental plaque is the principal etiological factor in the pathogenesis of periodontal diseases. The presence of plaque is necessary but is insufficient for periodontal diseases to occur [10].

As mentioned, the microbes involved in periodontal diseases are largely gram-negative anaerobic bacilli, with some anaerobic cocci and a large quantity of anaerobic spirochetes. The primary organisms linked with deep destructive periodontal lesions are *Porphyromonas gingivalis*, *Prevotella intermedia*, *Bacteroides forsythias*, *Aggregatibacter actinomycetemcomitans*, and *Treponema denticola* [11].

The human oral cavity harbors a substantial and continuously evolving load of microbial species. The ecological interactions between the host and microbes determine the severity of the disease [12, 13].

Although bacteria are well established as the etiological agents of periodontal disease, their presence alone cannot produce advanced tissue destruction in all individuals, suggesting that there is an individual response and adaptation to a certain amount of bacterial biofilm without progression of the disease [14].

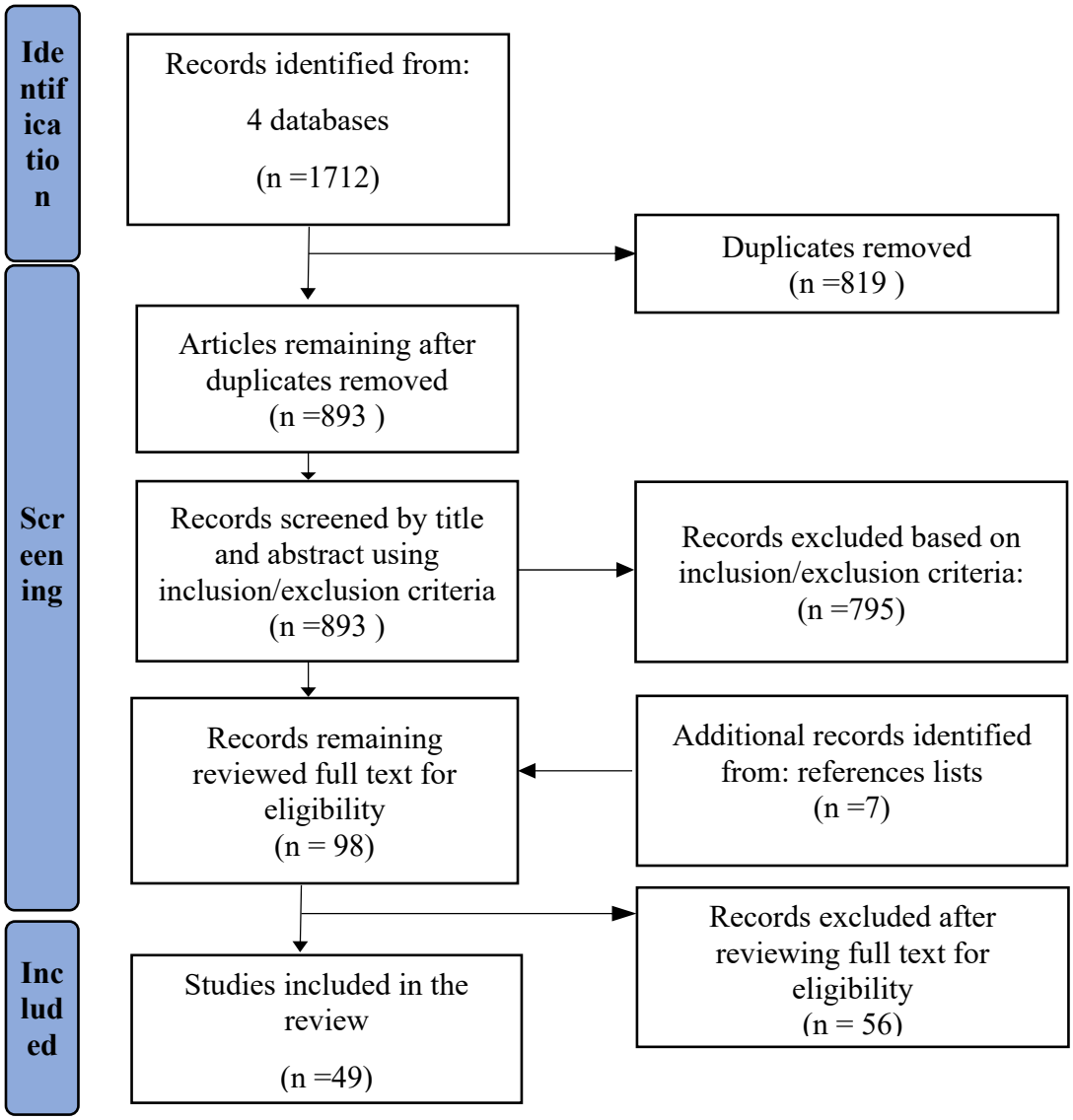

Figure 1. PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow diagram of inclusion/exclusion criteria [9].

The pathogenesis of periodontal diseases is complex, but it is generally agreed that the initiating etiologic event involves infection with a group of predominantly gram-negative anaerobic bacteria that colonize the subgingival area. The progression of the disease is dependent upon a complex inter-relationship between microbial activity and the host's inflammatory response to the microbial challenge, which progressively leads to connective tissue degradation and alveolar bone loss [15, 16].

The commensal oral bacteria are responsible for initiating and propagating the disease through dysbiosis, or microbial imbalance. The disease proceeds cyclically with periods of activity and inactivity until therapeutic action is taken, or the tooth and surrounding structures are destroyed by the disease process, which may result in tooth loss [17]. As periodontal diseases progress from gingivitis to periodontitis, a more significant number of anaerobic organisms colonize deeper periodontal pockets, such as *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis*, which triggers the host inflammatory response. This response includes the production and dissemination of C-reactive protein (CRP), a biomarker of inflammation, as well as various neutrophil and macrophage compounds such as tumor necrosis factor-alpha (TNF-a), matrix metalloproteinases (MMPs), and interleukins (IL-1 and IL-8). An elevated serum CRP level suggests that the inflammation arising from periodontitis may correlate with cardiovascular pathology [18]. Additionally, smoking creates an increasingly favorable environment for the growth of periodontal pathogens, thus furthering the disease process [13].

Risk Factors for Periodontal Disease

Several factors increase the risk of periodontal diseases. These risk factors, modifiable and non-modifiable, contribute toward the clinical significance of periodontal diseases (Tab. 1, Fig. 2) [19, 20].

Table 1

Risk factors of periodontal diseases

Modifiable factors		Non-modifiable factors
Acquired:	**Anatomical:**	**Acquired:**
Smoking Diabetes Cardiovascular Disease Poor diet Osteoporosis Stress	Malpositioned teeth Furcations Root grooves and concavities Enamel pearls	Socioeconomic status Genetics Sex Race and gender Age

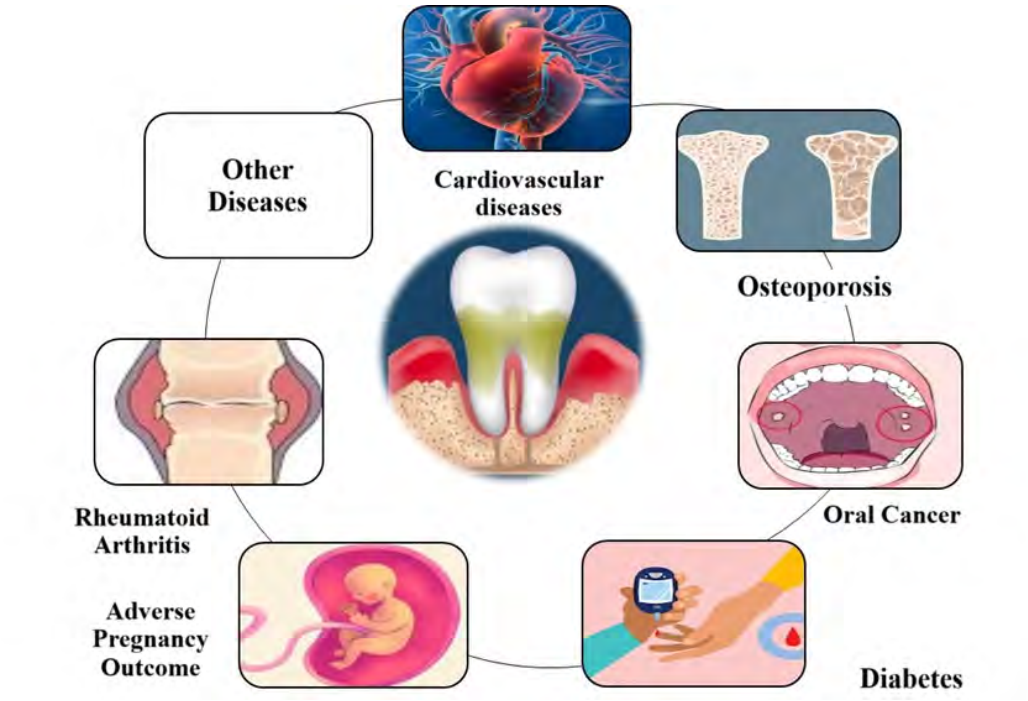

Figure 2. Links between periodontal diseases and other diseases (Figure is created by authors).

However, in this study, one of the critical domains we intended to examine is chronic stress, considering that chronic periodontitis and chronic stress have a bidirectional relationship.

Stress is a physiological response of the body but may not be so when the individual is persistently exposed to the stress trigger. Additionally, it negatively affects certain physiological functions, thus triggering psychosomatic diseases [21].

Nowadays, the term stress is used with extreme frequency to describe recurring negative experiences, as the psychological response of the organism to a perceived challenge or threat, occurring when an individual perceives that environmental demands exceed their adaptive capacity [22].

Stress

Stress refers to the strain, either physiological or psychological, that arises from negative physical, mental, emotional, internal, or external factors disrupting the normal functioning of an organism [23]. Stress-related diagnoses are receiving attention due to recent increases in traumatic events (e.g., war, terrorism, conflict, natural disasters). Traumatic experiences occur every day; it is estimated that 50% of adults will experience a traumatic event in their lifetime [24]. Given the ubiquity of traumatic events, it is not surprising that posttraumatic stress disorder (PTSD) – a common diagnosis following one of these experiences – is characterized as conferring a significant burden for individuals and society [24].

Stress disorders are a critical public health issue with potentially harmful outcomes that have a significant impact on those living with these disorders, the health care system, and society [25].

Pathophysiological factors of stress

In a highly coordinated response to physical and perceived stress, the brain activates the hypothalamic-pituitary-adrenal (HPA) axis, the sympathetic (SNS), and parasympathetic branches of the autonomic nervous system [26].

The HPA axis releases glucocorticoids, a class of steroidal hormones important in stress-related pathology [27]. Glucocorticoids exert myriad effects on

the body, ranging from immunosuppression to cytokine and blood glucose level modification. In particular, increased levels of glucocorticoids alter inflammatory activity by suppressing inflammatory cytokine production, including interleukins (IL)-1, IL-2, IL-6, and tumor necrosis factors [28].

The SNS also releases catecholamines such as epinephrine and norepinephrine from the adrenal medulla. Catecholamines are monoamine hormones that regulate cardiovascular functioning and are involved in mobilizing and redistributing immune cells throughout the body, often synergizing or antagonizing with the action of glucocorticoids [29]. Evidence from in vitro studies has suggested that levels of epinephrine, norepinephrine, and cortisol might directly influence growth patterns of single strains of periodontal-involved microorganisms [30], although more work is necessary to evaluate whether these growth changes persist when the microorganisms are cultured collectively [31].

Chronic activation of the hypothalamic-pituitary-adrenal axis may influence the initiation and progression of periodontitis, showing dysregulation of circulating cortisol and other glucocorticoids that affect immune function [32].

Stress and periodontal disease: is there a link?

Several pathways have been proposed to explain the stress-periodontal disease association [33, 34]. In general, mechanisms have been grouped into two broad categories:

– ″health-impairing behaviors″ associated with stress, such as increases in tobacco and alcohol consumption, poor oral hygiene, and poor nutritional intake [35];

– ″pathophysiological factors″ that lead to increases in stress hormones, which can indirectly influence inflammatory and immunological profiles and increase the susceptibility to periodontal disease [36].

Behavioral approach

Anxiety and stress affect cognition, decision-making, and performance of various individuals' daily activities. The altered emotional status would change health-related behaviors, such as oral hygiene, oral health examination, smoking, and diet, which might increase the risk of periodontitis [37, 38].

Neglect of oral hygiene

Proper oral hygiene partially depends on the patient's mental health status. Stress and psychological disturbances can lead patients to neglect oral hygiene, and plaque accumulation can harm the periodontal tissue [37]. Studies have shown that the stress generated by university studies results in decreased oral hygiene levels and increased crevicular interleukin-1β levels. The authors conclude that stress is a risk factor for gingival inflammation [38].

Changes in dietary intake

The psychological state of an individual affects the choice, consistency, and quantity of food they consume. This may include increased carbohydrate-rich and soft foods consumption, which do not require as much chewing. This type of diet can result in plaque accumulation. Overconsumption of high-fat foods induced by stress can lead to increased cortisol production and, thus, immunosuppression. Any changes to diet can cause stress and can be a risk factor for periodontal disease [39].

Smoking and other harmful habits

Among the many harmful oral habits, which are believed to be induced by emotional disturbances, smoking is possibly the most important in relation to worsened periodontal conditions [40]. The presence of nicotine in the blood also results in:

– vasoconstriction due to the release of adrenaline and noradrenaline [41]. This vasoconstriction blocks nutrients from reaching periodontal tissues;

– in vitro suppression in antibody response [42];

– inhibition of the neutrophil oral function [43].

Biological approach

Exposure to chronic stress will lead to biological mechanisms that will decrease the patient's immune response and chronic inflammation. These mechanisms are based on complex interactions between immunology, psychology, neurology, and endocrinology [44-49].

CONCLUSIONS

Recent research has highlighted a significant correlation between periodontal diseases and mental health disorders. Periodontal diseases, which include gingivitis and periodontitis, are inflammatory conditions affecting the supporting structures of the teeth. These oral health issues have been associated with a range of mental health conditions, including anxiety, depression, and stress.

The underlying mechanisms may involve systemic inflammation, as both periodontal disease and mental health disorders share common inflammatory pathways. Additionally, the impact of chronic pain, functional impairments, and social stigma related to oral health can exacerbate mental health issues.

Understanding this bidirectional relationship underscores the importance of comprehensive health approaches that integrate periodontal care and mental health support. Enhancing oral health not only contributes to improved physiological outcomes but may also lead to better mental well-being, highlighting the need for interdisciplinary strategies in patient care.

Future prospects of research. Developing strategies that raise awareness of the links between

mental health and oral health will motivate patients to seek integrated care; future research in this area may offer a better understanding of the complex relationships between chronic stress and periodontal disease by administering a perceived stress survey (PSS-10) directly in dental clinics, measuring levels of stress biomarkers (e.g., cortisol), and encouraging patients to maintain both mental well-being and periodontal health.

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The article was funded at the authors' own expense. The authors declare no conflict of interest.

COMPLIANCE WITH ETHICAL REQUIREMENTS

This study was conducted solely on the basis of an analysis of publicly available literature and did not involve human subjects (and no personal data was used), therefore this study does not require approval from the Ethics Committee.

AUTHOR CONTRIBUTIONS

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All authors read and approved the submitted version.

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Резюме

ЗВ'ЯЗОК МІЖ ЗАХВОРЮВАННЯМИ ПАРОДОНТУ ТА ПСИХІЧНИМ ЗДОРОВ'ЯМ: ОГЛЯД

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Вступ. Захворювання пародонту є поширеними захворюваннями порожнини рота, які вражають чисельне населення планети. Ці захворювання виникають завдяки бактеріям та запаленню ясен і деструкції кісткової тканини, що значно впливає на здоров'я ротової порожнини. Встановлений зв'язок між пародонтитом і системними захворюваннями підкреслює їх важливість, як проблеми громадського здоров'я. Хронічний стрес і депресія можуть призвести до загальної дисрегуляції імунної системи, як клітинних, так і гуморальних шляхів, що може сприяти патогенній інфекції та супутньому руйнуванню тканин пародонта.

Мета. Аналіз літературних даних наукових досліджень з даної теми для оцінки патогенетичних механізмів стресової реакції на стан тканин пародонта. Докази узгоджуються з гіпотезою про те, що стрес може змінити захист організму та прогресування пародонтальних інфекцій у пацієнтів, схильних до пародонтиту.

Матеріали та методи. Авторами проведено огляд сучасної літератури щодо зв'язку між психоемоційним стресом, захворюваннями пародонту та психічним здоров'ям. Було використано 4 бази даних для пошуку статей, опублікованих з 2010 по листопад 2024. Спочатку було ідентифіковано 1712 записів, а після перевірки та застосування конкретних критеріїв включення та виключення 98 статей було відібрано для повнотекстового перегляду. Зрештою, 56 записів було виключено після прочитання повного тексту, процес включення/виключення візуально зображено на блок-схемі PRISMA.

Результати. Захворювання пародонту в основному спричинені бактеріями в зубному нальоті, причому такі ключові збудники, як *Porphyromonas gingivalis* і *Aggregatibacter actinomycetemcomitans*, призводять до серйозного руйнування тканин пародонта. Хвороба прогресує через складну взаємодію між мікробною активністю та запальною реакцією організму людини, причому дисбактеріоз сприяє її циклічній природі. Хронічний стрес є значним фактором ризику, який створює двонаправлений зв'язок із пародонтитом і потенційно посилює його прогресування.

Висновки. Розуміння цього двонаправленого зв'язку підкреслює важливість комплексних підходів до охорони здоров'я, які поєднують пародонтологічну допомогу з підтримкою психічного здоров'я. Поліпшення здоров'я порожнини рота сприяє кращим фізичним результатам і покращує психічне благополуччя, підкреслюючи необхідність міждисциплінарних підходів у догляді за пацієнтами. Майбутні дослідження повинні висвітлити складну взаємодію хронічного стресу при захворюваннях пародонта.

Ключові слова: **психічне здоров'я, захворювання пародонта, психоемоційний стрес, хронічний стрес**

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