SCIENCE OF SEA SALT

First Edition

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Science of Sea Salt

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PREFACE

The mounting drift of marine natural products has been recently boosted by the spiraling inclination to recognize the immense health benefits of different biological activities. As new medical discoveries enhance biotechnology innovations, significant attention is focused on the ocean's natural resources, because the marine ecological setting is a unique storehouse of novel bioactive natural compounds.

Historically, natural products have been used since ancient times and in folklore for the treatment of many diseases and illnesses. Natural sea salt is produced by the process of evaporation of salt water bodies. The methodology involved in procuring sea salt also helps retain its natural state and therapeutic qualities. Sea salt contains 82 trace minerals which have been scientifically proven to be essential to the maintenance of optimal health, like magnesium, calcium, sulphur, bromide, potassium, zinc, and sodium. A great deal of scientific attention in terms of biomedical science, research studies, and clinical observation resulted in new discoveries about sea salt's enormous potential and its role in therapeutic science.

In the current book, the editor and authors have called attention to the science of sea salt. With key sections of the book covering basic human biology, ocean frontiers, sea salt discovery, sea salt in medicine, including oral biology, sea salt in food science, body modifications and innovations, including new inventions in the field of sea salt product developments. This book focuses on basic learning and clinical guidelines in the ongoing effort to further collect new science and development. The information provided in this book will be extraordinarily helpful in understanding the potential and resourcefulness of the ocean and sea salt in particular.

(Rajiv Saini)
Editor Content Lead,
Science of Sea Salt

Eddie Kolos

Eddie Kolos was one of the early believers in the science of sea salt. His unwavering dedication to researching ocean life and the importance of natural elements in the ocean earned him a patent for the reconstitution of ocean water in a purified sea salt solution to be used in or on the skin in the United States and other countries around the world. He began his entrepreneurial journey two decades ago in 2001, successfully laying the groundwork for his three companies (H2Ocean, Ocean Aid, and Vet-Aid) in Stuart, Florida. By thinking outside the box and seeking the natural healing power of the ocean, he developed innovative sea-salt based skin and oral care products which are now well established in the industry.

His work and products are well known in the medical field, leading to professional recognition such as the American Podiatric Medical Association Seal of Acceptance, The 2012 Governor's Innovation Awards "Top 50 Florida Companies to Watch", and the 2013 AT&T Innovation Award at the Environmental Stewardship Awards. Eddie is also at the forefront of ocean preservation with his creation of Project Blue Green which is entirely focused on restoring and preserving the ocean to help sustain the blue and green Earth as a whole. While he is working to protecting the world's oceans, he is also dedicated to the future development of sea salt-based products and innovations in the field of saving lives.

RAJIV SAINI

Rajiv Saini, holds multiple academic and research positions. He is a visiting professor at the University of Bari Aldo Moro, Bari, Italy; a visiting scientist at the Tecnologica Research Institute in Crotone, Italy; and the Stony Brook School of Dental Medicine in New York, USA. Rajiv is also involved in building science-driven scientific strategy and management consultancy for industry. The central motivating theme of all his previous research has focused on studying the host's response to periodontal infection, which triggers a complex cascade of inflammatory-immune sequential events that make periodontitis an established risk factor for many systemic diseases, such as diabetes, cancer, and cardiovascular events. Rajiv's research interests include: Prevention Deficit Human Disease Mapping, Clinical Trial & Research Data Mining, Pro-Active Intervention & Prevention, Oral Systemic Integration, Human Microbiome, Artificial Intelligence in Health Care, Clinical Research Quality Index Analysis, Systemic Health Balance, and Nutritional Bio Complex. His work in this field is well accepted and has yielded 170 peer-reviewed publications in scientific journals of repute. He has already published four books *(Dental Horizons: Essentials of Oral Health, Hospital Infection Control: Clinical Guidelines, Fundamentals in Clinical Trial and Research, and Dental Plaque: A Biofilm)* and authored three book chapters. Rajiv also serves in various editorial positions in scientific journals and on the editorial boards of over 25 scientific journals. He has attended several scientific conferences, presented his research work and awarded with various International, National and Institutional awards. He has completed numerous research studies and is currently involved in multiple investigational research activities. He is frequently invited as a guest speaker at international and national academic forums.

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ORAL SYSTEMIC LINKAGE

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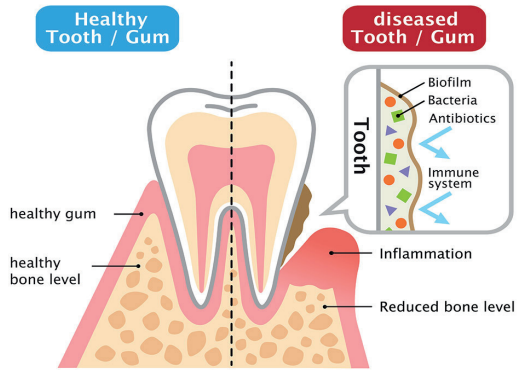
The Surgeon General's report on oral health highlights the relationship between oral and overall health, emphasizing that oral health involves more than dentition.[1] Mouth acts as a window to a lot of systemic diseases and serves as a port of entry of the various infections that can alter and affect the immune status of the person. The oral cavity has the potential to harbor at least 600 different bacterial species, and in any given patient, more than 150 species may be present, surfaces of tooth can have as many as billion bacteria in its attached bacterial plaque (Fig. 28.1) and oral care may not only reduce the microbial load of the mouth but the risk for pain and oral infections as well.[2] Periodontitis is defined as an inflammatory disease of supporting tissues of teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession or both.[3] Periodontal disease is a complex infectious disease resulting from interplay of bacterial infection and host response to bacterial challenge, and the disease is modified by environmental, acquired risk factors and genetic susceptibility.

Fig 28.1: Bacterial accumulation around teeth

Dental plaque represents a classic example of both a biofilm and a microbial community, in that it displays emergent properties, i.e., plaque displays properties that are more than the sum of its constituent members,[4] and microbial communities are ubiquitous in nature and usually exist attached to a surface as a spatially organized biofilm. Periodontitis and periodontal diseases are true infections of the oral cavity. There is an equilibrium that exists between microbial challenge and host's immune response; any alteration to that with addition of other modifying factors is responsible for clinical manifestation of periodontal disease. Pathogens of the subgingival microbiota can interact with host tissues even without direct tissue penetration, and the subgingival microbiota accumulate on the oral cavity to form an adherent layer of plaque with the characteristics of a biofilm (Fig. 28.2). The oral cavity works as a continuous source of infectious agents, and its condition often reflects progression of systemic pathologies. One of these is based around the potential for the inflammatory phenomenon of periodontitis to have effects by the systemic dissemination of locally produced mediators such as C-reactive protein (CRP), interleukins -1 beta (IL-1β) and -6 (IL-6) and tumor necrosis factor alpha (TNF-α).

Fig 28.2: Healthy tooth compared with bacterial biofilm



Acute periodontal disease primarily involves a local innate immune response to the microflora of the oral biofilm. Gingival epithelial cells recognize bacterial cell components via toll-like receptors (TLRs) and respond by producing IL-1 and TNF-α. Bacteria and bacterial products also penetrate into the underlying tissues. There they interact with fibroblasts and dendritic cells. These cells also produce proinflammatory cytokines. Additional immune signals are generated by alternative complement activation. These bacterial products and proinflammatory cytokines affect vascular endothelial cells as well. Endothelial cells express cellular adhesion molecules (ICAM and VCAM) that recruit circulating immune cells. Vascular permeability is also increased – allowing the influx of phagocyte cells and serum into the gingival tissue. Neutrophils and macrophages are attracted to the site of infection by chemotaxis following gradients of complement proteins, cytokines, and bacterial products. Activated macrophages produce IL-12 and interferon-gamma (IFN-γ). Overall, these processes result in gingival inflammation, and are responsible for the clinical manifestations of gingivitis further leading to periodontitis.[5]

CARDIOVASCULAR DISEASES

There is strong relationship between the periodontal and cardiovascular diseases and two directions have been the focus of delineating the relationship (Fig. 28.3): (I) bacteria from the oral cavity directly exacerbating the cardiovascular disease or altering systemic risk factors for cardiovascular disease; and, (II) the chronic periodontal inflammation at the focus of infection increasing circulating levels of host inflammatory macromolecules, and/or bacteria translocated to the circulation eliciting elevations in systemic host inflammatory macromolecules that exacerbate cardiovascular disease directly or alter other systemic risk factors for cardiovascular disease.[6] Several theories exist to explain the link between periodontal disease and heart disease. One theory is that oral bacteria can affect the heart when they enter the blood stream, attaching to fatty plaques in the coronary arteries (heart blood vessels) and contributing to clot formation. Coronary artery disease is characterized by a thickening of the walls of the coronary arteries due to the buildup of fatty proteins. Blood clots can obstruct normal blood flow, restricting the amount of nutrients and oxygen required for the heart to function properly (Fig. 28.4). This may lead to heart attacks. Another possibility is that the inflammation caused by periodontal disease increases plaque buildup, which may contribute to swelling of the arteries. Researchers have found that people with periodontal disease are almost twice as likely to suffer from coronary artery disease as those without periodontal disease.[7] Oral pathogens and inflammatory mediators (IL-1β, TNF-α) from periodontal lesions intermittently reach the bloodstream inducing chronic low-level bacteremia and systemic inflammatory reactants (C-reactive protein, systemic antibodies), all of which may represent a pathogenic link between periodontal disease and heart disease.[8]

Fig 28.3: Gingival inflammation bacteria affecting heart

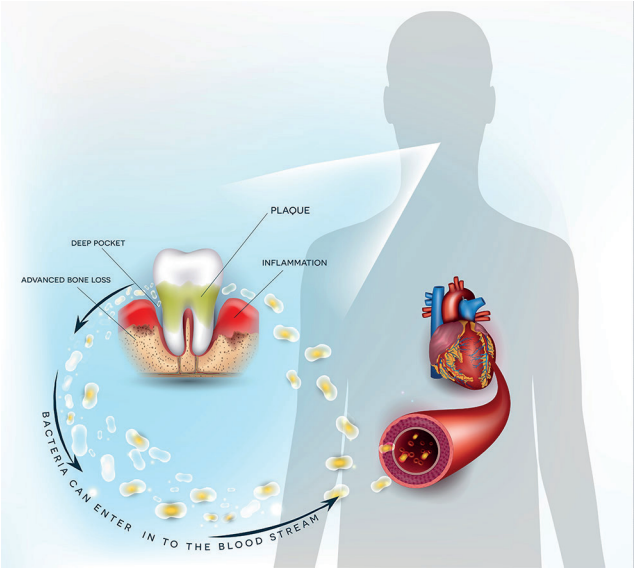
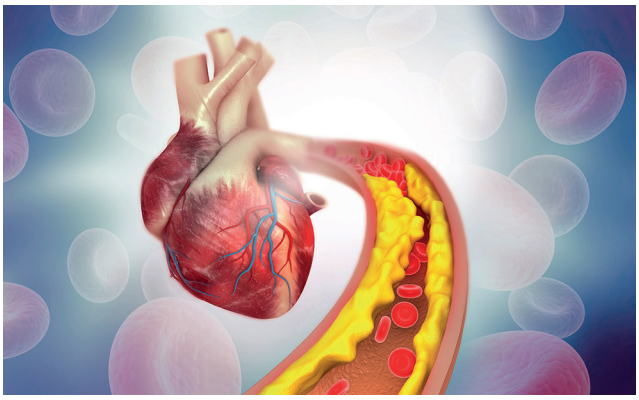


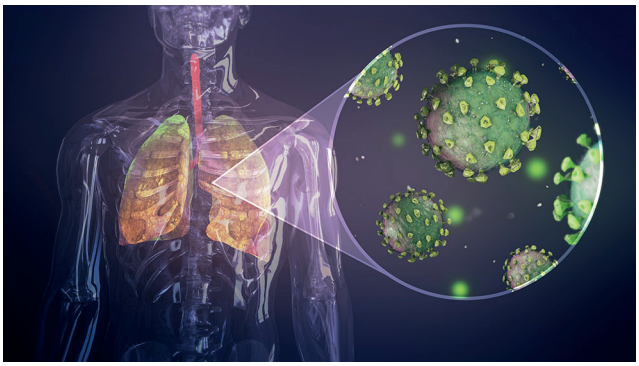
Fig 28.4: 3D illustration of plaque in artery supplying blood to heart



RESPIRATORY COMPLICATIONS

Respiratory infectious diseases such as bacterial pneumonia and chronic obstructive pulmonary diseases (COPD) are common and costly, especially in institutionalized and elderly inpatients. Respiratory infection is thought to rely in part on the aspiration of oropharyngeal flora into the lower respiratory tract and failure of host defense mechanisms to eliminate the contaminating bacteria, which then multiply to cause infection (Fig. 28.5). It has been suggested that dental plaque may act as a reservoir of respiratory pathogens, especially in patients with periodontal disease.[9] Several mechanisms have been proposed to explain the potential role of oral bacteria in the pathogenesis of respiratory infection, which include the following: (1) aspiration of oral pathogens (such as *Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans*, etc.) into the lung to cause infection; (2) periodontal disease-associated enzymes in saliva may modify mucosal surfaces to promote adhesion and colonization by respiratory pathogens, which are then aspirated into the lung; (3) periodontal disease-associated enzymes may destroy salivary pellicles on pathogenic bacteria to hinder their clearance from the mucosal surface; and (4) cytokines originating from periodontal tissues may alter respiratory epithelium to promote infection by respiratory pathogens.[10] In elderly patients living in chronic care facilities, the colonization of dental plaque by pulmonary pathogens is frequent. Notably, the overreaction of the inflammatory process that leads to destruction of connective tissue is present in both periodontal disease and emphysema. This over reaction may explain the association between periodontal disease and chronic obstructive pulmonary disease (COPD), the fourth leading cause of death in the United States; these findings underline the necessity for improving oral hygiene among patients who are at risk and those living in long-term care institutions.[11]

Fig 28.5: 3D illustration of respiratory infection



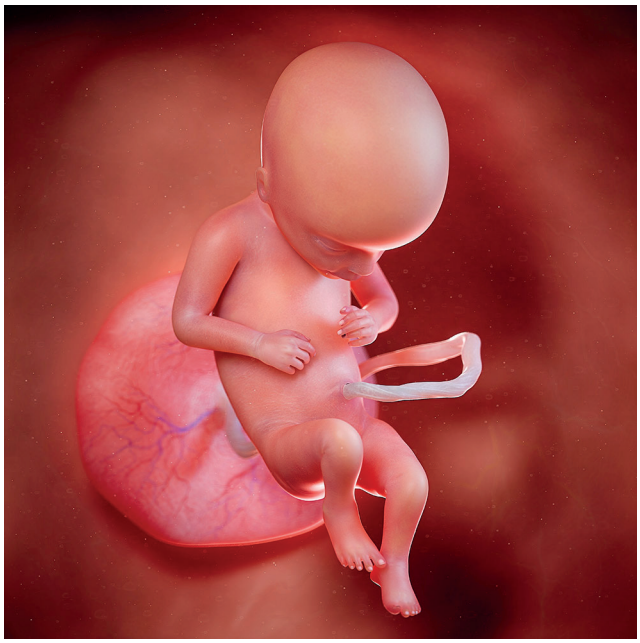
PREGNANCY COMPLICATIONS

Preterm delivery (PTD) is the major cause of neonatal mortality and of nearly one-half of all serious long-term neurological morbidity. Premature and low-birth-weight (PLBW) infants are still 40 times more likely to die during the neonatal period (Fig. 28.6). Research suggests that the bacteria that cause inflammation in the gums can actually get into the bloodstream and target the fetus, potentially leading to PLBW babies. One possible mechanism begins with endotoxins resulting from Gram-negative bacterial infections (such as periodontal disease). These endotoxins stimulate the production of cytokines and prostaglandins (IL-1β, IL-6, and TNF-α) and in appropriate quantities stimulate labor,[12] and proinflammatory mediators may cross the placenta barrier and cause fetal toxicity resulting in preterm delivery and low-birth-weight babies.[13] High concentrations of these cytokines, in pregnant women, are responsible for rupture of the uterine membranes causing premature birth and retardation. Several animal and clinical studies clearly indicate an association between periodontal infection and adverse pregnancy outcomes. Although no definitive

causal relationship has been established, and other explanations for the correlation might be offered, a model can nevertheless be envisaged wherein chronic periodontal infection could mediate this systemic effect through one or more of the following mechanisms:[14]

- Translocation of periodontal pathogens to the fetoplacental unit
- Action of a periodontal reservoir of Lipopolysaccharides (LPS) on the fetoplacental unit
- Action of a periodontal reservoir of inflammatory mediators (IL-1, IL-6, TNF-α, PGE2) on the fetoplacental unit.

Fig 28.6: 3D illustration of human foetus

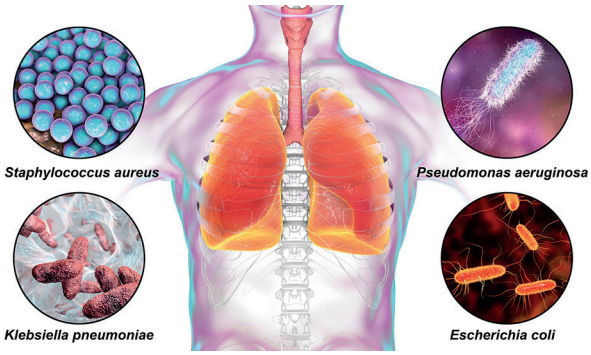


Thus, it can be concluded that periodontal disease appears to be an independent risk factor for PLBW and there is a need to expand accelerated oral health hygiene measures for pregnant women. Periodontal disease may burden pregnant women systemically with endotoxin, inflammatory cytokines, and oxidative stressors at the maternal-fetal interface; thus, it may be a vascular stressor that plays a role in the development of pre-eclampsia in pregnant women.[15]

VENTILATOR ASSOCIATED PNEUMONIA

Recent evidence indicates that colonization of the mouth with respiratory pathogens may contribute to the ventilator- associated pneumonia (VAP). Oral care may be an important preventive measure against VAP and not merely a comfort measure.[16] Normal oral flora has been shown to be altered in intensive care unit (ICU) patients, with the normal aerobic oral organisms being replaced by mainly Gram-negative organisms (Fig. 28.7), and the micro aspiration of oropharyngeal secretions is well recognized as a significant risk factor in the development of VAP.[17] Critically ill patients also have impaired immunological deficiencies and may be unable to respond to bacterial invasion of the lungs. Pathogens commonly responsible for the nosocomial pneumonia in ICU patients were found to colonize in the dental plaque and oral mucosa of these patients.[18] Assessment of the oropharynx and maintaining a favorable level of hygiene are difficult tasks to perform in both critically ill and in orally incubated patients due to the lack of access to the oral cavity.[19] The orally incubated patient is at an even greater risk of colonization of organisms because mouth care is often hampered by the presence of tape, tubes, and bite blocks.[20] Oral hygiene may provide a simple and cost-effective method of reducing the incidence of VAP and consequently morbidity and mortality in ICU patients.

Fig 28.7: 3D illustration of human respiratory pathogens

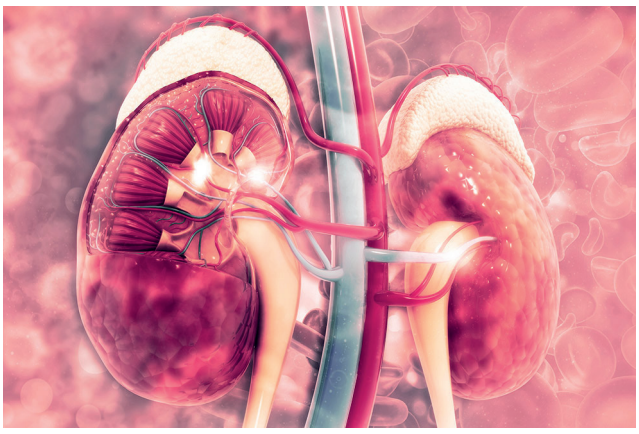


KIDNEY DISEASE

Chronic renal failure (CRF) is defined as a progressive decline in renal function associated with a reduced glomerular filtration rate (GFR), as measured clinically by the creatinine clearance rate. Several changes occur in the oral cavity in patients with CRF and uremia (Fig. 28.8).

Researchers estimate that up to 90% of renal patients will show oral symptoms.[21, 22] The most commonly discernible oral manifestations of CRF and related therapies are gingival enlargement, periodontal disease, xerostomia, malodor, mucosal lesions, oral candidiasis and other dental anomalies.[23] Perhaps the most common oral finding is pallor of the mucosa secondary to the anemia commonly seen in patients with renal failure who are undergoing hemodialysis.[21,24-26] These problems may be related to a variety of factors, such as a relative state of immunosuppression, medications, renal osteodystrophy and bone loss, and restriction of oral fluid intake. Untreated dental infection in immunosuppressed individuals can potentially contribute to morbidity and transplant rejection. Promoting good dental hygiene reduces the risk of oral infections that may predispose a patient to septicemia, endocarditis and possible endarteritis of the vascular access or line for hemodialysis or, of catheters for peritoneal dialysis.[27] It has been recommended that patients requiring a renal transplant have a detailed oral assessment and treatment prior to surgery, perhaps highlighting a need for appropriately trained oral health professionals to collaborate with renal physicians. The dental examination should be timed appropriately to allow any necessary dental treatment to be carried out in a planned manner. Most transplant centers worldwide have dental check-up in their pre-transplant protocol.[23]

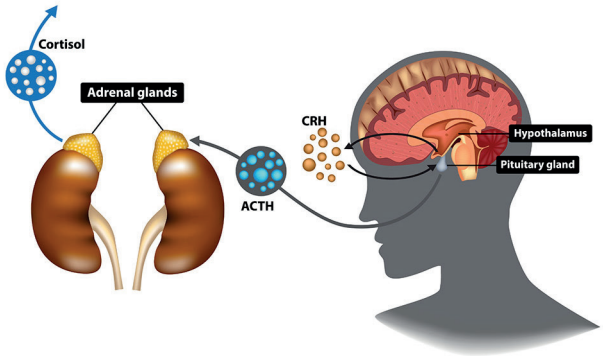
Fig 28.8: 3D illustration of human kidneys cross-section



PSYCHOLOGICAL STRESS

There are several studies that have demonstrated a relationship between psychological stress and inflammatory diseases such as rheumatoid arthritis and periodontitis. Even though investigators have studied the impact of the immune response and of psychological components on the extent and severity of periodontitis, few studies have evaluated the impact of relationship among psychosocial well-being, status of immune system and the health of periodontium, and the hypothesis was that stress elicits hypothalamus-pituitary-adrenal axis hyperactivation and leads to periodontitis (Fig. 28.9).[28] Several correlation questionnaires studies have observed a positive relationship between psychological stress and periodontal diseases. Deinzer et al.[29] analyzed the effects of academic stress on periodontal health, and Linden et al.[30] correlated occupational stress and periodontitis. In addition to bacterial infections, adverse effects of immunological changes, in particular, stress factors, may represent precipitating parameters of inflammatory periodontal diseases.[31] The impact of stress on the immune system has been well established, and a possible influence on chronic inflammatory diseases like periodontitis appears probable. Nevertheless, more recent studies indicate that psychological stress represents a risk indicator for periodontal disease, and should be addressed before and during the treatment.[32]

Fig 28.9: Hypothalamic pituitary adrenal (HPA) axis: Stress Response System

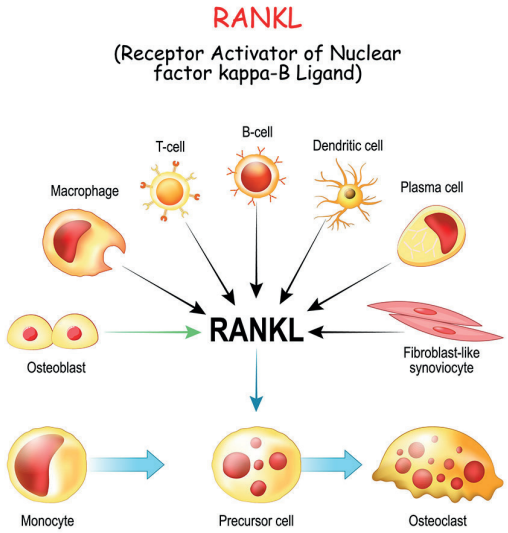


RHEUMATOID ARTHRITIS

Recently several mechanisms have been proposed for relationship of periodontitis

leading to rheumatoid arthritis (RA). RA is also a chronic destructive inflammatory disease characterized by the accumulation and persistence of an inflammatory infiltrate in the synovial membrane that leads to synovitis and the destruction of the joint architecture resulting in impaired function.[33] The current paradigm for RA includes an initiating event (possibly a microbial exposure or a putative autoantigen) leading to significant synovial inflammation and tissue destruction. As for periodontitis, there is an accumulation of inflammatory cells (T and B lymphocytes, neutrophils, and monocytes), tissue edema, endothelial cell proliferation, and matrix degradation. RA is also modified by systemic, genetic, and environmental variables.[34] Rheumatoid arthritis susceptibility and severity have been associated with genetic markers. The main genetic marker is carried by the highly polymorphic HLA-DRB1 locus. However, as for RA, in the same overall population, a similar association was described between the HLA-DRB1 subclasses encoded for the same shared epitope and periodontal disease.[35] Both RA and periodontal disease are associated with an imbalance between pro-inflammatory and anti-inflammatory cytokines (Fig. 28.10). IL1-β and TNF-α are the main proinflammatory cytokines detected in rheumatoid joints and gingival tissues.[36] Emerging evidence now suggests a strong relationship between the extent and severity of periodontal disease and RA.

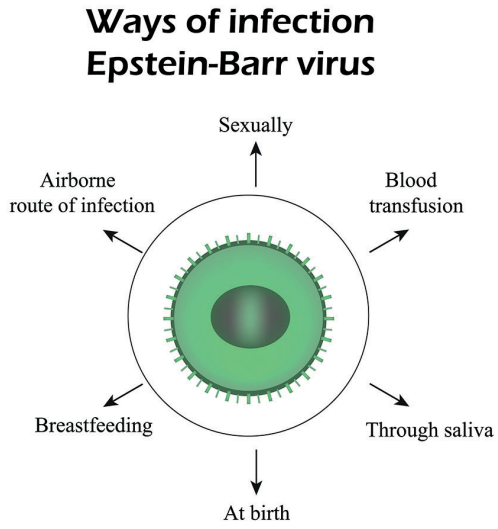
Fig 28.10: Receptor activation of RANKL



CANCER

Recent study in the Sweden stated that the chronic periodontal disease indicated by missing molars seemed to associate statistically with breast cancer. The study puts forward the idea that the risk of a woman developing breast cancer could be amplified by chronic gum disease. The study analyzed and evaluated over 3000 women between the ages of 30 and 40 years over a 16-year period. Among the women studied for the research, those who reported that they had suffered from chronic gum disease or had lost teeth due to periodontal disease were found to be more than two times as likely to be diagnosed with breast cancer as compared to those who had healthy gums.[37] Due to poor oral hygiene and gram-negative anaerobic bacterial infection, chronic periodontitis is closely associated with human cytomegalovirus and Epstein-Barr virus co-infection (Fig. 28.11). It is supposed that these viruses act together to control immune response to bacterial challenges; these viruses and bacteria act together to lead to low-degree chronic inflammation and carcinogenesis. Periodontal disease is associated with an increased production of reactive oxygen species which, if not buffered sufficiently, cause damage to the host cells and tissues. [38, 39]

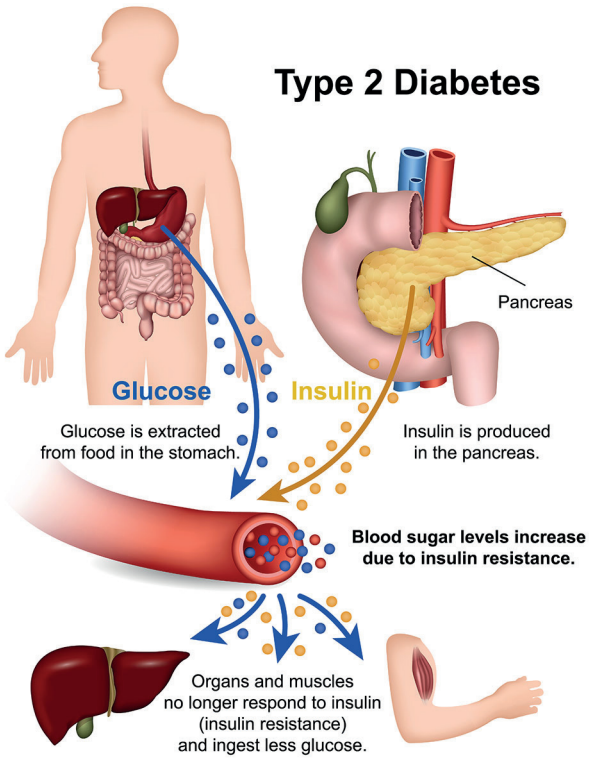
Fig 28.11: Epstein-Barr virus, methods of infection



DIABETES

The term "diabetes mellitus" (DM) describes a group of disorders characterized by elevated levels of glucose in the blood and abnormalities of carbohydrate, fat and protein metabolism.[40] DM is a metabolic disorder characterized by impaired action (Fig. 28.12), secretion of insulin or both, resulting in hyperglycemia presents with the classical triad of symptoms: Polydipsia, polyuria and polyphagia which are often accompanied by chronic fatigue and loss of weight; thus DM is a true metabolic disorder, and thus affects every tissue in the body.[41] The pathogenesis of periodontal disease is complex because it reflects a combination of the initiation and maintenance of the chronic inflammatory process by a diverse microbial flora and its numerous bacterial products. The subsequent host response to this infection mediates a complex cascade of tissue-destructive pathways.[42] Additional factors contributing to this multifaceted local disease process in the oral cavity include a number of systemic diseases, especially diabetes, that can exaggerate the host response to the local microbial factors (for example: endotoxin), resulting in unusually destructive periodontal breakdown.[43] Periodontal disease also is associated with hyperglycemia; the poorer the control of DM is, the greater the risk of developing periodontal disease. In fact, aggressive periodontitis is recognized as the sixth complication of diabetes according to Löe H.[44] who concluded that multiple epidemiological studies have demonstrated that both type 1 and type 2 diabetes are predictors of periodontal disease when the systemic condition is poorly controlled,[42] the other five complications are retinopathy, neuropathy, nephropathy, cardiovascular disease and peripheral vascular disease.[41] Many studies have been published describing the bidirectional inter-relationship exhibited by diabetes and periodontal disease.[45] Studies have provided evidence that control of periodontal infection has an impact on improvement of glycemic control evidenced by a decrease in demand for insulin and decreased hemoglobin A1c levels.[45-48] In addition to periodontal infection and gingival inflammation, a number of other oral complications have often been reported in patients with diabetes. These include xerostomia, dental caries, Candida infection, burning mouth syndrome, lichen planus and poor wound healing.[45] Thus, it can be accomplished that DM pessimistically affects each microvasculature bed, and the soft tissues and bones supporting the teeth are susceptible. There is also strong evidence that the presence of periodontal disease is associated with increased cardiovascular morbidity in patients with DM. The evidence reviewed supports viewing the relationship between diabetes and periodontal diseases as bidirectional. Further rigorous, systematic study is warranted to establish that treating periodontal infections can be influential in contributing to glycemic control management and possibly to the reduction of the burden of complications of DM.[49] It is possible to propose at least two hypotheses for testing the relationship between periodontitis and diabetes.

Fig 28.12: Type 2 Diabetes illustration



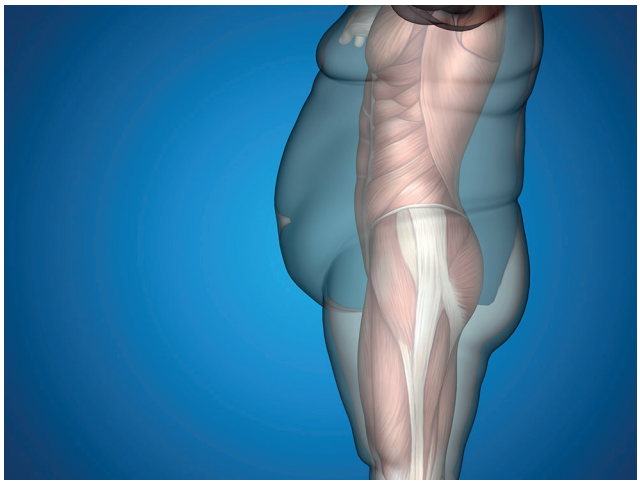
The first proposes a direct causal or modifying relationship in which the consequent hyperglycemia and hyperlipidemia of diabetes result in metabolic alterations which may then

exacerbate the bacteria-induced inflammatory periodontitis. The second hypothesis proposes that an unfortunate combination of genes (gene sets) could result in a host who, under the influence of a variety of environmental stressors, could develop both periodontitis and diabetes. This view is supported by the observation of common immune mechanisms involved in the pathogenesis of both diabetes and periodontitis; their genetic association with the HLA region of chromosome 6, where a number of genes involved in the immune response are situated; and the bidirectional association indicating that, not only is the prevalence of periodontitis higher in diabetics than in non-diabetics, but also that the prevalence of diabetes is higher in persons with periodontitis than in controls.

OBESITY

Obesity is a very prevalent chronic disease worldwide (Fig. 28.13) and has been suggested to increase susceptibility of periodontitis. The association between obesity and periodontitis is one of the most recent fields of research in periodontal medicine and the possible underlying biological mechanisms remain unclear. However, adipose tissue releases proinflammatory cytokines and hormones globally referred to as adipocytokines, which induce inflammatory processes and oxidative stress disorders, generating a similar pathophysiology between both diseases.[50,51] This association was first reported in animals in 1977 by Perlstein and Bissada[52], and in humans in 1998 by Saito et al.[53] Since then, the hypothesis that obesity is a risk factor for periodontitis has been evidenced by several epidemiological studies. Martinez-Herrera M et al [54] study results indicate the existence of an association between obesity and periodontitis, and although the causal mechanisms underlying this association remain unclear, the development of insulin resistance as a consequence of a chronic inflammatory state and oxidative stress could be implicated in the association between obesity and periodontitis.[54]

Fig 28.13: 3D illustration of obesity



ALZHEIMER DISEASE

There is evidence that periodontal disease may be a risk factor for dementia through the bacterial and viral infections commonly found in periodontal disease. Periodontal infections may result in elevating the systemic inflammatory response which in turn may contribute to existing brain and vascular pathologies that would impact brain function.[55]

OSTEOPOROSIS

The common factor between osteoporosis and periodontal disease is the excessive osteoclastic activity and bone loss initiated through chronic inflammatory conditions. This shared chronic inflammatory response may predispose individuals with periodontitis to osteoporosis. Estrogen modulates cytokines that regulate bone metabolism and the host inflammatory response. Lack of estrogen increases the number of osteoclasts causing an imbalance in bone metabolism and a reduction in bone density. Periodontitis also activates the inflammatory response and the osteoclasts. Many investigations have found a significant correlation between periodontal disease and estrogen deficiency. These two risk factors, working together, can induce osteoporosis.[56, 57]

STRESS

Although interactions between stress-endocrine-periodontal changes are not yet well- understood, some hypotheses have been proposed. It has been suspected that periodontal status is related to alterations in the concentration of adrenal corticoids by altering the response of oral tissues to bacterial toxins and other hormones involved in the general adaptation syndrome.[58] Direct association between periodontal disease and stress remains to be proven, which is partly due to lack of an adequate animal models, and difficulty to quantifying the amount and duration of stress. Furthermore, multiple variables affect the severity of periodontal disease, and there is uncertainty about the individual's onset of periodontal disease. Moreover, it is not possible to separate the effects of physical stress from emotional stress in these animal studies. In addition, it is likely that systemic diseases associated with periodontal disease such as diabetes, cardiovascular disease etc., may share psychosocial stress as a common risk factor. The available scientific evidence thus, does not definitively support a causal relationship between psychosocial factors and inflammatory periodontal diseases. The information reviewed above nevertheless does indicate the possible influence of psychosocial factors in the etiology of inflammatory periodontal diseases though at the moment, the more suggestive evidence relates to ANUG (acute necrotic ulcerative gingivostomatitis).[59]

CONCLUSION

Oral microbiome is defined as the collective genome of microorganisms that reside in the oral cavity. After the gut, it is the second largest microbial community in the humans. The microbial ecology of the oral cavity is complex, and is a rich biological setting with distinctive niches, which provide a unique environment for the colonization of the microbes.[60] The normal temperature of the oral cavity on an average is 37°C without significant changes, which provide bacteria a stable environment to survive. Saliva also has a stable pH of 6.5–7, the favorable pH for most species of bacteria. It keeps the bacteria hydrated, and also serves as a medium for the transportation of nutrients to microorganism.[61] More recently, the new molecular techniques based on next-generation sequencing (NGS) of the 16 rRNA gene of the bacterial genome, allowed to evidence the complexity of the bacterial component of the oral microbiome.[62]

The realization that oral health is linked to systemic disease and can affect the progression or development of diverse diseases has led to the search for biomarkers in the oral cavity that could detect systemic disease. The oral cavity is easily accessible, allowing for non-invasive tests in most cases; and patients usually visit dentists more often than general practitioners. Thus, use of the oral cavity for early diagnosis of systemic disease should increase the likelihood of successful treatment of many non-oral diseases.[63] The link between periodontitis and systemic disease remains the subject of continuous research and scientific validation of longitudinal studies. Oral health has a direct and/or indirect impact on the overall systemic health (Fig. 28.14). Medical specialists must recognize the developing and growing importance of oral health in comprehensive health care. Dentists must expand their awareness and clinical experience of related systemic conditions. Periodontal diseases and multiple systemic conditions share related genetic and/or environmental etiological factors, and therefore affected individuals may express manifestations of one or both diseases. Current treatment of plaque and oral diseases involve mouthwashes and professional teeth cleaning, and in more advanced cases, antibiotics or surgery.[64] The mechanical method of scaling and root planing is the gold standard for eliminating biofilm once established in the oral cavity (Fig. 27.15). However, novel preventive methods such as probiotics, nanotechnology, and natural oral rinses must be explored in order to maintain optimal oral health.

Fig 28.14: Periodontal complications

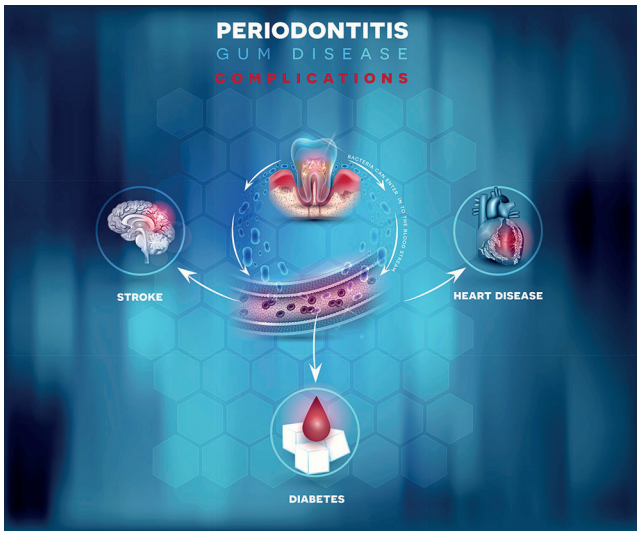


Fig 28.15: 3D illustration of scaling and root planing (conventional periodontal therapy)

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