Stress and periodontal health

A project submitted to the scientific committee of the department of periodontics, college of Dentistry, University of Baghdad, in partial fulfillment of the requirement for the B.D.S.

Done by

Ahmed Husham Adnan

Supervised by

Dr. Hadeel Mazin

B.D.S, M.Sc. Periodontics

Baghdad- Iraq

2023

بسم الله الرحمن الرحيم

صدق الله العظيم

Certification of the Supervisor

Certification of the Supervisor

I certify that this project entitled **"Stress and periodontal health"** was prepared by **Ahmed Husham Adnan** under my supervision at the College of Dentistry/University of Baghdad in partial fulfilment of the graduation requirements for the Bachelor degree in dentistry.

Supervisor's name

Dr. Hadeel Mazin

DEDICATION

TO MY BELOVED FAMILY THAT HAS ALWAYS SUPPORTED AND STOOD BY ME

TO MY BEST FRIEND MOHAMMAD MAHDI THAT I WOULDN'T BE ABLE TO GO THROUGH COLLEGE WITHOUT HIM

THANK YOU

Acknowledgement

*I would like to express my deep gratitude and gratefulness to my supervisor **Dr. Hadeel Mazin** for her continuous support, kindness, patience and putting up with my shortcomings.*

*and my sincere appreciation for the **whole department of periodontics** for their countless selfless efforts for their students.*

List of contents

List of Abbreviations

Abbreviations	Complete word
PD	Periodontal disease
PTSD	post-traumatic stress disorder
%	Percentage
> <	More or less than
CNS	Central nervous system
SNS	Sympathetic Nervous System
ANS	autonomic nervous system
GAS	General adaptation syndrome
IL	interleukin
MMPs	Matrix metalloproteinases
β	beta
GCF	gingival crevicular fluid
ANUG	Acute necrotizing ulcerative gingivitis
HPA	hypothalamic–pituitary–adrenal axis
ACTH	Adrenocorticotropic Hormone
Ig	Immunoglobulin
PMNs	Polymorphonuclear neutrophils

Aim of The Research

The aim of this research project is to bring to light and evaluate the effect of stress on the periodontal health.

Introduction

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. **(Breivik et al 1996)**

(De Marco 1976) 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 term "**stress**" originated from a Latin word "*stringere*," which means "tight strained." Currently, stress is classified as a "risk factor" for periodontal disease. Stress is regarded as a cognitive perception of uncontrollability and/or unpredictability, i.e., expressed in a physiological and behavioral response. Thus, stress can be viewed as a process with both psychological and physiological components. (**Koolhaas 2011)**

In (**1976, Selye)** 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 1996)** 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.

Review of literature

1.1 Periodontal Disease

Periodontal diseases are disease processes involving the periodontium, a term used to describe the supportive apparatus surrounding the tooth, which includes the gingival tissue, alveolar bone, cementum, and periodontal ligament. **(Pihlstrom BL et al 2005)**

Periodontal diseases can be seen in up to 90% of the global population, making it the most common oral disease.

In the United States alone, cross-sectional studies show that approximately 50% of adults currently have some form of gingivitis, and up to 80% have experienced some form of periodontal disease in their life.

Certain groups have been shown to have an increased incidence of periodontal diseases. These groups include older individuals, males, and African-Americans. Lower-income and education levels were also associated with severe periodontitis. **(Nazir MA 2017** ; **Ridgeway EE 2000)**

A more rapidly progressing form of periodontitis (formerly termed "juvenile periodontitis" and "aggressive periodontitis") presents with similar symptoms, but this form of condition has a predilection to younger patients. This form of the condition tends to target specific teeth (most commonly first molars and incisors) and often occurs in the absence of the significant plaque accumulation noted in chronic periodontitis. (**Wiebe CB 2000)**

1.1.1 Classification of Periodontal Disease

2017 world workshop on the classification of periodontal and peri-implant diseases and conditions: **(caton et al.)**

1. Gingivitis – Dental Biofilm-induced:

    - Associated with dental biofilm alone
    - Mediated by systemic or local risk factor

2. Gingival Diseases – Non-dental biofilm-induced:

    - Genetic/developmental disorders
    - Specific infections
    - Inflammatory and immune conditions
    - Reactive processes
    - Neoplasms
    - Endocrine, nutritional & metabolic diseases
    - Traumatic lesions

3. Periodontitis:

    - Stages: Based on severity and complexity of management:
      Stage I: Initial periodontitis
      Stage II: Moderate periodontitis
      Stage III: Severe periodontitis with potential for additional tooth loss
      Stage IV: Severe periodontitis with potential for loss of the dentition
    - Extent and distribution: localized; generalized; molar-incisor distribution
    - Grades: Evidence or risk of rapid progression, anticipated treatment response
      Grade A: Slow rate
      Grade B: Moderate rate of progression
      Grade C: Rapid rate of progression

4. Necrotizing Periodontal Diseases:

    - Necrotizing gingivitis
    - Necrotizing periodontitis
    - Necrotizing stomatitis

5. Periodontitis as Manifestations of Systemic Diseases.
6. Periodontal Abscesses and Endodontic-Periodontal Lesions.

7. Mucogingival Deformities and Conditions:

    - Gingival/soft tissue recession
    - Lack of gingiva
    - Decreased vestibular depth
    - Aberrant frenum/muscle position
    - Gingival excess
    - Abnormal color
    - Condition of the exposed root surface

8. Traumatic Occlusal Forces:

    - Primary occlusal trauma
    - Secondary occlusal trauma
    - Orthodontic forces

9. Tooth and Prosthesis-related Factors:
10. Peri-implant Soft and Hard Tissue Deficiencies

1.1.2 Causes and Risk Factors of Periodontal Disease

Periodontal diseases arise as a result of several factors, including both patient-specific risk factors and inadequate oral hygiene.

The risk factors can be subdivided into:

modifiable risk factors: including smoking tobacco, poor oral hygiene, medications and stress.

non-modifiable risk factors: like age and heredity, including genetic diseases.

Inadequate oral hygiene practices play a significant role in the initiation and development of periodontal diseases.

Improper oral hygiene techniques can lead to the build-up of bacteria and plaque on the teeth, initiating gingivitis and potentially progressing to periodontitis.

With inadequate oral hygiene, anaerobic organisms responsible for the progression of periodontal diseases can colonize in deeper areas of the periodontium where they can then execute their destructive actions.

The main bacteria found in periodontitis include Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola, and Tannerella forsythia.

When allowed to penetrate deep into the periodontium, these organisms produce inflammation by triggering the release of inflammatory mediators, and other defensive products from the host. **(Nazir MA 2017)**

1.1.3 Diagnosis of Periodontal Disease

Chronic periodontitis can present in patients of any age, but most often affects middle-aged to older adults.

The severity of the disease is based on the amount of clinical attachment loss (CAL).

It is described as mild when the CAL is 1 to 2 mm, moderate the CAL is 3 to 4 mm, or severe when the CAL is more than 5 mm. **(Wiebe CB 2000)**

Routine dental screenings are invaluable in recognizing early disease states and directing early intervention.

A review of the patient medical history and comorbidities raises the awareness of dental care providers.

Inspection of the tooth and gum can reveal inflammation of the gum line, receding gums, and sometimes even pus-filled pockets.

Periodontal probes are used to measure dental pockets adjacent to several teeth. A probing depth of greater than 3 mm may be indicative of periodontal disease.

Depths greater than 6 mm often require more aggressive therapy as these spaces are more difficult to treat mechanically.

Dental X-rays also can check for bone loss in accordance with the depth of pocket and disease severity. (**Pihlstrom BL 2005 ; Mehrotra N 2022)**

The diagnosis of periodontal diseases requires the comparison of findings in relation to the normal periodontium.

This comparison uses visual inspection, periodontal probing, and evaluation of bone levels seen radiographically.

he normal periodontium consists of stippled, pale pink gingiva that is well adapted to the underlying bone.

Between the gingiva and the tooth, there is a 1 to 3 mm physiological sulcus that normally displays no signs of bleeding.

Comparatively, signs of periodontal disease include active bleeding in response to mild or no tissue manipulation, pain, bad taste/odor, periodontal pocketing, radiographic bone loss, clinical attachment loss,

If left untreated, the bone loss will progress until there is inadequate tooth support, and the associated tooth will become mobile and eventually be lost. **(Highfield J 2009) / (Kinane DF 2017).**

The final diagnosis consists by sequence of :

- **Extent:**
  - Molar incisor pattern
  - Localized (less than 30 % bone loss)
  - Generalized (more than 30% bone loss)

- **Staging (severity of the disease)**

  By calculating the clinical attachment loss (CAL) using periodontal probe.
  - I cal 1-2 mm
  - II cal coronal third
  - III cal middle thrid
  - IV cal apical third
- **Grading (rate of progression of the disease)**

% bone loss worst site / patients age

  - A slow rate of progression (less than 0.5)
  - B moderate rate of progression (0.5-1)
  - C rapid rate of progression (more than 1)
- **Status**
  - Stable (BOP <10% , PPD < or equal to 4mm , no PPD on 4mm sites)
  - In remission (BOP >10% , PPD < or equal to 4mm , no PPD on 4mm sites)

- Unstable (PPD> or equal to 5mm , PPD > or equal to 4mm with BOP)

- **Risk factors**
  - **Smoking**
  - **Uncontrolled type 2 diabetes**
  - **Stress**

(Sanz M 2018)

1.1.4 Treatment and Management of Periodontal Disease

The treatment of periodontal disease involves a step-wise approach beginning with more conservative options.

The initial phase of treatment for all forms of periodontitis is a professional dental cleaning, which includes scaling of the teeth, and root planning to remove dental plaque and calculus found both above and below the gum line.

A major part of this dental cleaning is the oral hygiene instruction given by the dental professional to the patient to improve their at-home oral hygiene routine.

Following the completion of the cleaning appointment, the patient should return to the dentist for a reevaluation of the periodontal condition, which involves an examination that observes the state of the periodontium, and measures probing depths to see if the disease process was arrested. If the resolution of the condition can be confirmed, the patient should return to the dentist for regularly scheduled cleanings as periodontitis is a chronic disease that can reactivate if given the proper environment. **(Kinane DF 2017)**

The most important management of the periodontal disease is the treatment of risk factors.

Inadequate oral hygiene is one of the key initiators of periodontal disease.

Prevention of poor oral hygiene practices involves the promotion of proper self-performed oral hygiene as well as professional maintenance at regular intervals depending on the individual patient's risk.

The self-care recommended uses a three-step daily regimen that includes brushing, flossing, and rinsing. **(Kinane DF 2017)**

Diabetes mellitus has well-documented links to periodontal disease; it may enhance the destruction seen in periodontitis. Additionally, poor glycemic control is linked to increased disease progression.

Uncontrolled glucose levels are associated with higher mortality if the patient has severe periodontal disease.

Therefore, the management of diabetes mellitus and prediabetes mellitus may be required to improve the outcomes of periodontal therapy. **(Nazir Ma 2017)**

Another major modifiable risk factor that must be addressed is tobacco smoking.

Tobacco smoking has not only been shown to significantly increase the risk of developing periodontal disease but is also associated with a more severe disease course, and a significantly lower response to periodontal therapies.

The link between smoking and periodontal disease decreases with smoking cessation. **(Hilgers KK 2004)**

In cases of persistent periodontal disease that is refractory to non-pharmacologic therapies, antibiotics can be administered both locally and systemically, depending on the severity of the disease.

Chlorhexidine gluconate is a common antimicrobial compound used in adjunction to mechanical periodontal therapy.

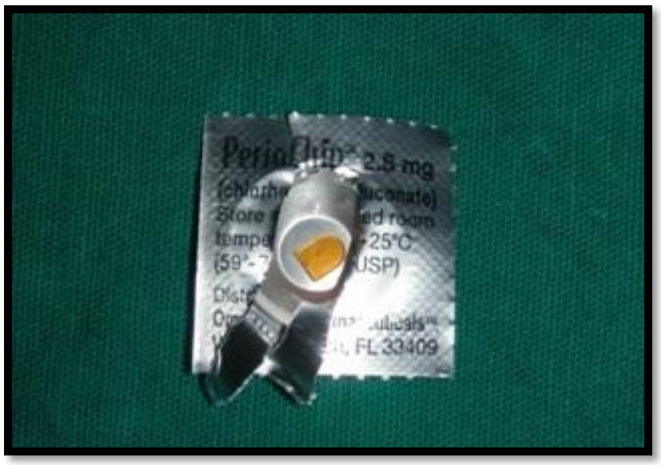
It is generally administered as a mouth rinse, but can also be used as a gel, varnish, and subgingival chip.

Using chlorhexidine, in addition to regular toothbrushing, can lead to a reduction of dental plaque build-up and thus can be very beneficial in the treatment of chronic periodontitis.

There is a relatively new advancement in pharmacotherapy for periodontal disease. It is a **chlorhexidine gluconate chip** that is inserted into the periodontal pocket following completion of cleaning and provides long-term, sustained-release of chlorhexidine gluconate into the affected area. **(Pietruska M 2006)**

Fig 1 : Chlorhexidine chip (Periochip containing 2.5mg of Chlorhexidine gluconate).

Fig 2 : Placement of Periochip into The Periodontal pocket.



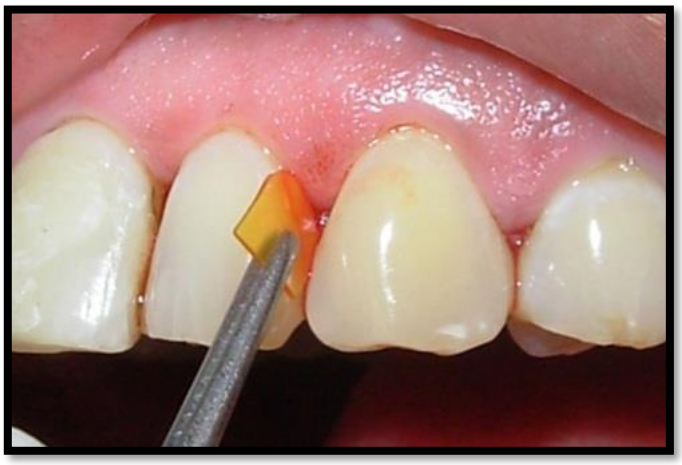


Fig 1 **Fig 2**

(Medaiah S 2014)

1.2 Stress

(Claude Bernard 1865/1961) noted that the maintenance of life is critically dependent on keeping our internal milieu constant in the face of a changing environment.

(Cannon 1929) called this "homeostasis."

(Selye 1956) used the term "stress" to represent the effects of anything that seriously threatens homeostasis.

The actual or perceived threat to an organism is referred to as the "stressor" and the response to the stressor is called the "stress response." Although stress responses evolved as adaptive processes, Selye observed that severe, prolonged stress responses might lead to tissue damage and disease. **(Selye 1956)**

Although various situations tend to elicit different patterns of stress responses, there are also individual differences in stress responses to the same situation. This tendency to exhibit a particular pattern of stress responses across a variety of stressors is referred to as "response stereotypy" **(Lacey & Lacey 1958)**

Across a variety of situations, some individuals tend to show stress responses associated with active coping, whereas others tend to show stress responses more associated with aversive vigilance. (**Kasprowicz et al. 1990, Llabre et al. 1998)**

1.2.1 Types of Stress Responses

1.2.1.1 Acute Stress Responses:

Following the perception of an acute stressful event, there is a cascade of changes in the nervous, cardiovascular, endocrine, and immune systems. These changes constitute the stress response and are generally adaptive, at least in the short term. **(Selye 1956)**

Two features in particular make the stress response adaptive. First, stress hormones are released to make energy stores available for the body's

immediate use. Second, a new pattern of energy distribution emerges. Energy is diverted to the tissues that become more active during stress, primarily the skeletal muscles and the brain. Cells of the immune system are also activated and migrate to "battle stations" (**Dhabar & McEwen 1997)**. Less critical activities are suspended, such as digestion and the production of growth and gonadal hormones. Simply put, during times of acute crisis, eating, growth, and sexual activity may be a detriment to physical integrity and even survival.

Stress hormones are produced by the SNS and hypothalamic-pituitary adrenocortical axis. The SNS stimulates the adrenal medulla to produce catecholamines (e.g., epinephrine). **(Brindley & Rollan 1989)**

Energy is then distributed to the organs that need it most by increasing blood pressure levels and contracting certain blood vessels while dilating others. **(Llabre et al.1998, Schneiderman & McCabe 1989)**

1.2.1.2 Chronic Stress Responses:

The acute stress response can become maladaptive if it is repeatedly or continuously activated. **(Selye 1956)**

For example, chronic SNS stimulation of the cardiovascular system due to stress leads to sustained increases in blood pressure and vascular hypertrophy. **(Henry et al. 1975)**

That is, the muscles that constrict the vasculature thicken, producing elevated resting blood pressure and response stereotypy, or a tendency to respond to all types of stressors with a vascular response. Chronically elevated blood pressure forces the heart to work harder, which leads to hypertrophy of the left ventricle. **(Brownley et al. 2000)**

1.2.2 Psychological Aspects of Stress

1.2.2.1 Stressors During Childhood and Adolescence and Their Psychological Sequelae:

The most widely studied stressors in children and adolescents are exposure to violence, abuse (sexual, physical, emotional, or neglect), and divorce/marital conflict. (**Cicchetti 2005)**

Psychological effects of maltreatment/abuse include the dysregulation of affect, provocative behaviors, the avoidance of intimacy, and disturbances in attachment. **(Haviland et al. 1995, Lowenthal 1998)**

Survivors of childhood sexual abuse have higher levels of both general distress and major psychological disturbances including personality disorders. **(Polusny & Follett 1995)**

Childhood abuse is also associated with negative views toward learning and poor school performance. **(Lowenthal 1998)**

Adult offspring of divorced parents report more current life stress, family conflict, and lack of friend support compared with those whose parents did not divorce. **(Short 2002)**

Exposure to nonresponsive environments has also been described as a stressor leading to learned helplessness. **(Peterson & Seligman 1984)**

Studies have also addressed the psychological consequences of exposure to war and terrorism during childhood. **(Shaw 2003)**

majority of children exposed to war experience significant psychological morbidity, including both post-traumatic stress disorder (PTSD) and depressive symptoms. For example, **(Nader et al. 1993)** found that 70% of Kuwaiti children reported mild to severe PTSD symptoms after the Gulf War. Some effects are long lasting.

Exposure to intense and chronic stressors during the developmental years has long-lasting neurobiological effects and puts one at increased risk for anxiety and mood disorders, aggressive dyscontrol problems, hypo-

immune dysfunction, medical morbidity, structural changes in the CNS, and early death. **(Shaw 2003)**

1.2.2.2 Stressors During Adulthood and Their Psychological Sequelae:

Life stress, Anxiety And depression is well known first depressive episodes that often develop following the occurrence of a major negative life event. **(Paykel 2001)**

Furthermore, there is evidence that stressful life events are causal for the onset of depression. **(Hammen 2005, Kendler et al. 1999)**

The diagnosis of a major medical illness often has been considered a severe life stressor and often is accompanied by high rates of depression . **(Cassem 1995)**

Other consequences of stress that could provide linkages to health have been identified, such as increases in smoking, substance use, accidents, sleep problems, and eating disorders. Populations that live in more stressful environments (communities with higher divorce rates, business failures, natural disasters, etc.) smoke more heavily and experience higher mortality from lung cancer and chronic obstructive pulmonary disorder. **(Colby et al. 1994)**

A longitudinal study following seamen in a naval training center found that more cigarette smoking occurred on high-stress days. **(Conway et al. 1981)**

Life events stress and chronically stressful conditions have also been linked to higher consumption of alcohol. **(Linsky et al. 1985)**

In addition, the possibility that alcohol may be used as self-medication for stress-related disorders such as anxiety has been proposed. For example, a prospective community study of 3021 adolescents and young adults **(Zimmerman et al. 2003)** found that those with certain anxiety disorders (social phobia and panic attacks) were more likely to develop substance abuse or dependence prospectively over four years of follow-up.

1.2.3 Psychological Stressors

Stressor is any stimuli, situation, or circumstance with the potential to induce stress reaction. The effect of the stress response includes anxiety, depression, impaired cognition, and altered self-esteem. **(Formicola 1970)**

(LeResche 2002) generally classified Psychosocial stressors as

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

Holmes and Rahe (1967) 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)**

Table 1: Life change scale

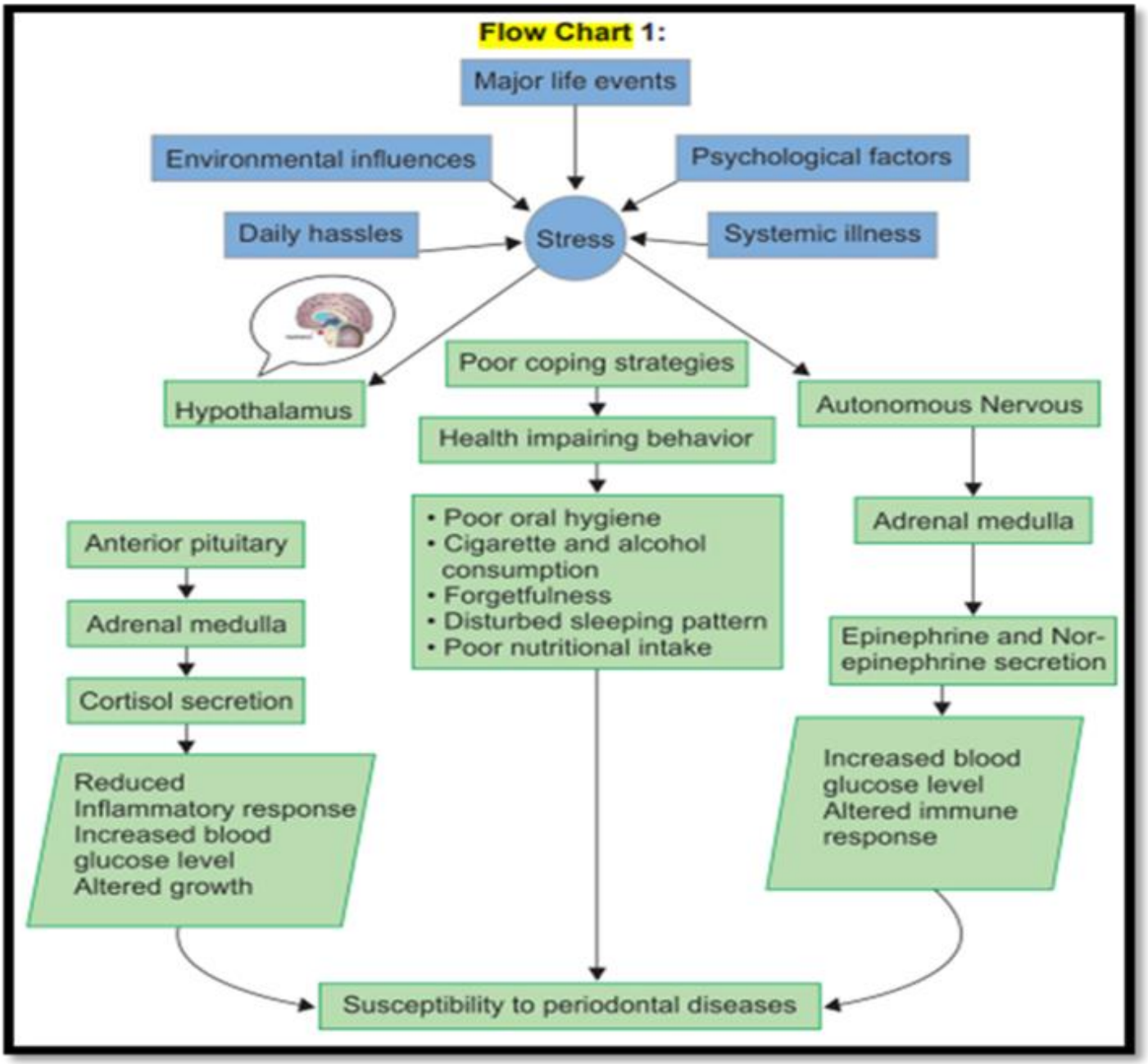
Events	*Score*
Death of spouse	100
Divorce	73
Marital separation	65
Death of close family member	60
Personal injury or illness	53
Marriage	50
Frustrated from job/loss of work	47
Conjugal reconciliation	45
Retirement	43
Life change scale	

Holmes and Rahe (1967)

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.

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. **(Breivik et al 1996)**



Flowchart 1 by (Wasu et al. 2017) "The Effects of psychological stressors on periodontal health"

1.3 Effects of Stress on Periodontal Disease

1.3.1 Biological Changes

1.3.1.1 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. **(Davis CH 1962)**

General adaptation syndrome (GAS) describes the process your body goes through when you are exposed to any kind of stress, positive or negative. It has three stages: alarm, resistance, and exhaustion. If you do not resolve the stress that has triggered GAS, it can lead to physical and mental health problems. **(Davis CH 1962)**

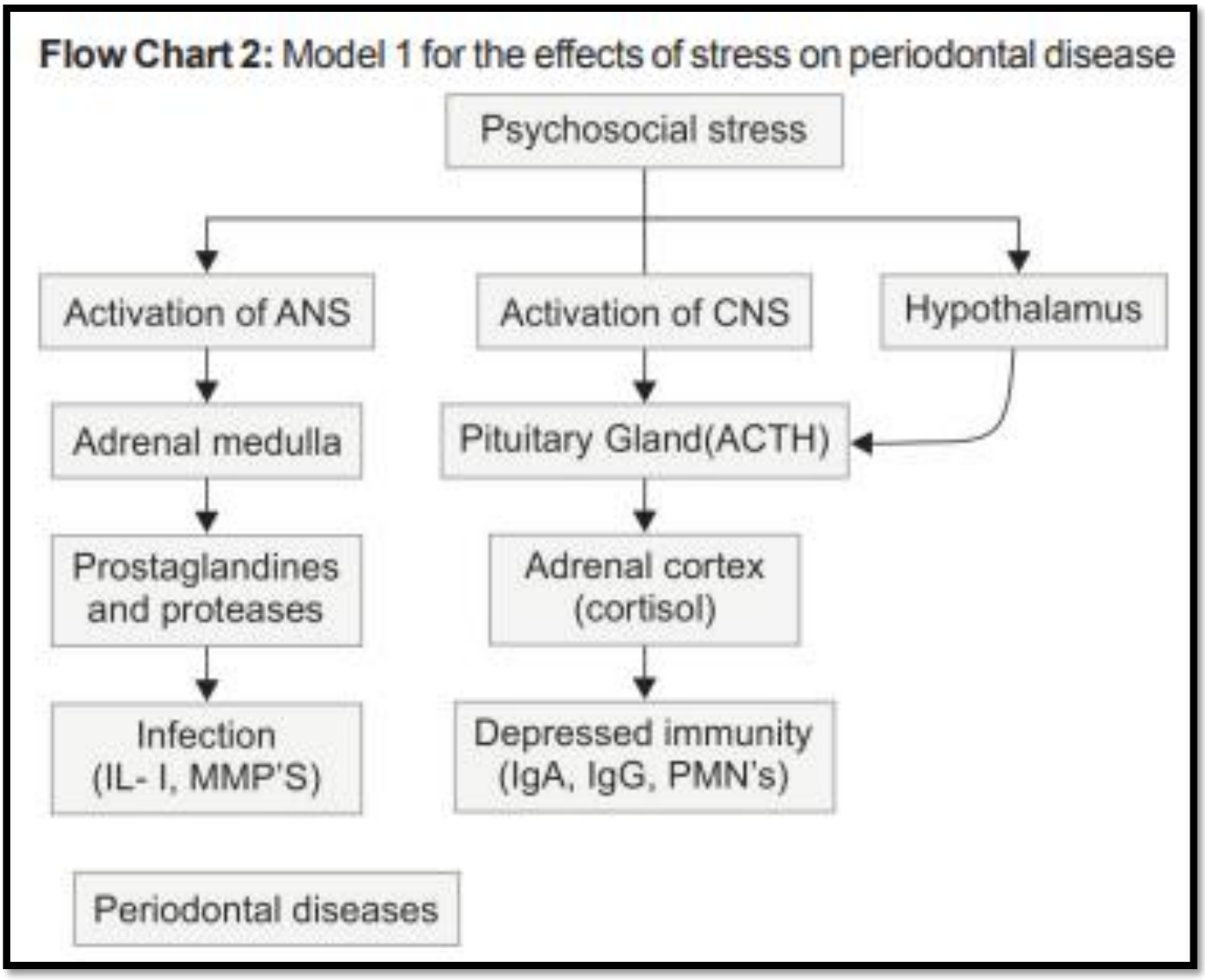
Model 1 offered a schematic model, which demonstrates the potential role that psychosocial stressors may play in initiating a cascade of events in the corticotropin-releasing hormone/hypothalamic–pituitary–adrenal (HPA) axis, the autonomic nervous system, and the central nervous system, the physiological consequences of which are to depress immunity, enhancing the likelihood of infection and, specifically, periodontal disease. **(Genco RJ 1998)**

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 stress and depression signs.

High cortisol levels may be especially negative on periodontal tissue because of the extremely fast turnover of some periodontal components.

(Axtelius B 1998) / (Deinzer R 2000)

Turnover rate defines the rate at which an enzyme converts its substrate, usually in terms of the number of substrate molecules that can be converted by a single enzyme molecule and can range from a few molecules to several million molecules per second. **(Bhagi-Damodaran A 2016)**



(Genco RJ 1998)

1.3.1.2 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. **(Manhold JH 1971)**

Muscle tone is traditionally defined as 'the tension in the relaxed muscle' or 'the resistance, felt by the examiner during passive stretching of a joint when the muscles are at rest. **(Campbell W 2019)**

1.3.1.3 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. **(Gupta OP 1966)**

1.3.1.4 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.

(Soell M 2002 ; Buduneli N 2008)

* Cytokines are small secreted proteins released by cells have a specific

Cytokine is a general name; other names include lymphokine (cytokines made by lymphocytes), monokine (cytokines made by monocytes), chemokine (cytokines with chemotactic activities), and interleukin (cytokines made by one leukocyte and acting on other leukocytes). fect on the interactions and communications between cells.

Cytokines may act on the cells that secrete them (autocrine action), on nearby cells (paracrine action like responses to allergens, tissue repair, the formation of scar tissue, and blood clotting), or in some instances on distant cells (endocrine action like thyroid secretions).

There are both pro-inflammatory cytokines and anti-inflammatory cytokines. **(Zhang JM 2007)**

Stress impairs the balance between proinflammatory and anti-inflammatory responses.

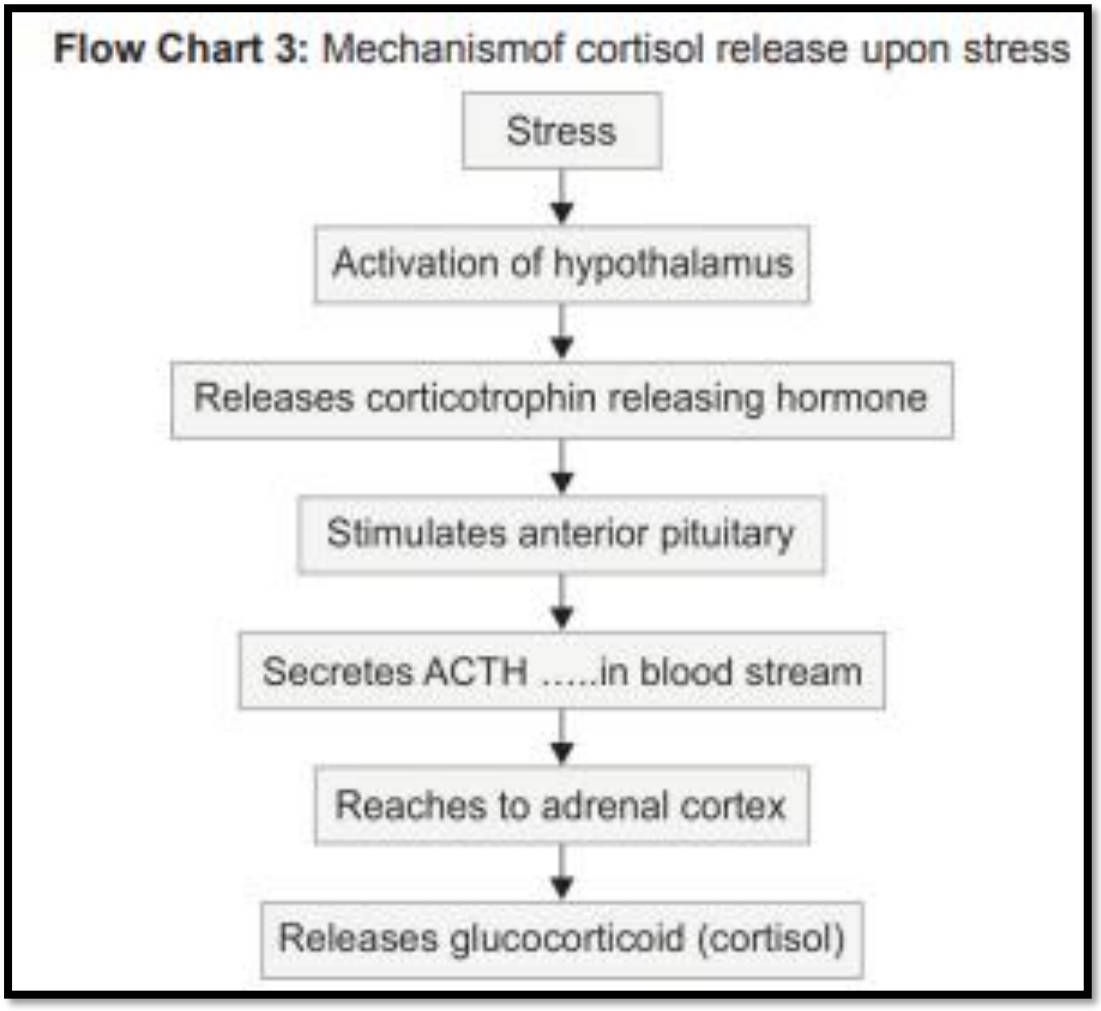
The relationship between stress and periodontal diseases might be mediated by alterations in GCF IL-1, IL-6 levels. **(Sheiham A 2005)**

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 (The Hypothalamus-Pituitary-Adrenal Axis) 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.

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)**. **(Ishisaka A 2007)**



1.3.2 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 "vicious cycle" of increasingly severe forms of advanced periodontal inflammation and disease. **(Ringsdorf WM Jr et al 1969 ; Hildebrand HC et al 2000)**

1.3.2.1 Negligence 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. **(Ringsdorf WM Jr et al 1969 ; Hildebrand HC et al 2000)**

1.3.2.2 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.

Stress leads to other behavioral changes, such as overeating, especially a high-fat diet, which then can lead to immunosuppression through increased cortisol production. **(Suchday S et al 2006)**

1.3.2.3.1 smoking

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. **(HaberJ 1994)**

One-third of the world's adult population are smokers (57% of these are men, 43% are women) . It is predicted that in 20 years this yearly death rate from tobacco use will be more than 10 million people. Smoking in developing countries is rising by more than 3% a year . We can assume periodontal diseasees will also rise. **(Hayman L et al 2011)**

Smoking can increase the risk for periodontal diseases 5-20 fold

Additionally, tobacco smoking is associated with greater levels of bone loss, attachment loss, deep periodontal pockets associated with the disease, and tooth loss, as compared to non-smokers.

In addition to the increased severity of periodontal diseases, tobacco smoking is also associated with a significant decrease in the effectiveness of treatments. **(Brothwell DJ et al 2001)**

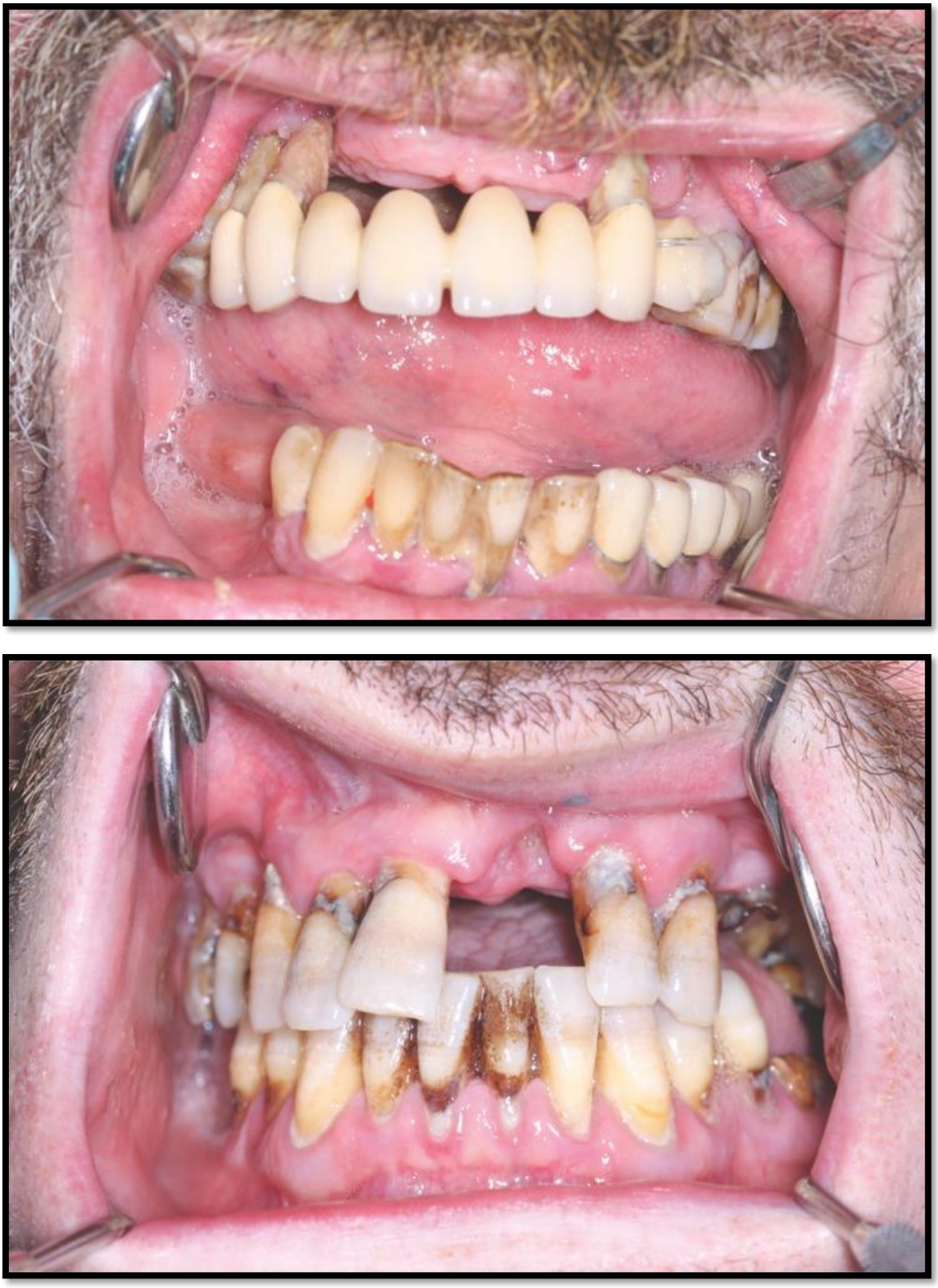
(HaberJ 1994) stated that Circulating nicotine results in :

- vasoconstriction, produced by the release of adrenaline and noradrenaline, which is supposed to result in a lack of nutrients for the periodontal tissue
- inhibition of oral neutrophil function

Smoking is well-established risk factor for periodontal disease. It changes the human microflora , human immune response that leads to destruction of the supporting tissues of the tooth.

It can take years before the patient seeks help, then it is often too late.

(Borojevic T 2012)



Generalized advanced chronic periodontitis in smoker.

(Borojevic T 2012)

1.3.2.3.2 Acute Necrotizing Ulcerating Gingivitis (ANUG) and it's Relation to Smoking

It is thought that both smoking and ANUG may be the result of underlying anxiety and stress.

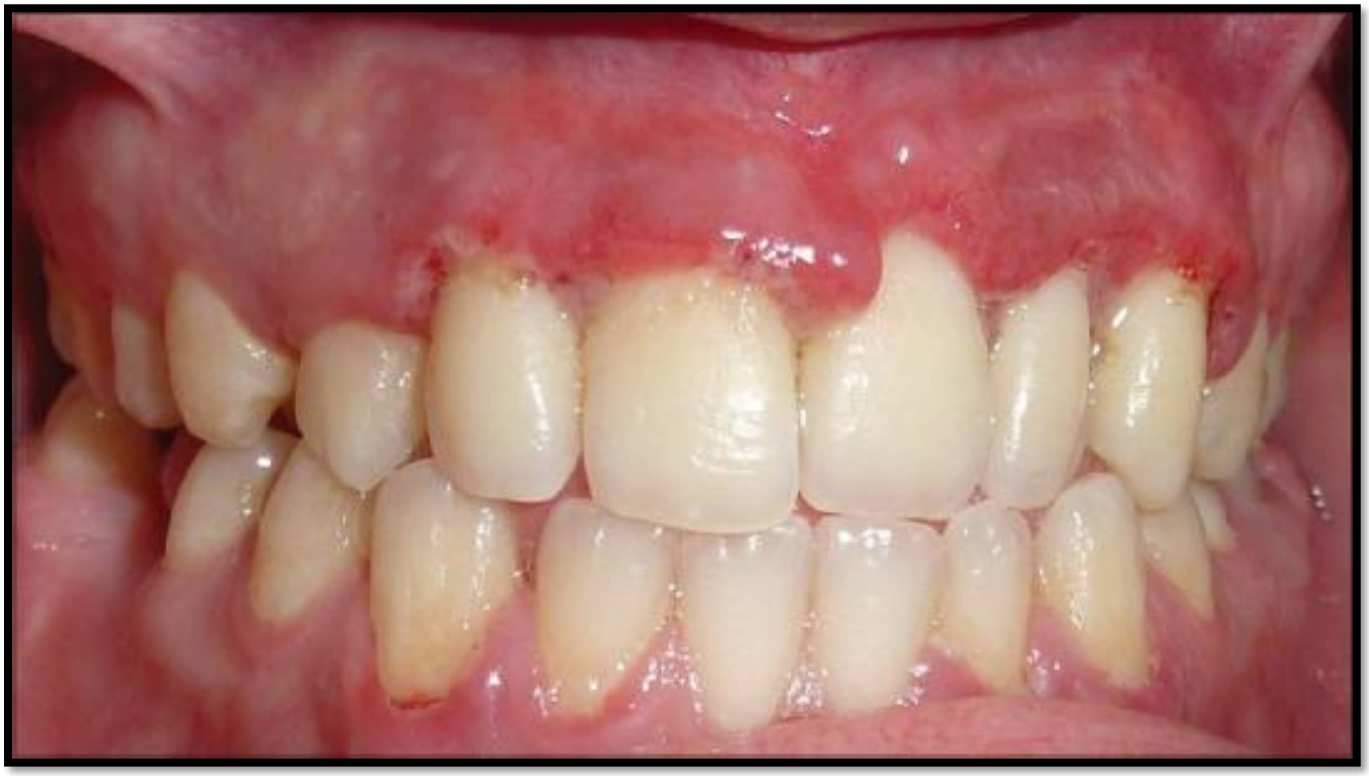
The condition involves primarily the free gingival margin, the crest of the gingiva, and the interdental papillae.

ANUG is characterized by punched-out papilla, pronounced gingival erythema, and spontaneous hemorrhage.

Local lymphadenopathy and slight elevation of temperature may also be present.

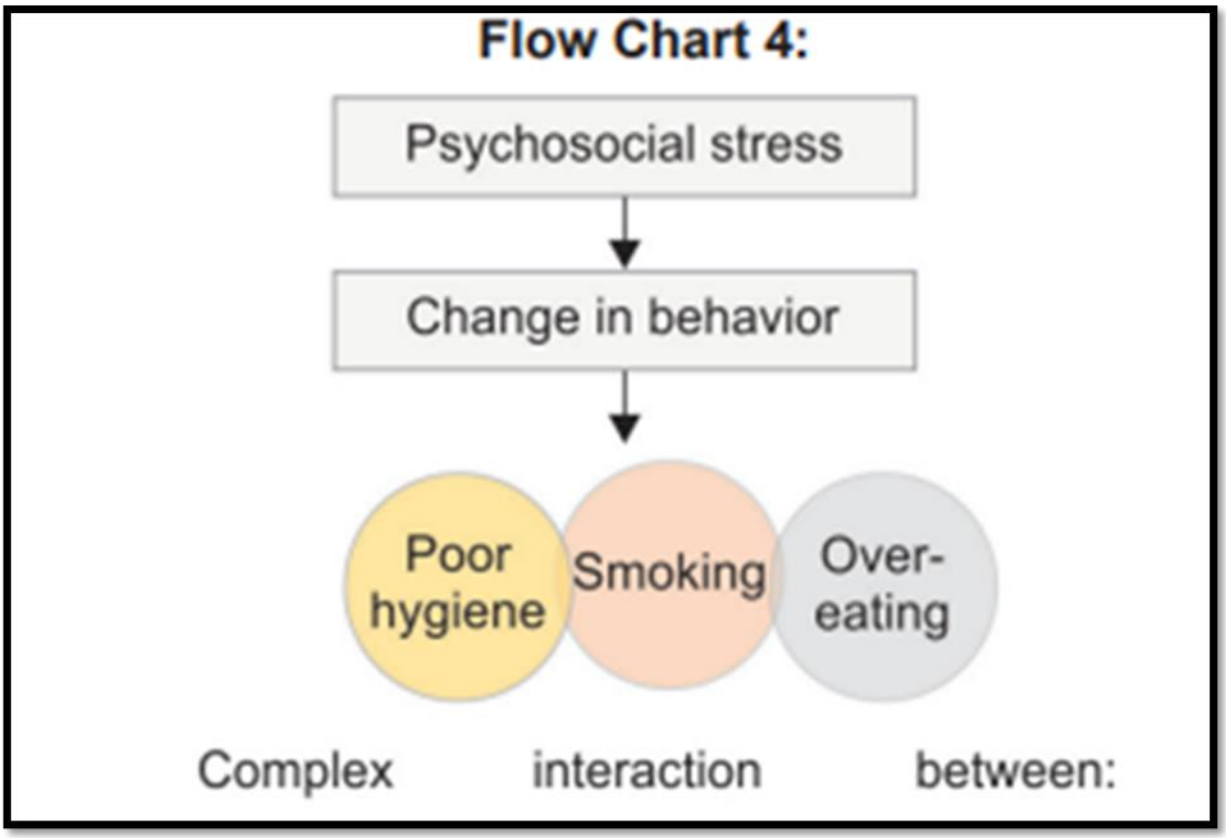
The disease may resolve spontaneously, but treatment usually consists of mechanical debridement and systemic antibiotic treatment.

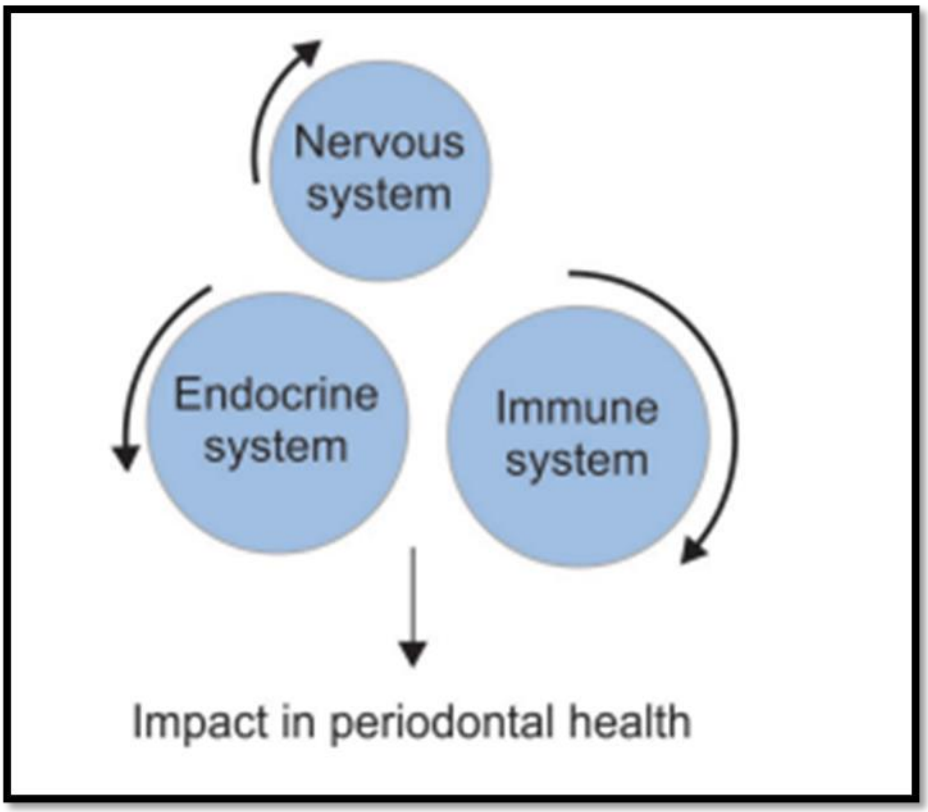
(Carranza FA 1996)



ANUG

(Malek R et al 2017)





Effefcs of stress on the individual's behavior

(wasu et al 2017)

1.3.2.4 Alcohol consumption

Alcohol may affect periodontal tissues through different mechanisms.

- There is evidence that alcohol has an adverse effect on host defense. (**Christen AG 1983 ; Drake CW. 1995)**
- Alcohol has a toxic effect on the liver, Prothrombin production and clotting mechanism may be disrupted and hemorrhage may take place.
  Exaggerated gingival inflammation, bluish-red discoloration, and bleeding with slightest provocation are commonly seen in alcoholics.
- Alcohol may interfere with protein metabolism and tissue healing,
  A history of combined vitamin B-complex and protein deficiency is often obtained from alcoholics.
- In vitro studies suggest that ethanol stimulates bone resorption and blocks the stimulation of bone formation. **(Farley JR et al 1985)**

1.3.2.5 Bruxism

Bruxism is a repetitive muscular activity of the jaw characterized by grinding or clenching the teeth and bracing or thrusting of the mandible, is mainly regulated centrally, and may involve more than dental contact.

Currently, bruxism has a distinction between sleep bruxism and awake bruxism. Sleep bruxism is a sleep-related movement disorder characterized as rhythmic or non-rhythmic of masticatory muscle activity.

In contrast, awake bruxism is a non-functional behavior during wakefulness characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible. **(Lobbezoo et al. F 2018 / Reissmann DR et al. 2017)**

The factors associated with the development of bruxism are:

bad habits: such as smoking, high alcohol, and coffee consumption.

Other causes like: sleep apnea syndrome, respiratory diseases, anxiety disorder, stress, and depression Recent studies show that emotional

changes may be associated with bruxism. **(Kuhn M et al. 2018 / Cruz Fierro N et al. 2018 / Soares-Silva L et al. 2019 / Yap AU et al. 2016)**

1.3.3 Periodontal Immune–Microbiome Balance in the Link between Chronic Stress, Depression, Periodontitis

It has been proposed that chronic stress may alter the composition of the commensal microbiota in the human microbiome , resulting in the so-called stress-related dysbiosis. **(Gur, T.L et al. 2015)**

It is well known that a dysbiotic microbiome may lead to a series of different diseases. **(Duran-Pinedo, A.E et al. 2018 / Barone, A et al . 2020 / Di Spirito et al. 2019)**

Periodontitis and peri-implantitis, which are initiated by bacterial aggregation, leading to periodontal and peri-implant tissues inflammation, subsequently progressing in an apical direction and invading bone compartment **(Tonetti M.S et al. 2018 / Berglundh, T et al. 2017)**,are also characterized by a dysbiotic microbiota, with decreased coccoid and straight rod microbial populations and increased motile organisms compared to periodontally healthy sites. **(Lindhe, J et al. 1980)**

2.1 Conclusion

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 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. **(wasu et al 2017)**

References

A

1. Albandar JM. Global risk factors and risk indicators for periodontal diseases. Periodontol 2000. 2002;29:177-206. [PubMed]
2. Axtelius B, Söderfeldt B, Nilsson A, Edwardsson S, Attström R. Therapy-resistant periodontitis. Psychosocial characteristics. J Clin Periodontol 1998 Jun;25(6):482-491

B

3. Barone, A.; Chatelain, S.; Derchi, G.; Di Spirito, F.; Martuscelli, R.; Porzio, M.; Sbordone, L. Effectiveness of antibiotics in preventing alveolitis after erupted tooth extraction: A retrospective study. Oral Dis. 2020, 26, 967–973. [Google Scholar] [CrossRef] [PubMed]
4. Berglundh, T.; Armitage, G.; Araujo, M.G.; Avila-Ortiz, G.; Blanco, J.; Camargo, P.M.; Chen, S.; Cochran, D.; Derks, J.; Figuero, E.; et al. Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J. Clin. Periodontol. 2018, 45, 286–291. [Google Scholar] [CrossRef] [PubMed][Green Version]
5. Bergström J. Tobacco smoking and chronic destructive periodontal disease. Odontology. 2004 Sep;92(1):1-8. [PubMed]
6. Bernard C. An Introduction to the Study of Experimental Medicine. Transl. HC Greene. New York: Collier; 18651961. [Google Scholar] [Ref list]
7. Bhagi-Damodaran A, Hosseinzadeh P, Mirts E, et al. Design of heteronuclear metalloenzymes. Methods Enzymol. 2016;580:501–537. [Crossref], [PubMed], [Web of Science ®], [Google Scholar]

8. Borojevic T. Smoking and periodontal disease. Mater Sociomed. 2012;24(4):274-6. doi: 10.5455/msm.2012.24.274-276. PMID: 23678331; PMCID: PMC3633395.
9. Borrell LN, Beck JD, Heiss G. Socioeconomic disadvantage and periodontal disease: the Dental Atherosclerosis Risk in Communities study. Am J Public Health. 2006 Feb;96(2):332-9. [PMC free article] [PubMed]
10. Breivik T, Thrane PS, Murison R, Gjermo P. Emotional stress effects on immunity, gingivitis and periodontitis. Eur J OralSci 1996 Aug;104(4 (Pt 1)):327-334.
11. Brindley D, Rollan Y. Possible connections between stress, diabetes, obesity, hypertension, and altered lipoprotein metabolism that may result in atherosclerosis. Clin. Sci. 1989;77:453–461. [PubMed] [Google Scholar]
12. Brothwell DJ. Should the use of smoking cessation products be promoted by dental offices? An evidence-based report. J Can Dent Assoc. 2001 Mar;67(3):149-55. [PubMed]
13. Brownley KA, Hurwitz BE, Schneiderman N. Cardiovascular psychophysiology. In: Cacioppo JT, Tassinary LG, Berntson GG, editors. Handbook of Psychophysiology. 2nd ed. New York: Cambridge Univ.; 2000. pp. 224–264. [Google Scholar]
14. Buduneli N, Biyikoğlu 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.

C

15. Campbell W.W., Barohn R.J. DeJong's The Neurologic Examination. 7th ed. Lippincott Williams & Wilkins; Philadelphia, PA, USA: 2019. Muscle Tone; pp. 467–474. [Google Scholar] [Ref list]
16. Cannon WB. Bodily Changes in Pain, Hunger, Fear and Rage. 2nd ed. New York: Appleton; 1929. [Google Scholar]

17. Carranza FA. Glickman's Clinical Periodontology. 8th ed. 1996. WB Saunders Co.
18. Cassem EH. Depressive disorders in the medically ill: an overview. Psychosomatics. 1995;36:S2–S10. [PubMed] [Google Scholar]
19. Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions– Introduction and key changes from the 1999 classification. J Periodontol 2018;89(Suppl 1): S1-S8
20. chneiderman N, Ironson G, Siegel SD. Stress and health: psychological, behavioral, and biological determinants. Annu Rev Clin Psychol. 2005;1:607-28. doi: 10.1146/annurev.clinpsy.1.102803.144141. PMID: 17716101; PMCID: PMC2568977
21. Christen AG. Dentistry and the alcoholic patient. Dent Clin North Am 1983;27:341.
22. Cicchetti D. Child maltreatment. Annu. Rev. Clin. Psychol. 2005;1:409–438. [PubMed] [Google Scholar]
23. Colby JP, Linsky AS, Straus MA. Social stress and state-to-state differences in smoking-related mortality in the United States. Soc. Sci. Med. 1994;38:373–381. [PubMed] [Google Scholar]
24. Conway TL, Vickers RR, Ward HW, Rahe RH. Occupational stress and variation in cigarette, coffee and alcohol consumption. J. Health Soc. Behav. 1981;22:156–165. [PubMed] [Google Scholar]
25. Cruz Fierro N, Martínez Fierro M, Cerda Flores RM, Gómez-Govea MA, Delgado-Enciso I, Martínez-De-Villarreal LE, et al.. The phenotype, psychotype and genotype of bruxism. Biomed. Rep. (2018) 8:264–8. 10.3892/br.2018.1041 [PMC free article] [PubMed] [CrossRef] [Google Scholar]

D

26. Davis CH, Jenkins CD. Mental stress and oral disease. J Dent Res 1962 Sep-Oct;41:1045-1049
27. De Marco T. Periodontal emotional stress syndrome. J Periodontol 1976 Feb;47(2):67-68

28. 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.
29. Dhabar FS, McEwen BS. Acute stress enhances while chronic stress suppresses cell-mediated immunity in vivo: a potential role for leukocyte trafficking. Brain Behav. Immun. 1997;11:286–306. [PubMed] [Google Scholar]
30. Di Spirito, F.; Argentino, S.; Martuscelli, R.; Sbordone, L. MRONJ incidence after multiple teeth extractions in patients taking oral bisphosphonates without “drug holiday”: A retrospective chart review. Oral Implantol. 2019, 12, 105–110. [Google Scholar]
31. Drake CW. Three-year tooth loss among black and white older adults in North Carolina. J Dent Res 1995;74:675- 680.
32. Duran-Pinedo, A.E.; Solbiati, J.; Frias-Lopez, J. The effect of the stress hormone cortisol on the metatranscriptome of the oral microbiome. NPJ Biofilms Microbiomes 2018, 4, 25. [Google Scholar] [CrossRef][Green Version]

F

33. Farley JR, Fitzsimmons R, Taylor AK, Jorch UM, Lau KH. Direct effects of ethanol on bone resorption and formation in vitro. Arch Biochem Biophys 1985;238:305-314.
34. Feldman RS, Bravacos JS, Rose CL. Association between smoking different tobacco products and periodontal disease indexes. J Periodontol. 1983 Aug;54(8):481-7. [PubMed] Flügge G, Korte SM, Meerlo P, Murison R, Olivier B, Palanza P,
35. Formicola AJ, Witte ET, Curran PM. A study of personality traits and acute necrotizing ulcerative gingivitis. J Periodontol 1970 Jan;41(1):36-38.

G

36. 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

37. Grossi SG, Skrepcinski FB, DeCaro T, Zambon JJ, Cummins D, Genco RJ. Response to periodontal therapy in diabetics and smokers. J Periodontol. 1996 Oct;67(10 Suppl):1094-102. [PubMed]
38. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, Norderyd OM, Genco RJ. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. J Periodontol. 1994 Mar;65(3):260-
39. Gupta OP. Psychosomatic factors in periodontal disease. Dent Clin North Am 1966 Mar:11-19
40. Gur, T.L.; Worly, B.L.; Bailey, M.T. Stress and the commensal microbiota: Importance in parturition and infant neurodevelopment. Front. Psychiatry 2015, 6, 5. [Google Scholar] [CrossRef] [PubMed][Green Version]

H

41. Haber J. Cigarette smoking: a major risk factor for periodontitis. Compendium. 1994 Aug;15(8):1002, 1004-8 passim; quiz 1014. [PubMed]
42. HaberJ. Smoking is a majorrisk factorfor periodontitis. Curr Opin Periodontol 1994:12-18.
43. Hammen C. Stress and depression. Annu. Rev. Clin. Psychol. 2005;1:293–319. [PubMed] [Google Scholar]
44. Haviland MG, Sonne JL, Woods LR. Beyond posttraumatic stress disorder: object relations and reality testing disturbances in physically and sexually abused adolescents. J. Am. Acad. Child Adolesc. Psychiatry. 1995;34:1054–1059. [PubMed] [Google Scholar]

45. Hayman L, Steffen MJ, Stevens J. Smoking and periodontal disease: discrimination of antibody responses to pathogenic and commensal oral bacteria. Clin Exp Immunol. 2011;164(1):118–126. [PMC free article] [PubMed] [Google Scholar]
46. Henry JP, Stephens PM, Santisteban GA. A model of psychosocial hypertension showing reversibility and progression of cardiovascular complications. Circ. Res. 1975;36:156–164. [PubMed] [Google Scholar]
47. Highfield J. Diagnosis and classification of periodontal disease. Aust Dent J. 2009 Sep;54 Suppl 1:S11-26. [PubMed]
    - Highfield J. Diagnosis and classification of periodontal disease. Aust Dent J. 2009 Sep;54 Suppl 1:S11-26. [PubMed]
48. 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
49. Hilgers KK, Kinane DF. Smoking, periodontal disease and the role of the dental profession. Int J Dent Hyg. 2004 May;2(2):56-63. [PubMed]
50. Hilgers KK, Kinane DF. Smoking, periodontal disease and the role of the dental profession. Int J Dent Hyg. 2004 May;2(2):56-63. [PubMed]
51. Holmes TH, Rahe RH. The social readjustment rating scale. J Psychosom Res 1967 Aug;11(2):213-218.

I

52. 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.

K

53. Kasprowicz AL, Manuck SB, Malkoff SB, Krantz DS. Individual differences in behaviorally evoked cardiovascular response: temporal stability and hemodynamic patterning. Psychophysiology. 1990;27:605–619. [PubMed] [Google Scholar]
54. Kendler KS, Karkowski LM, Prescott CA. Causal relationship between stressful life events and the onset of major depression. Am. J. Psychiatry. 1999;156:837–841. [PubMed] [Google Scholar]
55. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases. Nat Rev Dis Primers. 2017 Jun 22;3:17038. [PubMed]
56. 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.
57. Kuhn M, Türp JC. Risk factors for bruxism. Swiss Dent. J. (2018) 128:118–24. [PubMed] [Google Scholar]

L

58. Lacey JL, Lacey BC. Verification and extension of the principle of autonomic response stereotyping. Am. J. Psychol. 1958;71:50–73. [PubMed] [Google Scholar]
59. 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.
60. Lindhe, J.; Liljenberg, B.; Listgarten, M. Some microbiological and histopathological features of periodontal disease in man. J. Periodontol. 1980, 51, 264–269. [Google Scholar] [CrossRef]
61. Linsky AS, Strauss MA, Colby JP. Stressful events, stressful conditions, and alcohol problems in the United States: a partial test of the Bales theory of alcoholism. J. Stud. Alcohol. 1985;46:72–80. [PubMed] [Google Scholar]

62. Llabre MM, Klein BR, Saab PG, McCalla JB, Schneiderman N. Classification of individual differences in cardiovascular responsivity. The contribution of reactor type controlling for race and gender. Int. J. Behav. Med. 1998;5:213–229. [PubMed] [Google Scholar]
63. Llabre MM, Klein BR, Saab PG, McCalla JB, Schneiderman N. Classification of individual differences in cardiovascular responsivity. The contribution of reactor type controlling for race and gender. Int. J. Behav. Med. 1998;5:213–229. [PubMed] [Google Scholar]
64. Lobbezoo F, Ahlberg J, Raphael KG, Wetselaar P, Glaros AG, Kato T, et al.. International consensus on the assessment of bruxism: report of a work in progress. J. Oral Rehabil. (2018) 45:837–44. 10.1111/joor.12663 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
65. Lowenthal B. The effects of early childhood abuse and the development of resiliency. Early Child Dev. Care. 1998;142:43–52. [Google Scholar]

M

66. Malek R, Gharibi A, Khlil N, Kissa J. Necrotizing Ulcerative Gingivitis. Contemp Clin Dent. 2017 Jul-Sep;8(3):496-500. doi: 10.4103/ccd.ccd_1181_16. PMID: 29042743; PMCID: PMC5644015
67. 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.
68. Medaiah S, Srinivas M, Melath A, Girish S, Polepalle T, Dasari AB. Chlorhexidine chip in the treatment of chronic periodontitis - a clinical study. J Clin Diagn Res. 2014 Jun;8(6):ZC22-5. doi: 10.7860/JCDR/2014/8808.4477. Epub 2014 Jun 20. PMID: 25121059; PMCID: PMC4129267
69. Mehrotra N, Singh S. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): May 8, 2022. Periodontitis. [PubMed]

N

70. Nader KO, Pynoos RS, Fairbanks LA, al Ajeel M, al-Asfour A. A preliminary study of PTSD and grief among the children of Kuwait following the Gulf crisis. Br. J. Clin. Psychol. 1993;32:407–416. [PubMed] [Google Scholar]
71. Nazir MA. Prevalence of periodontal disease, its association with systemic diseases and prevention. Int J Health Sci (Qassim). 2017 Apr-Jun;11(2):72-80. [PMC free article] [PubMed] Neurosci Biobehav Rev 2011 Apr;35(5):1291-1301.

P

72. Papapanou PN, Sanz M, Buduneli N, Dietrich T, Feres M, Fine DH, Flemmig TF, Garcia R, Giannobile WV, Graziani F, Greenwell H, Herrera D, Kao RT, Kebschull M, Kinane DF, Kirkwood KL, Kocher T, Kornman KS, Kumar PS, Loos BG, Machtei E, Meng H, Mombelli A, Needleman I, Offenbacher S, Seymour GJ, Teles R, Tonetti MS. Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol. 2018 Jun;89 Suppl 1:S173-S182. [PubMed]
73. Paykel ES. Stress and affective disorders in humans. Semin. Clin. Neuropsychiatry. 2001;6:4–11. [PubMed] [Google Scholar]
74. Peterson C, Seligman MEP. Causal explanations as a risk factor for depression: theory and evidence. Psychol. Rev. 1984;91:347–374. [PubMed] [Google Scholar]
75. Pietruska M, Paniczko A, Waszkiel D, Pietruski J, Bernaczyk A. Efficacy of local treatment with chlorhexidine gluconate drugs on the clinical status of periodontium in chronic periodontitis patients. Adv Med Sci. 2006;51 Suppl 1:162-5. [PubMed]
76. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. Lancet. 2005 Nov 19;366(9499):1809-20. [PubMed]

77. Polusny MA, Follette VM. Long-term correlates of childhood sexual abuse: theory and review of the empirical literature. Appl. Prev. Psychol. 1995;4:143–166. [Google Scholar]

R

78. Reissmann DR, John MT, Aigner A, Schön G, Sierwald I, Schiffman EL. Interaction between awake and sleep bruxism is associated with increased presence of painful temporomandibular disorder. J. Oral Facial Pain Headache. (2017) 31:299–305. 10.11607/ofph.1885 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
79. Ridgeway EE. Periodontal disease: diagnosis and management. J Am Acad Nurse Pract. 2000 Mar;12(3):79-84. [PubMed]
80. Ringsdorf WM Jr, Cheraskin E. Emotional status and the periodontium. J Tenn State Dent Assoc 1969 Jan;49(1):5-18

S

81. Schneiderman N, McCabe P. Psychophysiologic strategies in laboratory research. In: Schneiderman N, Weiss SM, Kaufmann PG, editors. Handbook of Research Methods in Cardiovascular Behavioral Medicine. New York: Plenum; 1989. pp. 349–364. [Google Scholar] Sci 1996 Aug;104(4 (Pt 1)):327-334.
82. Selye H. The Stress of Life. New York: McGraw-Hill; 1956. [Google Scholar]
83. Shaw JA. Children exposed to war/terrorism. Clin. Child Fam. Psychol. Rev. 2003;6:237–246. [PubMed] [Google Scholar]
84. Sheiham A, Nicolau B. Evaluation of social and psychological factors in periodontal disease. Periodontol 2000 2005;39: 118-131.
85. Short JL. The effects of parental divorce during childhood on college students. J. Divorce Remarriage. 2002;38:143–156. [Google Scholar]
86. Soares-Silva L, Tavares-Silva C, Fonseca-Gonçalves A, Maia LC. Presence of oral habits and their association with the trait of anxiety in pediatric patients with possible sleep bruxism. J. Indian Soc. Pedod.

Prev. Dent. (2019) 37:245–50. 10.4103/JISPPD.JISPPD_272_18 [PubMed] [CrossRef] [Google Scholar]
87. 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.
88. 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.

T

89. Tonetti, M.S.; Greenwell, H.; Kornman, K.S. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. J. Periodontol. 2018, 89, S159–S172. [Google Scholar] [CrossRef] [PubMed][Green Version]

W

90. Wasu, Shilpa Prashant, Prashant Wasu, Kaustubh Suresh Thakare, Bhagyashri Umale and Amit Kumar. “Stress and Periodontal Disease.” (2017).
91. Wiebe CB, Putnins EE. The periodontal disease classification system of the American Academy of Periodontology--an update. J Can Dent Assoc. 2000 Dec;66(11):594-7. [PubMed]

Y

92. Yap AU, Chua AP. Sleep bruxism: current knowledge and contemporary management. J. Conserv. Dent. (2016) 19:383–9. 10.4103/0972-0707.190007 [PMC free article] [PubMed] [CrossRef] [Google Scholar]

Z

93. Zhang JM, An J. Cytokines, inflammation, and pain. Int Anesthesiol Clin. 2007 Spring;45(2):27-37. doi: 10.1097/AIA.0b013e318034194e. PMID: 17426506; PMCID: PMC2785020
94. Zimmerman P, Wittchen HU, Hofler M, Pfister H, Kessler RC, Lieb R. Primary anxiety disorders and the development of subsequent alcohol use disorders: a 4-year community study of adolescents and young adults. Psychol. Med. 2003;33:1211–1222. [PubMed] [Google Scholar]