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REVIEW ARTICLE

Stress and Periodontal Disease

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ABSTRACT

Stress is an unavoidable consequence in life. It is described as adverse emotions or reactions to unpleasant experiences. Thus, stress can be viewed as a process with both psychological and physiological components. The possible mechanisms by which psychosocial factors act on periodontal tissues are oral-hygiene negligence, changes in dietary intake, smoking, bruxism, gingival circulation, alteration in salivary component and flow, hormonal changes, and lowered host resistance.

During stress, the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system interact and release glucocorticoids, which have myriad effects that disrupt homeostasis and lead to increased susceptibility to periodontal diseases.

Stress results in delayed healing of the connective tissues and bone, apical migration of the junctional epithelium, and formation of the periodontal pocket. The relationship between periodontal illness and the psychological predisposing factors is well established in specific conditions, such as acute necrotizing ulcerative gingivitis, which is identified to be significantly associated with high levels of trait anxiety, depression, and other emotional disturbances.

The dental practitioner may always decide to refer patients to appropriate professionals for assistance and counseling. Thus, it is important to recognize patients who are in stress and advice patients about the possible effects of stress on periodontal disease.

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INTRODUCTION

The term "stress" originated from a Latin word "stringere," which means "tight strained." Currently, stress is classified as a "risk indicator" for periodontal disease. Stress is regarded as a cognitive perception of uncontrollability and/or unpredictability, i.e., expressed in a physiological and behavioral response.[3] Thus, stress can be viewed as a process with both psychological and physiological components.

In 1976, Selye[4] was basically responsible for defining stress as the response state of an organism to physical and mental forces beyond the adaptive capacity that lead to diseases of adaptation and eventually to exhaustion and death. He recognized stressors that act to produce positive changes in the body (e.g., exciting, pleasurable), leading to a response state that he defined as "eustress," or stressors could be negative that induce sensations that threaten homeostasis with pain, discomfort, and physical pathology. He defined the negative response state as "distress."

According to Breivik et al,[5] stress is not what happens to someone, but how someone reacts to what happens. They define stress as the psychophysiological response of an organism to perceived threat or challenge. Periodontal diseases are defined as inflammatory diseases caused by pathogenic microflora organized in biofilms surrounding the teeth, resulting in destruction of teeth-supporting tissues that can lead to tooth loss.

Although bacteria play an essential role, they seem to be insufficient to explain the occurrence or progression of the disease. There are several factors, such as age, tobacco use, systemic diseases, and psychological stress that have been identified as important risk factors for periodontitis.

De Marco[6] coined the term "Periodontal Emotional Stress Syndrome" for individuals with severe periodontitis who had emotional stress associated with active service in Vietnam suggesting a role of occupational stress in the progression of periodontitis.

The etiopathogenesis of periodontal disease indicates that periodontitis is a multifactorial disease caused by periopathogens in which host and environmental factors play an important role. Bacteria play an essential role as primary etiological agents, but alone seem to be insufficient to explain the occurrence or progression of the disease. Periodontal disease onset and progression are influenced by various systemic diseases, environmental factors, and psychologic stress that have the potential to alter periodontal tissues and host immune response, resulting in more severe periodontal destruction.

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PSYCHOLOGICAL STRESSORS

Stressor is any stimuli, situation, or circumstance with the potential to induce stress reaction.[1] The effect of the stress response includes anxiety, depression, impaired cognition, and altered self-esteem.

While much more is known about the role of disease processes, such as infection and cancer as stressors capable of inducing far-flung and prolonged inflammatory and classic stress syndromes, it is now considered likely that emotional, behavioral, and psychosocial stressors are also capable of activating the stress system, along with associated immune system effects.

Psychosocial stressors are generally classified as[7]:

- Major stressful life events
- Minor daily stressors or "hassles"

Holmes and Rahe[8] developed a scale to measure stress in terms of life changes. In this scale, the life events are ranked in order, from the most stressful (death of a spouse) to the least stressful (minor violations of the law) (Table 1).

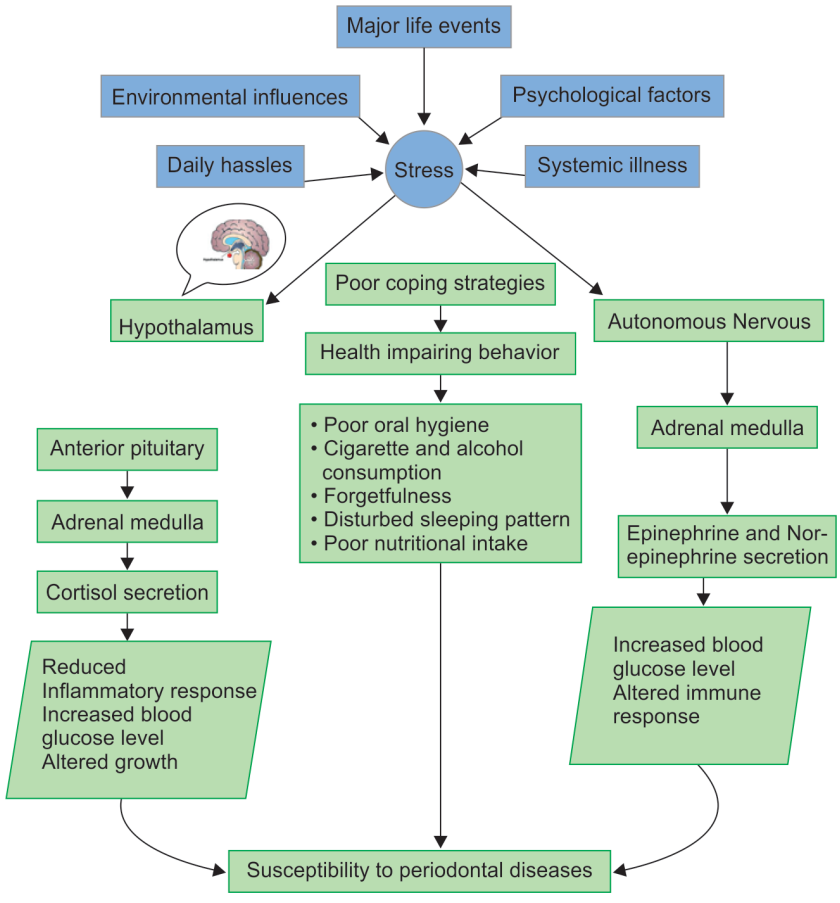
Another set of psychosocial stressors are well-known behavioral and emotional responses to common sequela of advancing periodontal disease, which include such negative and dysphoric conditions as pain, bleeding, unpleasant tastes, and odors emanating from the mouth and unsightly appearance of the teeth and the surrounding hard and soft supporting structures.

Table 1: Life change scale

Events	*Score*
Death of spouse	100
Divorce	73
Marital separation	65
Death of close family member	60
Personal injury or illness	53
Marriage	50
Frustrated from job/loss of work	47
Conjugal reconciliation	45
Retirement	43
Life change scale	

Other signs and symptoms, such as abscess formation with pathogenic exudates and intense pain, loosening of teeth, and the perceived threat of losing one's teeth in early adulthood are also often highly worrisome, hence, serving as potentially powerful negative emotional stressors. Moreover, treatment of periodontal disease is often associated with pain and discomfort as well as being time-consuming and often expensive. All these perceptions, attributions, and emotions associated with illness can themselves come to constitute and act as an important set of stressors that may induce stress-system responses that are further deleterious to periodontal health.[5]

Flow Chart 1:



EFFECTS OF STRESS ON PERIODONTAL DISEASE

A number of mechanisms have been proposed, which could mediate the putative relationship between psychosocial conditions and inflammatory periodontal diseases.

Endocrine Changes

It has been suspected that periodontal status is related to alterations in the concentration of adrenal corticoids and by altering the responses of oral tissues to bacterial toxins and other hormones involved in the general adaptation syndrome.[9]

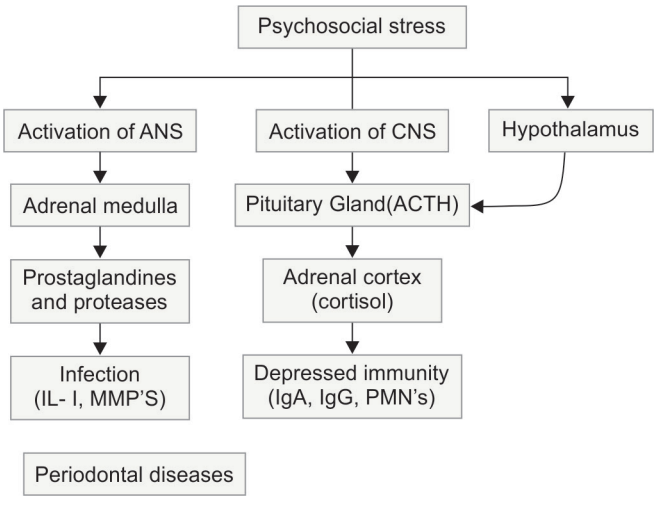
Model 1 (Flow Chart 2)[10] offered a schematic model, which demonstrates the potential role that psychosocial stressors may play in initiating a cascade of events in the corticotropin-releasing hormone/hypothalamic–pituitary–adrenal (HPA) axis, the autonomic nervous system, and the central nervous system, the physiological consequences of which are to depress immunity, enhancing the likelihood of infection and, specifically, periodontal disease. Recent studies had confirmed the fact that the concentrations of cytokines [interleukin (IL)-6, IL-1β, etc.,] and cortisol in the gingival crevicular fluid (GCF) are higher in persons showing depression signs.[11-15]

High cortisol levels may be especially negative on periodontal tissue because of the extremely fast turnover of some periodontal components. Elevated levels of glucocorticoids can decrease *in vitro* fibroblasts, collagen production and *in vivo* sulphated glycosaminoglycans. These alterations may be enough to cause imbalances in the synthesis and breakdown of periodontal tissues, especially if preexisting inflammation is present.

Gingival Circulation

The tonus of the smooth muscle of blood vessels may be altered by the emotions by way of the autonomic nervous system. Furthermore, in long or continued emotions, a constant constriction of blood vessels could alter the supply of oxygen and nutrients to the tissues.[16]

Flow Chart 2: Model 1 for the effects of stress on periodontal disease



Alteration in Salivary Flow and Components

It is assumed that both increase and decrease in salivary flow, induced by emotional disturbance, may affect the periodontium adversely. Emotional distress may also produce changes in saliva pH and chemical composition like immunoglobulin (Ig)A secretion. These relationships between salivary physiology and psychological status do not necessarily demonstrate causation of periodontal disease, but they show a pathway in which periodontal health is influenced by salivary changes.[17]

Lowered Host Resistance

Periodontal diseases are inflammatory diseases associated with local and systemic elevations of proinflammatory cytokines, such as tumor necrosis factor a, IL-6, and prostaglandins and result in tissue destruction by the contribution of matrix metalloproteinases.[18,19] Stress impairs the balance between proinflammatory and anti-inflammatory responses. The relationship between stress and periodontal diseases might be mediated by alterations in GCF IL-1, IL-6 levels, and reduction in polymorphonuclear leukocyte chemotaxis and phagocytosis, and reduced proliferation of lymphocytes.[20]

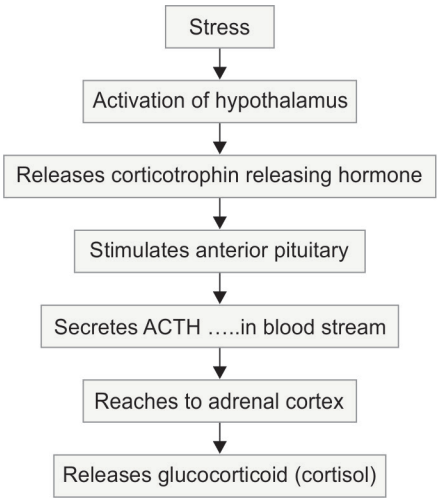
Psychosocial stress stimulates the brain where its stimulation or inhibition is dependent on adaptive and maladaptive coping respectively. On stimulation, the autonomic nervous system leads to prostaglandin and protease secretion that leads to periodontal disease progression. The HPA leads to a production of glucocorticoids (cortisol) that depresses the immune system by diminishing the IgA and IgG secretions, thereby enhancing the periodontal disease progression and poor treatment response.[11] Subsequently, this process could increase vulnerability of periodontal tissues to pathogenic microorganisms by activation of cellular responses leading to local tissue destruction (Flow Chart 3).[21]

Patients suffering from periodontitis, who are under stressful conditions, have increased levels of IL-6[22] and IL-1b[23] in GCF, and similarly, patients with aggressive forms of periodontitis have elevated levels of IL-6 and IL-1b in serum.

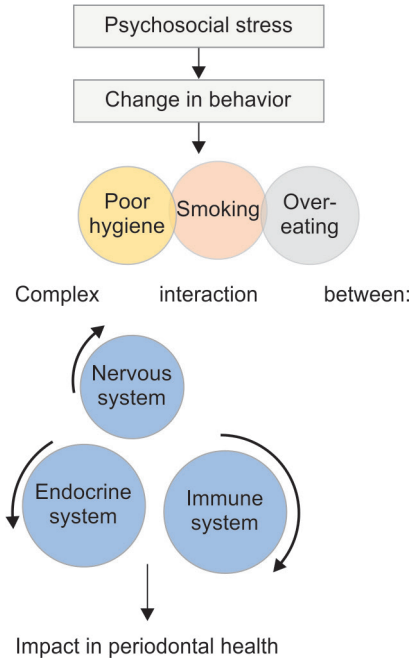
Stress and Behavior Changes

Stress influences the consequences of behavioral patterns, extending from negligence of oral hygiene to dietary inadequacies, poor sleep patterns, use of tobacco products, alcohol consumption that contributes to the

Flow Chart 3: Mechanismof cortisol release upon stress



Flow Chart 4:



"vicious cycle" of increasingly severe forms of advanced periodontal inflammation and disease.

Neglect of Oral Hygiene

It is obvious that proper oral hygiene is partially dependent on the mental health status of the patient. It has been reported that psychological disturbances can lead patients to neglect oral hygiene and that the resultant accumulation of plaque is detrimental to periodontal tissue. Academic stress was reported as a risk factor for gingival inflammation with increasing crevicular IL-b levels and a diminution of quality of oral hygiene.[24-29]

Changes in Dietary Intake

Emotional conditions are thought to modify dietary intake, thus indirectly affecting periodontal status. Psychological factors affect the choice of foods, the physical consistency of the diet, and the quantities of food eaten. This can involve, for instance, the consumption of excessive quantities of refined carbohydrates and softer diets requiring less vigorous mastication and, therefore, predisposing to plaque accumulation at the approximal risk site.[30] Stress leads to other behavioral changes, such as overeating, especially a high-fat diet, which then can lead to immunosuppression through increased cortisol production.

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.[31] Circulating nicotine results in (i) vasoconstriction, produced by the release of adrenaline and noradrenaline, which is supposed to result in a lack of nutrients for the periodontal tissue; (ii) suppression of *in vitro* secondary antibody responses; and (iii) inhibition of oral neutrophil function.

Oral Habits

Neurotic needs find oral expression. The mouth may be used to obtain satisfaction, to express dependency or hostility, and to inflict or receive pain. Sucking, biting, sensing, and feeling may become habitual as in thumb sucking, tongue thrusting, infantile swallowing, and biting of tongue, lip, cheek, or fingernail. These actions also figure in bruxing, clenching, tooth doodling, and smoking. Such habits may lead to tooth migration, occlusal traumatism, and occlusal wear.

Stress and Acute Necrotizing Ulcerative Gingivitis

Acute Necrotizing Ulcerative Gingivitis (ANUG) is the most studied periodontal disorder in relation to psychosocial predisposing factors. A psychogenic origin has been suggested for ANUG. Psychogenic factors probably predispose to the disease by favoring bacterial overgrowth and/or weakening host resistance.[32]

Host tissue resistance may be changed by mechanisms acting through the autonomic nervous system and endocrine glands resulting in elevation of corticosteroid and catecholamine levels. This may reduce gingival microcirculation and salivary flow and enhance nutrition of *Prevotella intermedia*, and at the same time also depress neutrophil and lymphocyte functions, which facilitate bacterial invasion and damage.

It has been reported that ANUG patients compared with controls presented with:

- Depressed polymorphonuclear leukocyte chemotaxis and phagocytosis; and
- Reduced proliferation of lymphocytes upon stimulation by a nonspecific mitogen.

CONCLUSION

Acute stress conditions are immune-enhancing, while chronic stress is immunosuppressive. Stress is associated with more severe periodontal disease as well as poorer healing responses to traditional periodontal therapy. Thus, stress should be assessed and managed properly, as it influences the periodontal tissue destruction, tissue healing, and periodontal therapy outcome.

Stress can cause behavior modification (e.g., smoking, alcohol abuse) and immunosuppressant effect (decreased polymorphonuclear leukocytes, altered T helper 1 cell/T helper 2 cell ratio), which may result in greater recurrence of periodontal disease. The role of the dentist is to discuss lifestyle in a broader concept than just oral hygiene; they should be more psychologically oriented. It is very important to understand the patient's situation to help them to maintain a healthy periodontium.

REFERENCES

1. Formicola AJ, Witte ET, Curran PM. A study of personality traits and acute necrotizing ulcerative gingivitis. J Periodontol 1970 Jan;41(1):36-38.
2. Cohen S, Williamson GM. Stress and infectious disease in humans. Psychol Bull 1991 Jan;109(1):5-24.
3. Koolhaas JM, Bartolomucci A, Buwalda B, de Boer SF, Flügge G, Korte SM, Meerlo P, Murison R, Olivier B, Palanza P, et al. Stress revisited: a critical evaluation of the stress concept. Neurosci Biobehav Rev 2011 Apr;35(5):1291-1301.
4. Selye H. Stress in health and disease. Boston: Butterworths; 1976.
5. Breivik T, Thrane PS, Murison R, Gjermo P. Emotional stress effects on immunity, gingivitis and periodontitis. Eur J Oral Sci 1996 Aug;104(4 (Pt 1)):327-334.
6. De Marco T. Periodontal emotional stress syndrome. J Periodontol 1976 Feb;47(2):67-68.
7. LeResche L, Dworkin SF. The role of stress in inflammatory disease, including periodontal disease: review of concepts and current findings. Periodontol 2000 2002;30:91-103.
8. Holmes TH, Rahe RH. The social readjustment rating scale. J Psychosom Res 1967 Aug;11(2):213-218.
9. Davis CH, Jenkins CD. Mental stress and oral disease. J Dent Res 1962 Sep-Oct;41:1045-1049.
10. Genco RJ, Ho AW, Kopman J, Grossi SG, Dunford RG, Tedesco LA. Models to evaluate the role of stress in periodontal disease. Ann Periodontol 1998 Jul;3(1):288-302.
11. Axtelius B, Söderfeldt B, Nilsson A, Edwardsson S, Attström R. Therapy-resistant periodontitis. Psychosocial characteristics. J Clin Periodontol 1998 Jun;25(6):482-491.
12. Mengel R, Bacher M, Flores-De-Jacoby L. Interactions between stress, interleukin-1beta, interleukin-6 and cortisol in periodontally diseased patients. J Clin Periodontol 2002 Nov;29(11):1012-1022.
13. Johannsen A, Rylander G, Söder B, Asberg M. Dental plaque, gingival inflammation, and elevated levels of interleukin-6 and cortisol in gingival crevicular fluid from women with stress-related depression and exhaustion. J Periodontol 2006 Aug;77(8):1403-1409.
14. Deinzer R, Förster P, Fuck L, Herforth A, Stiller-Winkler R, Idel H. Increase of crevicular interleukin 1beta under academic stress at experimental gingivitis sites and at sites of perfect oral hygiene. J Clin Periodontol 1999 Jan;26(1):1-8.
15. Deinzer R, Kottmann W, Förster P, Herforth A, Stiller-Winkler R, Idel H. After-effects of stress on crevicular interleukin-1beta. J Clin Periodontol 2000 Jan;27(1):74-77.
16. Manhold JH, Doyle JL, Weisinger EH. Effects of social stress on oral and other bodily tissues. II. Results offering substance to a hypothesis for the mechanism of formation of periodontal pathology. J Periodontol 1971 Feb;42(2):109-111.
17. Gupta OP. Psychosomatic factors in periodontal disease. Dent Clin North Am 1966 Mar:11-19.
18. Soell M, Elkaim R, Tenenbaum H. Cathepsin C, matrix metalloproteinases, and their tissue inhibitors in gingiva and gingival crevicular fluid from periodontitis-affected patients. J Dent Res 2002 Mar;81(3):174-178.
19. Buduneli N, Biyikoğu B, Sherrabeh S, Lappin DF. Saliva concentrations of RANKL and osteoprotegerin in smoker versus non-smoker chronic periodontitis patients. J Clin Periodontol 2008 Oct;35(10):846-852.
20. Sheiham A, Nicolau B. Evaluation of social and psychological factors in periodontal disease. Periodontol 2000 2005;39: 118-131.
21. Ishisaka A, Ansai T, Soh I, Inenaga K, Yoshida A, Shigeyama C, Awano S, Hamasaki T, Sonoki K, Takata Y, et al. Association of salivary levels of cortisol and dehydroepiandrosterone with periodontitis in older Japanese adults. J Periodontol 2007 Sep;78(9):1767-1773.
22. Johannsen A, Rydmark I, Söder B, Asberg M. Gingival inflammation, increased periodontal pocket depth and elevated interleukin-6 in gingival crevicular fluid of depressed women on long-term sick leave. J Periodontal Res 2007 Dec;42(6): 546-552.
23. Giannopoulou C, Kamma JJ, Mombelli A. Effect of inflammation, smoking and stress on gingival crevicular fluid cytokine level. J Clin Periodontol 2003 Feb;30(2):145-153.
24. Ringsdorf WM Jr, Cheraskin E. Emotional status and the periodontium. J Tenn State Dent Assoc 1969 Jan;49(1):5-18.
25. Meyer MJ. Stress and periodontal disease: a review of the literature. J N Z Soc Periodontol 1989 Nov;68:23-26.
26. Deinzer R, Rüttermann S, Möbes O, Herforth A. Increase in gingival inflammation under academic stress. J Clin Periodontol 1998 May;25(5):431-433.
27. Deinzer R, Hilpert D, Bach K, Schawacht M, Herforth A. Effects of academic stress on oral hygiene – a potential link between stress and plaque-associated disease? J Clin Periodontol 2001 May;28(5):459-464.
28. Deinzer R, Granrath N, Spahl M, Linz S, Waschul B, Herforth A. Stress, oral health behaviour and clinical outcome. Br J Health Psychol 2005 May;10(Pt 2):269-283.
29. Hildebrand HC, Epstein J, Larjava H. The influence of psychological stress on periodontal disease. J West Soc Periodontol Periodontal Abstr 2000;48(3):69-77.
30. Suchday S, Kapur S, Ewart CK, Friedberg JP. Urban stress and health in developing countries: development and validation of a neighborhood stress index for India. Behav Med 2006 Fall;32(3):77-86.
31. Haber J. Smoking is a major risk factor for periodontitis. Curr Opin Periodontol 1994:12-18.
32. Reners M, Brecx M. Stress and periodontal disease. Int J Dent Hyg 2007 Nov;5(4):199-204.