

PERIODONTAL DISEASE AND STRESS: A REVIEW

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ABSTRACT:

Stress and depression are more common in subjects who experience a high financial strain along with inadequate coping strategies and more plaque accumulation. Stress and depression associated periodontal disease may be related to psychoneuroimmunologic changes, at-risk health behavior, or a combination of both. Stress is an association of physiological and psychological reactions of a person confronted to a change of situation. The relationship between stress and any disease is explained by hormonal modifications and behavioural changes induced by the stress. Research has suggested that stress and depression are two factors that play a role in the development and progression of periodontal disease. It is not clear, however, whether these factors lead to periodontal disease through physiological or behavioural changes, or by some combination of the two. The purpose of the present review article is to explore the associations between psychological factors, psycho immunologic variables, behaviour, and clinical measures of periodontal disease.

Key words: Stress, Depression, Periodontal Disease.



INTRODUCTION:

Periodontitis is an inflammatory disease caused by periodontopathic bacteria in the dental biofilm, leading to destruction of the tooth supporting tissues. Systemic diseases, habits, social factors, and psychologic stress are considered risk factors influencing disease onset and progression.^[1] Research has also suggested that stress, depression, and ineffective coping may contribute to the development of periodontitis.^[2] Stress is an association of physiological and psychological reactions of a person

confronted to a change of situation. The relationship between stress and any disease is explained by hormonal modifications and behavioural changes induced by the stress. Research has suggested that stress and depression are two factors that play a role in the development and progression of periodontal disease.^[3] Psychological stress, particularly if sustained over an extended period of time, can have deleterious effects on the body, representing an important example of the mind-body interaction. The role of psychological factors in periodontal

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disease has a significant history. Currently, stress is classified as a risk indicator for periodontal disease.^[4]

DEFINITION OF STRESS:

The term stress is used loosely, in layman terms, to describe adverse emotions or reactions to unpleasant experiences the term Stress has a precise physiological definition. It is a state of physiological or psychological strain caused by adverse stimuli, physical, mental, or emotional, internal or external, that tend to disturb the functioning of an organism and which the organism naturally desires to avoid. Thus, stress can be viewed as a process with both psychological and physiological components.^[4] A stressor is any stimulus, situation or circumstance with the potential to induce stress reactions. Whether or not a subject exhibits a stress response depends on a myriad of factors, including coping behaviors, genetic predisposition, concomitant stressors, levels of social support, and other lifestyle factors. Potential effects of the stress response that may be observed, or even measured, include anxiety, depression, impaired cognition, and altered self-esteem.^[5] Definitions of stress differ in the periodontal literature, as seen by the variety of ways in which stress is evaluated for example, subjective measurements of so-called stressful situations, to detailed questionnaires, to measures of specific markers such as plasma cortisol. Selye (1936) coined the term "Stress". 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 by the activation of the adreno – cortico – pituitary axis.^[6]

TYPES OF STRESS

Primary Categorization of Stress: Stress can be primarily divided into the following two types:

1. **Negative Stress:** Negative stress is considered as a causative factor in minor conditions like ulcers, digestive problems, skin complaints, headaches, and insomnia. Spiritual, mental, and physical health can be very badly harmed due to excessive, prolonged, and unrelieved stress.
2. **Positive Stress:** Stress not only has negative effects on an individual, but there are some positive effects of stress as well that affect a person by stimulating awareness and motivation that provides the urge effectively deal with challenging circumstances. When confronting threatening situations, it is the positive stress that provides the sense of alertness and urgency that are needed for effectively tackling of the circumstances.

Secondary Classification of Stress: Stress is secondarily classified into the following four

types: **Eustress:** This secondary type of stress belongs to positive stress, one of the two primary types of stress as, although a short-term stress, it provides immediate strength. Increased creativity, enthusiasm, and physical activity are the reasons eustress is experienced by an individual. **Distress:** Belonging to the negative stress, feelings of unfamiliarity and discomfort are brought by distress. This secondary type of stress gets further categorized into the following two types. Acute Stress, which is a quickly arriving and disappearing intense stress, and Chronic Stress, which exists for weeks, months, or even years. **Hyper-stress:** This secondary type of stress is suffered by people who are pushed beyond what they can tackle. Even little things can trigger a powerful emotional reaction when someone is hyper-stressed. **Hypo-stress:** Opposite of hyper-stress, hypo-stress occur to the individuals who are unchallenged and bored. Uninspired and restless are the conditions suffered by people who undergo hypo-stress.

More Types of Stress:

1. Post traumatic stress
2. Work stress
3. Stress incontinence
4. Teen stress
5. Job stress
6. Life stress
7. Stress headache
8. Oxidative stress

9. Emotional stress

Acute vs chronic stress

Acute stress refers to the sudden and temporary incidence of a stressor whereby a definite onset and offset is present. This short term stressor causes the body to activate a fight or flight response via the sympathetic nervous system and can be viewed as an alarm safety mechanism to keep individuals alert and prepared to avoid danger and harm. Chronic stress refers to an ongoing, intermittent or a series of stress sequences over a continuous period of time whereby individuals face continual stress without relief between such episodes. Such long term stressors cause more prolonged physiological responses as a result of steroid hormone secretion and result in exhaustion.^[7]

Short term physiological effects of stress (fight or flight)

A portion of the brain to activate the adrenal medulla via the autonomic nervous system (ANS), stimulates the sympathetic division which regulates involuntary activities of smooth muscles, cardiac muscles and glands (Marieb & Hoehn, 2007). The sympathetic division initiates a sequence of physiological changes within the body as it generates the secretion of adrenalin and noradrenalin.

Long term physiological effects of stress – exhaustion

Continued exposure to stressful stimuli generates a more moderate physiological response, causing the hypothalamus to activate the adrenal cortex via hormonal

signals (secretion of steroid hormones). Extensive continued exposure to stress can cause adaptation resources to be depleted and thus potentially leads to exhaustion (Marieb & Hoehn, 2007).^[8]

MOLECULAR AND ENDOCRINE MECHANISMS OF THE STRESS RESPONSE:

Stress can result in the degeneration of the immune system, mediated primarily through the hypothalamic – pituitary – adrenal and sympathetic adrenal medullary axis. The stress-induced responses are transmitted to the hypothalamic/pituitary/adrenal (HPA) axis to promote the release of corticotropin-releasing hormone (CRH) from the hypothalamus and glucocorticoids from the adrenal cortex. Stress perceived by the brain stimulates the hypothalamus to produce CRH, which is released into the hypophyseal portal system, activating the pituitary gland to release adrenocorticotropic hormone (ACTH), which in turn induces release of corticosteroids from the adrenal cortex.^[9]

Glucocorticoids, including cortisol, exert major suppressive effects through highly specific mechanisms at multiple levels. For example, in vivo glucocorticoids reduce the number of circulating lymphocytes, monocytes, and eosinophils. They also inhibit the accumulation of eosinophils, macrophages, and neutrophils at inflammatory sites. At the molecular level, glucocorticoids profoundly inhibit important functions of inflammatory cells including macrophages, neutrophils, eosinophils, and mast cells in functions such

as chemotaxis, secretion, and degranulation.^[10]

Glucocorticoids also inhibit the cascade of the immune response by inhibiting macrophage-antigen presentation, lymphocyte proliferation, and lymphocyte differentiation to effector cell types such as helper lymphocytes, cytotoxic lymphocytes, natural killer cells, and antibody-forming B cells. Corticosteroids also inhibit production of cytokines including IL-1, IL-2, IL-3, and IL-6, tumor necrosis factor, interferon gamma, and granulocyte and monocyte colony stimulating factors. Glucocorticoids inhibit arachidonic acid-derived proinflammatory mediators such as prostaglandins and leukotrienes. Glucocorticoids also induce endogenous anti-inflammatory proteins and lipocortins, which have the capability of inhibiting phospholipase A2, thereby inhibiting generation of eicosanoids. Hence, the stress-related stimulation of the HPA axis with the production of glucocorticoids such as cortisol has major suppressive actions on immune and inflammatory responses. This represents the major effector arm of the CNS-hormonal axis. There is also an afferent or feedback arm consisting of stimulation of the HPA axis by cytokines.^[11]

Glucocorticosteroids, including cortisol, then depresses immunity including secretory IgA, IgG, and neutrophil functions, all of which may be important in protection against infection by periodontal organisms. Secretory IgA antibodies may protect by reducing initial colonization of periodontal pathogens. IgG antibodies may exert protection by opsonizing periodontal

organisms for phagocytosis and killing by neutrophils. ^[11] This then gives rise to increased susceptibility, which leads to the establishment of periodontal infection which, in turn, results in destructive periodontitis. Periodontitis is brought about by tissue-destroying factors such as IL-1 and matrix metalloproteinases activated by the periodontal pathogens, as well as by the direct effects of pathogenic bacteria.¹¹ The second major pathway to be activated is the sympathetic nervous system. Stress induced activation of the hypothalamic nervous system in the release of highly active hormone such as catecholamines. These catecholamines released during stress, contributes to the development of hyperglycemia by directly stimulating glucose production and interfering with the tissue disposal of glucose.^[4] Catecholamines are known to alter the blood flow. Peripheral vasoconstriction may affect important oxygen-dependent healing mechanisms, such as angiogenesis, collagen synthesis and epithelialization. The release of Catecholamines results in hormonal secretion of norepinephrine from the adrenal medulla, which results in a range of effects that may act to modulate immune responses. Increased sympathetic stimulation can also act to decrease salivary secretions typically experienced as anxiety induced dry mouth. Stress that is associated with immune challenge has been called immune stress or inflammatory stress.^[4]

Depression is reliably associated with relationship conflict and lower social support, providing one psychological mechanism through which close relationships influence inflammation. There

is a robust association between inflammation and depression not only in clinically depressed samples, but also in community-based samples; some non-replications may be due to confounding factors such as body mass index (BMI) or medication use. Moreover, there is some evidence that depression may sensitize the inflammatory response, thus effectively promoting larger cytokine increases in response to stressors or antigen challenge. Additionally, depression and stress contribute to a greater risk for infection, prolonged infectious episodes, and delayed wound healing; all processes that can fuel sustained pro-inflammatory cytokine production. Furthermore, depression alters inflammation-relevant health behaviors; for example, disturbed sleep, a common correlate of depression and stress, promotes IL-6 production. ^[12]

Psychologic stresses can downregulate the cellular immune response in three different ways: 1) the hypothalamo-pituitary-adrenal (HPA) axis; 2) the peripheral release of neuropeptides; and 3) the sympathetic nervous system (SNS) via the release of adrenaline and noradrenaline. The psychoneuroimmunologic model tries to link stress and periodontitis via mentally triggered alterations of immunologic responses. It is assumed that periodontitis is negatively influenced by inappropriate coping behavior under stress, which leads to a centrally mediated immune suppression.^[1]

POTENTIAL PSYCHONEUROIMMUNOLOGIC MECHANISMS:

Stress and depression-associated periodontal disease may be related to psycho neuroimmunologic changes, at-risk health behavior, or a combination of both. The relationship between stress or depression and immune-regulated inflammation is complex. Negative emotions prompt the release of polypeptides from the sympathetic noradrenaline-transmitting and sensory peptidergic nerve fibers and from endocrine glands. In turn, these hormones help to regulate immune responses triggered by bacterial antigens.

In particular, the hypothalamus releases corticotropin-releasing hormone, which then stimulates the release of adrenocorticotropic hormone from the pituitary. In response, the adrenal cortex produces CORT, a GC that helps to regulate inflammatory responses and lymphocytic activity. Short-term elevations in CORT reduce inflammation and mobilize immune components. However, over the long-term, Glucocorticosteroids may reduce immunocompetency through inhibition of immunoglobulin A (IgA), immunoglobulin G (IgG), and neutrophil function. IgA antibodies reduce the initial colonization of periodontal organisms, and IgG antibodies may make periodontal pathogens more vulnerable to phagocytosis by neutrophils. There is also evidence that longer-term elevations in CORT may be associated with chronic inflammation because the GC loses its ability to inhibit inflammatory responses initiated by the immune system. Therefore,

depressed immunity and chronically elevated CORT may result in inflammation and more destructive periodontitis.¹³

MENTAL STRESS RESPONSE TRIGGERING THE HPA AXIS WITH IMMUNOSUPPRESSIVE EFFECTS:

Glucocorticosteroids, including cortisol then depress immunity including secretory IgA, IgG, and neutrophil functions, all of which may be important in protection against infection by periodontal organisms. Secretory IgA antibodies may protect by reducing initial colonization of periodontal pathogens. IgG antibodies may exert protection by opsonizing periodontal organisms for phagocytosis and killing by neutrophils. This then gives rise to increased susceptibility, which leads to the establishment of periodontal infection which, in turn, results in destructive Periodontitis. Periodontitis is brought about by tissue-destroying factors such as IL-1 and matrix metalloproteinases activated by the periodontal pathogens, as well as by the direct effects of pathogenic bacteria. Mental as well as physical stress can also result in responses being transmitted to the autonomic nervous system and then to the adrenal medulla, resulting in secretion of catecholamines such as epinephrine and norepinephrine. Catecholamines then affect Prostaglandin and proteases, which in turn, could enhance periodontal destruction. ^[14]

HEALTHY RESPONSE TO STRESS:

The brain handles (mediates) the immediate response. This response signals the adrenal medulla to release

norepinephrine. The hypothalamus (a central area in the brain) and the pituitary gland initiate (trigger) the slower, maintenance response. This response signals the adrenal cortex to release cortisol and other hormones. Many neural (nerve) circuits are involved in the behavioral response. This response increases arousal (alertness, heightened awareness), focuses attention, inhibits feeding and reproductive behavior, reduces pain perception and redirects behavior.

The combined results of these three components of the stress response maintain the internal balance (homeostasis), increase energy production and utilization, and alter electrolyte (chemical elements) and fluid balance. The sympathetic nervous system operates by increasing the heart rate, increasing blood pressure, redirecting blood flow to the heart, muscles, and brain and away from the GI tract, and releasing fuel (glucose and fatty acids) to help fight or flee the danger.^[15]

STRESS AND IMMUNITY:

Stress and its biochemical mediators may modify the immune response to microbial challenge which is an important defense against inflammatory periodontal disease. Under stress the release of adrenaline and nor adrenaline may not only induce a decrease in the blood flow but possibly also in those blood elements necessary for maintaining resistance to microbes. There is increasing evidence that the central nervous system (CNS) can influence the immune response via a complex network of bidirectional signals

linking the nervous, endocrine, and immune systems.^[9]

Psychoneuroimmunology (PNI) is an emerging field concerned with the influence of behavior on brain-immune interactions, Neuroendocrine hormones released from the pituitary gland by the activation of the HPA axis influence the immune system. Both lymphoid and myeloid cells express receptors for these hormones and neuropeptides, and lymphocytes can also synthesize hormones such as prolactin, growth hormone, and adrenocorticotrophic hormone (ACTH). Furthermore, neurohormones e.g. glucocorticoids, and peptides such as ACTH, endorphins and somatostatin are able to modulate many aspects of the immune response, including cytokine production, chemotaxis of monocytes and neutrophils and natural killer (NK) cell activity.

These bidirectional pathways between the CNS and immune system provide a feedback mechanism for immune regulation. For example, **IL-1 released** from activated immune/inflammatory cells **stimulates** the hypothalamus to produce **more corticotrophin-releasing hormone (CRH)**, which in turn, triggers the release of two “stress” hormones, ACTH from the pituitary gland and corticosterone from the adrenal cortex. These stress hormones can downregulate immune responses. Thus, physical or psychological stressors can activate the CNS and HPA axis to release catecholamines and glucocorticoids that alter these feedback mechanisms and disrupt homeostasis.^[16]

MECHANISM OF ACTION OF GLUCOCORTICOIDS:

1. Transactivation

Glucocorticoids bind to the **cytosolic glucocorticoid receptor (GR)**. This type of receptor is activated by ligand binding. then newly formed receptor-ligand complex **translocates itself into the cell nucleus**, where it binds to **glucocorticoid response elements (GRE)** in the promoter region of the target genes resulting in the **regulation of gene expression**. This process is commonly referred to as transactivation. The proteins encoded by these up-regulated genes have a wide range of effects, for example: **Anti-inflammatory** – lipocortin 1, p11/calpactin binding protein and secretory leukoprotease inhibitor 1 (SLPI). Lipocortin-1 suppresses phospholipase A2, thereby blocking eicosanoid production, and inhibits various leukocyte inflammatory events (epithelial adhesion, emigration, chemotaxis, phagocytosis, etc.) also inhibit the two main products of inflammation, prostaglandins and leukotrienes

2. Transrepression

The opposite mechanism is called transrepression. The activated hormone receptor interacts with specific transcription factors (such as NF- κ B) and prevents the transcription of targeted genes. Glucocorticoids are able to prevent the transcription of **proinflammatory genes**, including interleuki

ns IL-1B, IL-4, IL-5, and IL-8, chemokines, cytokines, and TNF α genes.

ALTERATION OF THE T HELPER 1 CELL/T HELPER 2 CELL RATIO

It has been well established that periodontal disease is a result of an inappropriate host response to infecting microorganisms. The host response may be divided into innate and adaptive immunity. Innate immune mechanisms operate without any previous exposure to the pathogenic organism. Innate immunity includes physical barriers, various cell populations (such as neutrophils, monocytes and natural killer cells), complement, and a large number of antimicrobial peptides. Adaptive immunity is a highly specific response that is amplified on exposure to specific pathogens. It consists of humoral and cell mediated immunity. The nature of the adaptive immune response, in particular the nature of lymphocytes involved in immunity, may partly explain the differences in host response between individuals.^[4]

Breivik and co-workers have proposed a model in which the extent of inflammatory periodontal disease maybe predicted by reactivity of the hypothalamic–pituitary–adrenal axis and its effects on T-lymphocyte numbers . Helper T lymphocytes can be divided into two subpopulations – T helper 1 cells and T helper 2 cells – based on their cytokine production. T helper 1 cells stimulate cellular immunity through the production of interferon- γ and interleukin-2. T helper 2 cells promote B cell

differentiation and humoral immunity through the release of interleukin-4, interleukin-5, interleukin-6 and interleukin-10.^[16]

Numerous in vitro and animal studies support the contention that increased levels of plasma glucocorticoid may provoke an inappropriate T helper 2 cell response. Marshall and coworkers demonstrated a cytokine profile, consistent with a T helper 2 cell response, in medical students during stressful examination periods. It has been hypothesized that a dominant T helper 2 cell response increases susceptibility to infectious diseases. In fact T helper 1 cell responses may actually be protective against periodontitis, whereas a T helper 2 cell response may increase periodontal breakdown. In order to test this hypothesis, two genetically distinct types of rat, which differed in response to stress, were compared.^[16,17]

One group of rats were high cortisone-responding rats; the other were low cortisone-responding rats. Ligature-induced periodontitis was produced in the maxillary right second molars of all rats and was measured using digital radiographic and histological examination. Previous work has demonstrated that in comparison to low cortisone-responding rats, the high cortisone-responding group generates a stronger T helper 2 cell response to infectious agents. The high cortisone-responding rats exhibited greater alveolar bone loss and more loss of connective tissue fibers. These data lend weight to the contention that a shift to the T helper 2 cell immune response may be more

destructive. These findings have more recently been refuted.^[17] Using a mouse model, *P. gingivalis* was implanted subcutaneously, and the immune response was monitored in two groups undergoing restraint or isolation stress and in a control stress free group. Chamber exudates and serum was collected and analyzed for antibodies to *P. gingivalis*. The results showed a lower immunoglobulin G1/immunoglobulin G2 ratio, which indicated an elevated T helper 1 cell response during the stressful conditions. The above studies confirm the contention that an altered T helper 1 cell/T helper 2 cell lymphocyte profile, resulting from chronic stress, can exacerbate periodontal disease in animal models. This remains to be confirmed in human subjects.^[17]

Glucocorticoids suppress immunity^[9]

Glucocorticoids suppress cell-mediated immunity by inhibiting genes that code for the cytokines IL-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-8 and IFN- γ , the most important of which is IL-2. Smaller cytokine production reduces the T cell proliferation. Glucocorticoids also suppress the humoral immunity, thereby causing a humoral immune deficiency. Glucocorticoids cause B cells to express smaller amounts of IL-2 and of IL-2 receptors. This diminishes both B cell clone expansion and antibody synthesis.

IMPACT OF STRESS ON LATENT HERPES VIRUSES

Recurrent herpes simplex virus infections are common sequelae of surgical and other traumatic procedures in the oral cavity. Unlike common cold viruses, which often

cause acute infections and are effectively eliminated by the immune system, herpes viruses latently infect target cells where the viral genome resides for life. In individuals who are immunosuppressed due to medical treatments (i.e., organ trans-plantation), or immunosuppressive diseases, one or more latent herpes viruses are commonly reactivated. In severely immune-compromised patients, these viruses can induce severe complications and death. Among normal individuals, reactivation of one of the herpes viruses may result in a clinical outcome, e.g., a cold sore caused by HSV-1. However, evidence for the reactivation of a latent herpes virus may be more subtle, i.e., a rise in the IgG class of antibodies to various viral proteins in the absence of clinical symptoms or infectious virus. It is believed that the loss of immunological control over viral latency is associated with changes in the cellular immune response. It is the increase in viral antigens synthesized after reactivation which induces memory B cells to increase antibody production.

Several studies also show that pharmacological and, more importantly, physiological levels of glucocorticoid hormones can reactivate certain latent herpes viruses from virus genome-positive cells in vitro. Increases in glucocorticoids associated with stress can result in some degree of suppression of the cellular immune response. Thus, reactivation of latent herpes viruses could be the result of a combination of immunosuppression and changes in the steady state expression of the latent virus induced directly by the hormones. Varicella-zoster virus (VZV),

another herpes virus, is the causative agent of the acute primary disease varicella (chicken pox) and also the painful vesicular disease called shingles (herpes zoster). Zoster is caused by VZV reactivation, and the incidence of zoster greatly increases with age and immunosuppression.⁴⁶ Stress has also been reported as a risk factor for reactivation of VZV and development of zoster, especially in older adults.^[18]

POSSIBLE MECHANISMS OF ACTION OF PSYCHOSOCIAL FACTORS ON PERIODONTAL TISSUE

Neglect of oral hygiene :It is obvious that proper oral hygiene is partially dependent on the mental health status of the patient. Some may be so disturbed or distracted psychologically that personal hygiene is neglected. Other patients may intentionally ignore oral hygiene to fulfill deep neurotic needs.

Moulton and Theiman (1952) suggest that oral hygiene may be neglected during depression and deep anxiety. Dependent individuals may exhibit chronic neglect as if they were expecting such care to be the responsibility of others.^[19]

Changes in Dietary Intake: Emotional conditions are thought to modify dietary intake. Thus indirectly affecting periodontal status (Miller 1947). 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^[20]

Smoking and other Harmful Oral Habits:

Smoking potentially acts by affecting tissue moisture or temperature. Smoking according to some studies is related to NUG and probably to other oral disease as well (Goldhaber and Giddon 1969) Smoking is also inversely related to many psychosocial variables associated with mental health.^[21] Pindborg (1951) reported that 98% of his ANUG patients were smokers and that the frequency of ANUG increased with an increasing exposure to tobacco smoke. It has not been established whether this correlation occur because tobacco smoke has a direct toxic effect on the gingiva, vascular or other changes are induced by nicotine or other substance, smoking and ANUG are both reflections of stress.^[22]

Kenney et al (1988) mentioned that the circulating nicotine have following effect.

i Vasoconstriction, produced by the release of Adrenaline and nor adrenaline which is supposed to result in lack of nutrients for the periodontal tissue.

ii. Suppression of invitro secondary antibody responses

iii. Inhibition of oral neutrophil function.^[23]

Bruxism:

Bruxism is the clenching or grinding of the teeth when the individual is not chewing or swallowing .Bruxism can occur as brief, rhythmic strong contraction of the jaw muscles during eccentric lateral jaw movement or in maximum intercuspation, which is called clenching Bruxism often occurs without any neuralgic disorders or defects and can be viewed as a

phenomenon present in healthy individuals. However bruxism may lead to tooth wear, fractures of the teeth or dental restorations or uncosmetic muscle hypertrophy. The patients may be completely unaware of these repeated and sustained forced contacts of the teeth that seem to have no functional significance in humans (Parafunction).Bruxism may be interrelated in a psychophysiologic manner. Tooth contact lasting up to 17 seconds have been reported concomitant with alterations in pulse rate and stroke. These periods of bruxing contact were directly related to periods of emotional reaction

Gingival circulation:

The tonus of the smooth muscle of blood vessels may be altered by the emotions by way of the ANS. For example, Prolonged contraction could alter the supply of oxygen and nutrients to the tissues. They found a lower ability of the tissues of rats under stress to utilize oxygen. However according to Rugh et al., (1984), this proposed mechanism remains obscure because did not perform detailed metabolic analysis.²⁴ Furthermore, smoking and stress have been implicated in reducing gingival blood flow which in turn, could increase the possibility of necrosis of tissues, with subsequent reduced resistance to plaque (Clarke et al 1981).^[25]

Alteration in saliva flow and components:

Psychologic factors are known to influence the rate of secretion and composition of Saliva. Saliva in turn, relates to plaque formation, calculus deposition, and antibacterial and proteolytic activities all of

which may have a bearing on periodontal disease.

These relationships between salivary physiology and psychologic status do not necessarily demonstrate causation of periodontal disease, but they show a pathway in which periodontal health is influenced by salivary changes

Endocrine changes: It has been known that stress can affect the endocrine system. Although interaction between stress-endocrine-periodontal changes are not yet well understood. Some hypothesis have been proposed. Moulton et al (1952) suspected the periodontal status is related to alterations in the concentration of adrenal corticoids and other hormones involved in the general adaptation syndrome. Moulton et al (1952) hypothesized stress can alter pituitary function and subsequently influence carbohydrate and calcium metabolism, affecting the mouth. Some studies indicate that glucocorticoids released under stress may have some role in the pathogenesis of ANUG.

Stress induced hormones in the gingival crevicular fluid could provides a nutrient that favors the subgingival growth of pathogenic microbial populations ^[19]

Lowered host resistance: Stress and its biochemical mediators may modify the immune response to microbial challenge, which is an important defense against inflammatory periodontal disease under stress, release of adrenaline and nor-adrenaline may not only induce a decrease in blood flow, but possibly also in those

blood elements necessary for maintaining resistance to disease related microbes.

It may be that glucocorticoids released during stress prolong these vascular response (Meyer 1989).^[15]

CONCLUSION:

The role of stress in human periodontal disease has a plausible pathophysiological basis. Evidence has suggested that stress is associated with more severe periodontal disease, as well as poorer healing responses to traditional periodontal therapy. This is because stress can cause behavior modification (e.g. smoking, alcohol abuse, etc.) and immunosuppressive effects (e.g. decreased polymorphonuclear leukocyte function, altered T helper 1 cell/T helper 2 cell ratio, etc.), which may result in greater severity of periodontal disease as well as delayed wound healing.

This suggests that stress management may be a valuable component for current periodontal practice. However, at present, the majority of the literature consists of case series and retrospective studies. There are even fewer studies dealing with the role of stress and periodontal wound healing. Thus, the exact role of psychological factors in periodontal wound healing remains to be elucidated, and further well controlled, prospective clinical trials are warranted.

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