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Research Article

ROLE OF STRESS IN THE PATHOGENESIS OF PERIODONTITIS

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ABSTRACT

Periodontitis is a chronic inflammatory disease caused by specific bacteria (periopathogens). These bacteria act as triggering factors but host defence mechanisms seem to be responsible for most of the tissue damage and progression of the diseases. The host immune response is influenced by systemic and local factors. For centuries an interrelationship between stress and periodontal disease has been suggested. Direct association between increased susceptibility of periodontal disease and stress remains to be proven. Stress modulates the immune response by biological mechanism (activation of central and autonomic nervous system) and behavioural mechanism. Several stress markers are found in patients with periodontal diseases in blood and saliva. The aim of this review is to provide an insight into the relationship between psychological stress and periodontal diseases.

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INTRODUCTION

The etiopathogenesis of periodontal disease indicates that periodontitis, a multifactorial disease caused by periopathogens in which host and environmental factors play an important role. Bacteria plays an essential role as primary etiological agents but alone seem to be insufficient to explain occurrence or progression of the disease. Periodontal disease onset and progression is influenced by various systemic disease, environmental factors, and psychologic stress that have the potential to alter periodontal tissues and host immune response, resulting in more severe periodontal destruction¹.

Stress is present universally as a part of the human condition in varying degrees and with different effects on different individuals². Stress affects almost all in some way or the other. Currently, stress is classified as a 'risk indicator' for periodontal disease^{3,4}. Stress also delays wound healing and negatively influences the outcome of periodontal therapy.

The term stress has been coined by Hans Seyle who defined it as the sum of all non-specific changes caused by function or damage, including the biologic phenomenon necessary for reestablishment of normal resting state. The term 'STRESS' originates from a latin word "Stringere" which means 'tight', 'strained'. Stress is defined as '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

process with both psychological and physiological components⁵.

Stressors and Coping

Stressors are forces that had the potential to challenge the adaptive capacity of the organism as suggested by Seyle in 1976. He recognized that stressors acting to produce changes in the body could be positive (e.g. exciting, pleasurable), leading to a response state he defined as 'eustress', or stressors could be negative, threatening homeostasis with pain, discomfort and physical pathology. He defined the negative response state as 'distress'⁶.

Stressors (either physical or emotional) are capable of inducing prolonged inflammatory and classic stress syndromes, and also capable of activating the stress system, along with associated immune system effects.⁷

Coping is the response of the individual to stress. *Dewe & Guest* (1990) and *Folkman* (1984) defined coping as "the cognitive and behavioural efforts used to master, reduce or tolerate the internal and external demands that are created by the stressful transaction."

In 1966, *Lazarus* identified two forms of coping: direct action and palliative. In 1984, *Lazarus and Folkman* changed the names of these two forms to:

1. problem-focused and
2. emotion-focused, respectively.

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Problem-focused efforts can be directed outward to alter some aspect of the environment or inward to alter some aspect of self. Emotion-focused coping strategies are directed toward decreasing emotional distress⁸.

Models of Stress

The two proposed mechanistic links proposed to explain the association between stress and periodontal disease are:

MODEL 1: Stress induced immunosuppressive effects-Biological mechanism

These changes are brought by two mechanisms:

1. Through activation of central nervous system
2. Through activation of autonomic nervous system

A direct role of psychosocial stress and coping behaviors via central nervous system

Genco *et al.* in 1998 offered a schematic model demonstrating that psychosocial stressors may play in starting a cascade of events leading to the activation of the central nervous system. According to this model, the central nervous system and hypothalamus are activated by psychosocial stress to release corticotrophin releasing hormone (CRH). CRH then stimulates the release of adrenocorticotropin hormone (ACTH) from the pituitary, which in turn results in production of cortisol by the adrenal cortex. Glucocorticosteroids, including cortisol, depress immunity by inhibiting the production of secretory immunoglobulin A and G, and various neutrophil functions. This results in unchecked bacterial proliferation due to impaired defense in periodontal tissues leading to destructive periodontal disease.

A direct role of psychosocial stress and coping behaviors via autonomic nervous system

The autonomic nervous system activated by stress, leads to the secretion of catecholamines (epinephrine and norepinephrine). The catecholamines in turn then affect prostaglandins and proteases secretion, that enhances periodontal tissue breakdown.

Model 2: Stress-induced behavioural changes-Behavioural mechanism

In this model, it is hypothesized stress leads to behavioural changes such as overeating, especially a high-fat diet, which then can lead to immunosuppression through increased cortisol production in at-risk health behaviours such as smoking, poor oral hygiene, and poor compliance with dental care. These changes can act as confounding factors in the periodontal tissue breakdown.

Stress Markers

Systemic and oral health status can be monitored by using blood and saliva. There are numerous stress markers produced at different aspects of stress response which influence the genesis and development of periodontal disease.⁹ These markers generally act as hormonal mediators and play a role in the ability of stress to promote disease by influencing the host response. They include:

Catecholamines: Psychological stress factors leads to elevated levels of catecholamines in blood. Salivary CAs may be a

useful index of sympathetic adrenomedullary system activity (SAM)¹⁰.

Cortisol

Cortisol predominantly present in blood in bound form to plasma protein and is also present in free form can be detected in saliva, blood and gingival crevicular fluid(GCF).

Chromogranin A

It is a novel stress marker in saliva and can act as a useful index of psychological stress. Chromogranin A, acidic phosphorylated secretory glycoprotein that is stored and released by exocytosis with CAs from the adrenal medulla and sympathetic nerve endings as well as by serous and ductal cells of human submandibular gland.

Neuropeptides

Neuropeptides are found within neural tissue. Substance P, may be of particular interest for immune reactions in the gingiva and the periodontium when triggered by dental plaque bacteria (Breivik *et al.*, 1996). Substance P is stimulatory at low concentrations and inhibitory at high concentrations. It is detected in gingival tissues and gingival crevicular fluid in higher levels in periodontitis patients than healthy individuals. Its release is regulated by noradrenaline. Continued long lasting stress increases its release which promote tissue damage and destruction of alveolar bone, resulting in chronic periodontal diseasedue to imbalanced inflammatory reactions

Salivary alpha-amylase

One of the major salivary enzymes secreted in response to sympathetic stimuli in humans. It is an indirect indicator of (autonomic nervous system) activity that displays its inhibitory activity against microorganisms.

Measurement of Stress

In the absence of a gold standard measurement of stress, modern scientists adopt three approaches of stress assessment:

1. The environmental approach referring to the occurrence of demanding events (stressors),
2. The psychological approach meaning the perceived by the individual stressfulness of each stressor and
3. The biological approach that focuses on the biological elements of the stress response¹¹.

Questionnaires and interviews are the main measurement tools of the first two approaches and biomarkers of the biological one. Questionnaires and interviews include

1. Holmes and Rahe scale,
2. Perceived stress scale.
3. Personal stress navigator scale
4. Beck depression inventory scale
5. Self-report psychiatric screening questionnaire –20 scale

Biological approach includes the estimation of biomarkers like cortisol, chromogranin A, salivary alpha amylase, neuropeptide levels.

Management of Stress

Stress management refers to the wide spectrum of techniques and psychotherapies aimed at controlling a person's levels

of stress, especially chronic stress, usually for the purpose of improving everyday functioning.

Careful history should be taken to look for an underlying stress by the doctor and referred to the specialist psychiatrist for help. There are four basic approaches to dealing with stress¹²:

1. Removal or alteration of the source of stress
2. Learning to change how you see the stressful event
3. Reducing the effect on your body that stress has
4. 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, distress and coping behaviours are regarded as important indicators for periodontal disease⁴. Acute stress conditions are immune-enhancing while chronic stress is immunosuppressive. Non-specific tissue reactions are produced by continuous exposure to stress, but how exactly it affects is not fully understood. 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 influence the periodontal tissue destruction, tissue healing and periodontal therapy outcome. Determining patients under chronic stress could develop specific multidisciplinary treatment strategies for ultimate effects of coping with stress factors and improvement of prognosis and outcome of periodontal treatment. This suggests that stress management may be a valuable component for current periodontal practice.

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