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REVIEW ARTICLE

Stress and Periodontium: A Review of Concepts

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ABSTRACT

Stress is nevertheless a confirmed and important factor in the etiology and maintenance of many inflammatory diseases, including periodontal disease. Stress results in delayed healing of the connective tissues and bone, apical migration of the junctional epithelium and formation of periodontal pocket. This paper describes general overview of stress, the relationship between stress & periodontium, the current models for understanding how stress mechanisms interact to regulate the onset and course of the disease and the evidence in favor of stress and against the stress being the etiological factor. Thus, it is important to recognize patient who are in stress and to be able to advise patients about the possible effects of stress on periodontal disease if the level of stress cannot be lowered.

KEYWORDS: *Stress, Periodontium, Psychosocial stressors*

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Hippocrates thought of health as a harmonious balance of the elements comprising the quality of life while disease represented disruption of harmony among those elements. In the seventeenth century, Sydenham suggested that pathological states represented diseases of adaptation – failure of the adaptive processes to restore well – being(1). Selye coined the term "Stress". He was responsible for giving the stress its current saliency in relation to the contest between health and disease. Selye defined forces that had the potential to challenge the adaptive capacity of the organism as "stressors" and stated that stressors could be physical or mental (e.g. emotional). He recognized that stressor acting to produce changes in the body could be positive that he defined as "Eustress" or stressors could be negative that he defined as "Distress". Selye also postulated that the mechanism of action central to stress phenomenology was activation of the adreno – cortico – pituitary axis. Disease of adaptation resulted from intense largely uncontrollable chronic distress. Thus chronic distress was seen as potentially contributing to such diseases of adaptation as peptic ulcers, arthritis, asthma, and other pathological conditions mediated by inflammation(2).

Stress is defined as a total transaction from demand to resolution in response to an environmental encounter that requires appraisal, coping and adaptation by the individual. Coping is the response of the individual to stress (emotionally and physically).
Types:

- **Occupational Stress:** E.g. Athletes, Boxers, Diamond cutters.
- **Involuntary Stress:** E.g. Soldiers, Recovery from General anesthesia.
- **Voluntary Stress:** E.g. Dancers, Musicians.

Stress is part of human condition, which is universally present, but to varying degree and with different effects on individuals(3).

Stress is compatible with good health being very necessary to cope with the challenges of everyday life. Problem starts when the stress response is inappropriate to the size of the challenge.

MODELS OF STRESS

Current understanding of stress phenomenology retains the notion of the general adaptation response as essential for survival. Both physical and psychological stressors are capable of initiating central and peripheral responses to maintain homeostasis. In a review of literature by Chrousos and Gold(4), these adaptation responses are summarized into two categories: Behavioral adaptation and Physical adaptation.

Two principal brain components, acting in a highly remarkable fashion, constitute the physiological and biochemical basis for the general adaptation response. Firstly the Corticotropin releasing hormone system, and Secondly the Locus ceruleus nor – epinephrine system. These basic brain elements of the stress system function via the hypothalamic – pituitary – adrenal axis and the systemic adreno – medullary sympathetic nervous system respectively, to modulate the widely dispersed effects of the stress system as it seeks to return both the brain and peripheral organ systems to homeostasis(5).

The communication between the neuroendocrine (Hypothalmic – pituitary – adrenal) and immune inflammatory systems functions as a feedback loop that regulates the immune components of the inflammatory responses. The cells of the immune system are widely distributed throughout the body, when an infection occurs it is the inflammatory response that allows marshalling of immune system elements at specific sites. Early events in the inflammatory reaction to infection are typically clinically undetectable. As the infectious process becomes more chronic, clinically evident inflammation occurs, generating high levels of cytokines and other mediators of inflammation associated with activation of stress system. If the inflammatory reaction is prolonged and profound enough, systemic illness manifestations can also become clinically evident, as might occur with rheumatoid arthritis and also certain forms of periodontal diseases (6,7).

PSYCHOSOCIAL STRESSORS AND STRESS SYNDROMES

Emotional, behavioral and psychosocial stressors are also capable of activating the stress system, along with associated immune system effects.

One of the important impetuses to investing the relationship between psychosocial stress and disease states emerged from the work of Holmes (8) and colleagues, who demonstrated significant relationships between important life event changes and the onset and course of disease.

Subsequent studies clarified two different aspects of this relationship. First, it was major negative life events (9) that more dependably occurred in close proximity to the onset or exacerbation of the illness. Second, the relationship between negative life events and disease was mediated by the immune system, as initially elaborated by Glaser and Kiecolt – Glaser (10-12). Since the work of Holmes relating the major life event changes to illness, other researchers have found that more minor daily stressors may also influence the course of disease and that these relationships are also mediated through the stress – immune system (13).

To summarize, it seems fair to conclude that dysregulation of the stress system is involved in a number of major health problems, but it would be difficult to distinguish between cause and effect since the system is, to a large extent, nonspecific and responds in a similar ways to a wide variety of endogenously and exogenously arising stressors. Moreover, the system seems capable of inappropriate responses that can be highly maladaptive, acting as stressors themselves in sustained 'vicious cycles' of disease and maladaptive dysregulation(14).

STRESS AND PERIODONTIUM

The pioneers who suggested psychological stress might play a role as an aetiological agent of periodontal disease were Dean and Dean (1945) and Schluger (1949).

Stress, distress and coping behaviors are regarded as important indicators for periodontal disease (7). Psychosocial factors can modify the periodontal status through behavioral changes regarding oral hygiene, smoking, dietary intake, bruxism and drug use.

In addition, mechanisms through physiologic pathways may influence periodontal tissues through alteration in saliva, changes in gingival blood circulation, endocrine imbalances and altered host resistance. Psychoneuroimmunologic effects were confirmed by findings of poorer immune functions in persons who experienced stressful life events or chronic stress (15).

Stress hormones may also alter cytokines production, which causes an imbalance between T helper cell phenotypes with a shift to TH2 cell dominance, which is associated with the progression of periodontitis (16).

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 collagen production fibroblasts in vitro and in skin in vivo and sulphated glycosaminoglycans. These alterations may be enough to imbalance the synthesis and breakdown of periodontal tissues, especially if preexisting inflammation is present.

MODELS OF ROLE OF STRESS IN PERIODONTAL DISEASE

Several excellent reviews have sought to synthesize current concepts underlying stress phenomenology into evidence –

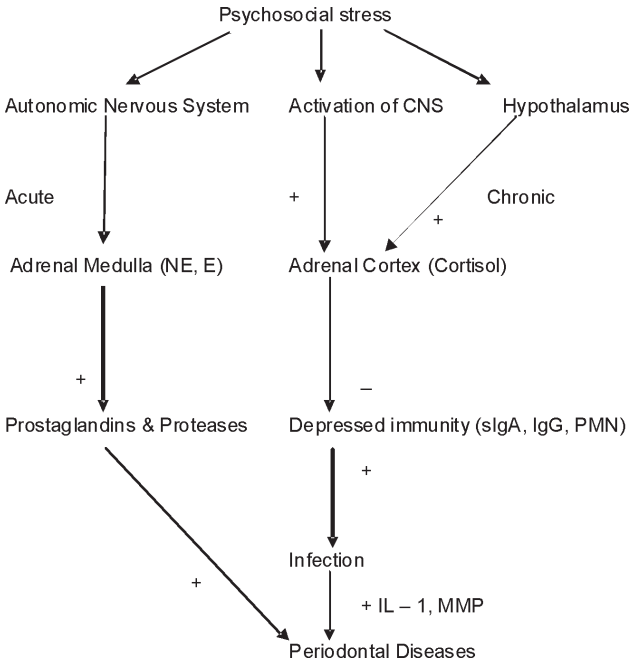

Figure 1: Physiological model for the effects of stress on periodontal diseases.CNS, Central Nervous System; NE, norepinephrine; E, epinephrine; sIgA, secretory Immunoglobulin A;Ig G Immunoglobulin GF; PMN, polymorpho nucleocytes ; IL, interleukin MMP, matrix metalloproteinase

based models linking stress with periodontal disease (7,15). Some of these models include elaboration of stressors from both the physical and psychosocial domains that may serve as risk factors for periodontal disease (16,17).

The relationship between stress and periodontal disease frequently do not consider how stress, through its effects on the course of diabetes (18) and smoking behavior, may influence, in turn, periodontal disease. The presence of environmental stressors may exacerbate the maladaptive behaviors associated with loss of diabetic control (19,20) and influence smoking as well. In addition, both smoking and diabetes act as physical stressors capable of activating the stress – immune system. (20)

Breivik *et al* (16) have stated 'The issue facing us is no longer whether the psyche influences immune cell activities… but rather how influence the development of chronic infections such as gingivitis and periodontitis.

Genco *et al*. (7) offered a schematic model (FIGURE 1), which demonstrates the potential role that psychosocial stressors may play in initiating a cascade of events in corticotrophin releasing hormone/hypothalamic – pituitary – adrenal axis, the autonomic nervous system and the central nervous system, the physiologic consequences of which are to depress immunity, enhancing likelihood of infection and, specifically, periodontal disease. They also proposed that at-risk health behaviors such as poor oral hygiene and smoking might influence periodontal disease directly.

One set of psychosocial stressors not represented in the model are well-known behavioral and emotional responses to common sequelae 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 surrounding hard and soft supporting structures. While gingivitis and periodontal disease in their most common and early forms are typically not painful, symptoms that emerge eventually are known to include fluctuating exacerbations of acute pain that can be very intense or mildly annoying; at later stage, exacerbations of more advanced inflammatory disease are known to include periodontal swelling, abscess formation with pathogenic exudates and intense pain. Other signs and symptoms, such as loosening of teeth and the perceived threat of losing one's teeth in early adulthood are also often worrisome, hence serving as potentially powerful negative emotional stressors. Moreover, treatment of periodontal disease is associated with pain and discomfort, as well as being time consuming and often expensive. All these factors can understandably be viewed as important psychosocial stressors that may induce stress system responses that are further deleterious to periodontal health (21). Alternatively, these signs and symptoms may trigger sufficient motivation to institute healthy behaviors in an attempt to become more comfortable, halt or reverse the ravages of periodontal disease, and perhaps prevent further hard and soft tissue destruction and/or loss.

The consequences of behavioral pathogens, extending from neglect of oral hygiene to dietary inadequacies, poor sleep

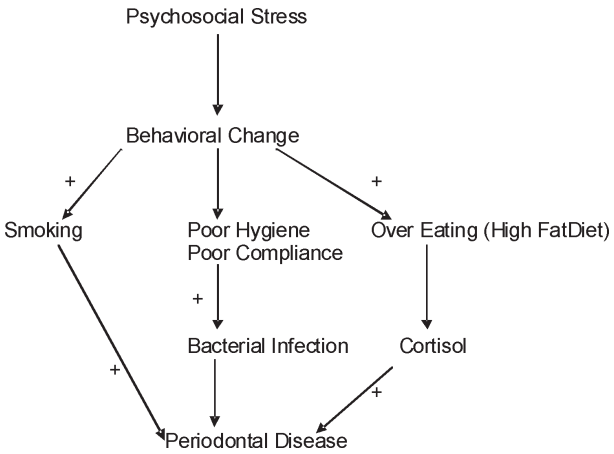

Figure 2: Psychosocial stress and its effect on behavior as manifested by alterations in periodontal disease

patterns, use of tobacco products and other substance abuse constitute an important class of psychosocial stressors that contribute to the 'vicious cycle' of increasingly severe forms of advanced periodontal inflammation and disease. The less common forms of periodontal disease – aggressive periodontitis, periodontal disease associated with diabetes – similarly are associated with a myriad of intra- and interpersonal stressors that are significant risk factors for exacerbating the underlying periodontal disease condition (14).

EVIDENCE FOR THE ROLE OF STRESS IN PERIODONTAL DISEASE

Monteiro *et al.* (15) distinguish between acute necrotizing ulcerative gingivitis (ANUG) and adult periodontitis, concluding that the evidence is strong for stress as a predisposing factor to ANUG, while the evidence for psychosocial factors as etiological agents in periodontitis is not as substantive.

In a study of gingivitis in 314 children aged 6–8years by Vanderas *et al* (22), levels of urinary catecholamines (epinephrine, norepinephrine and dopamine) were not related to gingivitis as recorded by a gingival bleeding index. Physical factors that included dental plaque levels and number of proximal decayed surfaces were significantly associated with gingivitis; when demographic factors and psychosocial stressors were added to the analyses, only mother's education carried any association to gingivitis, suggesting that for these young children, emotional stress may not be a contributor to developing gingivitis.

Deinzer *et al.* (23) conducted prospective observational study, assessing clinical signs of periodontal disease (such as gingival bleeding on probing) and diagnosed gingivitis. Severe deterioration in gingival health from baseline levels was observed significantly more frequently in a cohort after they had undergone a period of academic examinations compared to a peer-control group not experiencing such academic testing.

Deinzer *et al.* (24) also conducted experimental intervention study to assess the relationship between academic stress and gingival inflammation, assessing changes in interleukin- 1â, a component of the immune system thought to play a role in periodontal tissue destruction. Using a split mouth design, medical students voluntarily neglected oral hygiene of two quadrants for 21 days, to induce an experimental gingivitis in those quadrants, while maintaining high levels of oral hygiene in the remaining two quadrants. It was observed that examination students showed significantly higher levels of interleukin-1â at both the experimental gingivitis sites and the sites of good oral hygiene, indicating that stress may affect periodontal health through suppressed immune system activity, and that such a relationship would be more pronounced when oral hygiene was not maintained.

Renate *et al* (25) conducted a study to correlate relationship between psychological stress and periodontal disease. Twenty-six medical students participating in major examinations (Academic stress) and the same number of students not participating in any exam were the subjects. Bleeding on probing was assessed 4 weeks prior to examination period and last day of examination. They concluded that severe deterioration in gingival health was seen frequently in examination students. These results support the hypothesis that psychological stress is a significant risk factor for periodontal inflammation.

Kerr (26) observed that a greater number of ANUG occurred in students during the time of examination than any other time of the year.

Monteiro da Silva *et al* (27) compared groups of 50 subjects each with rapidly progressive periodontitis, chronic adult periodontitis and subjects without significant periodontal destruction. Statistical adjustments were made for smoking, alcohol use, oral hygiene, age and education. Psychosocial variables were related to the periodontal destruction. The RPP group showed significantly increased depression and loneliness compared with the other groups, suggesting that different types of periodontal disease are not equally associated with psychosocial factors. Yet, it is difficult to determine whether diagnosis and prognosis influenced the mental conditions or vice versa.

Moulton *et al* (28) conducted study on 22 patients and found that attack of ANUG was always precipitated by acute anxiety arising from life situations or conflicts about dependency and or sexual needs.

Page *et al* (29) reported that active phase of the disease was after associated with depression.

Shannon *et al* (30) conducted a study to investigate the possible relationship between stress, as measured by urinary steroid excretion rate, in 478 males being screened in military duty. The subjects were divided into 6 groups according to the condition of the gingival – mild gingivitis, moderate gingivitis, severe gingivitis, periodontitis, ANUG and normal control groups. The patients with ANUG showed increased advent corticosteroid activity in urine. The view stress is an etiologic factor in ANUG is further strengthened by adrenocortical activity. The increased activity of ANUG was seen in times of anxiety and stress suggests stress as an etiologic factor.

Clay and Bell (31) conducted a study on 11 patients presenting with ANUG, collected 24 hours urine sample before and after the course of ANUG. They measured 17 – Hydroxy corticosteroid content in urine which is physiological measure of stress. They concluded that patient had a significant high level of 17 – Hydroxy corticosteroid during the course of ANUG.

Davies *et al* (32) conducted a study on destructive form of periodontal disease in adolescent and young adults. They concluded rapidly progressive periodontitis was frequently associated with stress and depression.

Genco *et al* (33) analyzed data from 1400 people of 25 – 75 years age to find out if stress, distress and poor coping behaviors are risk factors of periodontitis. He concluded that people with financial stress had more severe periodontal disease.

Genco *et al* (34) concluded the evidence for a relationship between psychosocial stresses, coping in response to stress, and periodontal disease was also observed in a cross-sectional epidemiological study of 1426 adults, aged 25–74 years. Results indicated a significant role for financial strain in relation to greater alveolar bone and periodontal attachment loss, after adjusting not only for age and gender, but also for smoking. Interestingly, those individuals with a problem-solving coping style for managing the stressors of daily living fared better than those who exhibited a more emotionally focused and less adequate coping response to psychosocial strain. The pattern of results from this study suggests that effects of psychosocial stress on periodontal disease can be modulated by adequate coping behaviors.

Ability to cope with psychological stressors has been thought to influence onset and progression of periodontal disease because ineffectiveness of coping with the stress may lead to neglect in oral hygiene, yielding increased levels of the most common forms of adult periodontitis as well as exacerbating the less frequently occurring rapidly progressive periodontitis.

However, a study by Monteiro daSilva *et al.* (35) failed to confirm this commonly assumed link between type of periodontal disease, psychological stressors that included depression, anxiety, perceived stress and loneliness, and dental plaque as the major dependent variable. A greater prevalence of smoking was found in the rapidly progressive periodontitis group.

Croucher *et al.* (36) enrolled 100 clinic cases with at least one periodontal site with pocket depth exceeding 5mm and controls that had no periodontal pockets greater than 3mm. They found a significant relationship between major life events as measured by the Holmes and Rahe Social Readjustment Rating Scale and physical indicators of periodontal disease, notably, dental plaque level. The association between periodontitis and impact of negative life events remained statistically significant even after controlling for other variables, such as level of education, number of missing teeth and marital status, but did not remain significant after controlling for smoking.

For several decades, studies have been conducted to examine the possible association between personality traits and periodontal disease. Such studies are intuitively appealing, because they hypothesize a relationship between long-standing predispositions of people's psychological and emotional make-up and periodontal disease, which is also characterized by a long-standing clinical course (37).

In a study of therapy-resistant periodontitis (38), it was observed that patients not responding to conventional periodontal therapy, compared to a treatment responsive group, tended to show greater psychosocial strain and higher prevalence of a passive-dependent personality. Perhaps surprisingly, the responsive patient group showed a more rigid personality structure but with less self reported psychosocial stress.

By and large, studies examining personality characteristics and periodontal disease have not been rewarding and no clear pattern emerges favoring certain personality types, such as Type A, obsessive compulsive or neurotic. More recent attempts to develop clusters of personality traits that include coping styles, cognitive factors and emotional characteristics may represent a more fruitful path for investigating long-standing relationships between personality and periodontal disease.

Given the high variability associated with self-report measures of psychological stress (e.g. depression, anxiety, personality type, coping styles) it is likely that such small sample studies will be statistically underpowered to detect hypothesized relationships among psychosocial factors and indicators of disease. In addition, the use of different major dependent variables across studies – dental plaque levels, gingival bleeding on probing, pocket depth, attachment loss, loss of alveolar bone, etc. – makes comparisons across studies difficult and limits the ability to generalize about the specificity of the relationship between psychosocial stress and periodontal disease; some studies (36) use self report symptom checklists as indicators of periodontal disease in lieu of clinical measurements.

It is clear that findings are almost universally in the hypothesized direction of a meaningful relationship between parameters of periodontal disease, physical stressors, such as smoking and co-morbid diseases like diabetes, and psychosocial stressors, whether the latter is defined as psychological disturbance (e.g. depression, anxiety, loneliness) or job/ financial stress.

TREATMENT

Doctor needs to take a careful history to look for an underlying stress or psychological disorder that could be the source of patient's stress symptoms. Many times, a careful interview can be the best source of information about the cause of patient's symptoms. Patient should be referred to a psychiatrist for help.

There are four basic approaches to dealing with stress:

- Removal or alteration of the source of stress
- Learning to change how you see the stressful event
- Reducing the effect on your body that stress has
- Learning alternative ways of coping

Jacobson's Progressive muscle relaxation (JPMR), Breathing exercises and Guided imagery are simple ways of relieving stress and achieving wellbeing as a whole.

CONCLUSION

Stress also results in delayed healing of the connective tissues and bone in artificially induced gingival wounds but does not affect the epithelium. In chronic stress osteoporosis of alveolar bone, apical migration of the junctional epithelium and formation of periodontal pocket occurs. For clinicians, it is important to recognize patient who are in stress and to be able to advise patients about the possible effects of stress on periodontal disease if the level of stress cannot be lowered. In addition all other risk factors for periodontal disease should be minimized, promoting good oral hygiene and smoking cessation.

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