Annals of
Reviews and Research

Juniper Publishers
key to the Researchers

Review Article
Volume 1 Issue 3 - February 2018

Ann Rev Resear

On Lines: Gum Lines and Waist Lines

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Submission: January 23, 2018; **Published:** February 13, 2018

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Abstract

Patients are concerned with esthetics of dentistry as well as looking healthy with esthetically pleasing with a pleasant looking body shape. With inflammation being posited as a common denominator in systemic diseases, oral inflammation has its place in contributing to better health and better quality of life. People pay enormous amounts to look fit and enhance their quality of life, but forget that oral health contributes to overall health. Diabetes, preterm low birth weight babies, periodontitis, gingivitis, cardiovascular disease, some cancers, rheumatoid arthritis are all examples of inflammatory diseases. Can the inflammation in one area exacerbate symptoms of inflammation in others? Use of a reasonably priced tooth brush in the correct technique has been shown to reduce gingivitis and assist in the maintenance of periodontal health. With obesity and periodontitis sharing the same inflammatory reactions, this manuscript will put forward a theory of waist line reduction being tied to gum line reduction of inflammation.

Keywords: Inflammation; Diabetes; periodontal disease; TNF-α, IL-6; immune system

Introduction

Clinical practice and clinical research has evolved so that there is more to consider when carrying out a differential diagnosis on a patient with disease be it systemic or orally related. A literature review shows that although no cause and effect can be proved between periodontitis and obesity, there are many factors contributing to the association. It is known that inflammation has myriad contributing factors with sequels as observed in humans. Can it be true that a relatively inexpensive toothbrush, used regularly at the gum line, contributes to healthier waist line measurements resulting in improved overall systemic health?

In the last few years, periodontal disease has been linked with many different systemic conditions such as cardiovascular disease, diabetes, and low birth weight, pre-term babies. A search in the literature data banks will reveal this to be true. In scientific terms, cause and effect relationships are difficult to prove, but the complexities of diseases known to man is inevitably due to factors we call risk factors. Smoking is associated with lung disease, extended exposure to ultraviolet rays of the sun can result in skin cancer; high intakes of alcohol may result in liver disease and so on. Just as in visible results of direct assaults of human to human contact, the underlying equalizer of many of the diseases can be traced to the process of inflammation. Inflammation may be "visually" localized in slight assaults (redness/swelling from a sliver in the finger) but nevertheless, there are systemic ramifications in the human body as demonstrated by laboratory tests of blood, saliva, or urine to pinpoint the disease. When one reads the scientific literature related to periodontal diseases, one is informed of the relationship of dental plaque biofilm located on oral tissues and the host's inflammatory reaction to it. Although specific bacteria have been implicated in periodontal disease, there are no laboratory tests to predict which healthy person is going to succumb to the disease and by which bacterium.

Humans have evolved by coping with assaults of many kinds. Host homeostasis dictates the levels within which the human can function comfortably. These limits are patrolled and through feedback loops, the body can adjust to cope with them. Tolerance levels are another coping mechanism demonstrated by the immune system's vigilance at all times [1]. The host can manage certain levels of assault that are not deemed dangerous unless it moves above or below that level drastically. The immune system evokes the process of inflammation to gain hold on the perpetrators - be they virus, bacterium, or fungus. Inflammation has been linked with diabetes, pre-term low birth weight babies, rheumatoid arthritis and control of HbA1c levels [2]. The inflammation process engages the immune system rather intensively [3] and arthritis is one such process. Periodontitis is a chronic inflammatory disease thought to be initiated by oral biofilm and perpetuated by the immune system [4] modulated through inflammation.

Being termed overweight and the term obesity are defined as abnormal or excessive fat accumulation that may impair health

[5]. According to the WHO, 2016, more than 1.9 billion adults, 18 years and older, were overweight and of those, there were over 650 million termed obese. The terms are based on body mass index (BMI) which can be calculated knowing a person's weight and height. In itself, this condition causes hardship for the patient in day to day life activities (eg. burden on the knees), and may lead to systemic problems (cardiovascular disease) causing death. It is difficult for the lay person to equate obesity with oral disease except for possible discomfort when eating or drinking. As dental professionals, we know that generally, only later stage oral disease can cause pain. The following excerpt from Goodson [6] (523) sums up the various ways obesity and oral disease can go hand in hand.

"The reasons for a relationship between obesity and oral bacteria are undoubtedly complex and varied. The relationship may be circumstantial, as being related to diet. It could be opportunistic, such as proliferation driven by metabolic changes that have occurred in the host. It could also be causal, as participating in initiation or propagation of the disease. Whatever the reasons, it is clear that the parallel microbiological universe that travels with many changes as many changes, and appears to be affected by a tendency to gain weight".

It remains then, that the possibility is real. With the persistence of obesity comes the reluctance to follow an exercise regimen possibly due to the burden of weight on the joints. Lack of exercise potentially contributes to further weight gain [7]. Suvan et al. [8] quoted Falagas & Kompoti [9], & Maury & Brichard [10] when they stated that obesity modulates a pro-inflammatory state of the host immune system. As we know that periodontal diseases are inflammatory in nature, we can surmise that such an inflammatory state modulated by the immune system may relate in the same manner as does obesity.

Inflammation overview

The process of inflammation is complex and involves the contribution from numerous host systems. When tissue is damaged, receptors in the tissue recruit assistance to contain any intruders - like pathogens - and to heal the affected area. The inflammation response has been studied and described elsewhere, essentially though, acute inflammation is usually attended to by white blood cells especially polymorphonuclear leukocytes. This innate immune system is recruited for protection and aggressive attacks on intruders in the acute stage. Basic responses of the acute stage include dilation of blood vessels increasing blood flow to carry the necessary cells that immigrate into the damaged area initiating containment and promoting healing. Healing tissues result in a diminishing inflammatory response and a return to normal activity via the balancing act of homeostasis. Destructive host reactions occur in chronic inflammatory situations when the host is never rid of the pathogenic agent and the innate immune system has to ramp up and sustain actions.

Agents of the Innate Immune System

In the initial stages of inflammation, leukocytes migrate into the damaged tissue for the destruction of intruders [11]. This reaction is immediate and is limited to acute inflammation. The leukocytes dominate for the first 24 to 48 hours (according to Gabay citing others). Chronic inflammation however, involves mononuclear cells, like macrophages and lymphocytes. The switch from acute to chronic inflammatory cell reactions is brought about by the production of cytokines from those macrophages and monocytes in particular [12]. The chronic inflammation associated cytokines are believed to include interleukin-6 (IL6), and tumour necrosis factor – alpha (TNF- α) among others [13,14]. Periodontal disease is a multi factorial inflammatory condition evoked by chronic retention of dental plaque biofilm harbouring gram negative bacteria that then shows elevated levels of these two cytokines in gingival crevicular fluids.

Genco et al. [15] reported that the inflammatory adipokines (TNF- α and IL-6) may be implicated in chronic periodontitis with resulting breakdown of periodontal tissue support. Graves & Cochran [16] reported TNF-α to be a key periodontal pathogen induced cytokine. In their report, they found that destruction in periodontal disease presented with elevated levels of TNF-α. It has also been reported that TNF-α stimulates the formation of osteoclasts (bone resorbing cells) and in response to bacteria in periodontal disease, regulates matrix metalloproteinases (MMP's) known to degrade connective tissue [17].

Polymorphonuclear leukocyte and macrophage production of IL-6, also a cytokine, can elicit both pro- and anti-inflammatory effects [18]. IL-6 is prominent in both destructive periodontal disease and in obesity. A study done earlier in the century showed that adipocytes secrete TNF- α [19] and that IL-6 is an inflammatory adipokine present in adipocytes15. Many studies read for this review have shown the elevated presence of these two cytokines in chronic periodontal disease [13-15,20,21]. Further reading directed toward the presence of adipocytes in obesity, showed that adipocytes produce adipokines and that adipokines are involved in periodontal disease [17,22]. Although TNF-α can be seen as a factor in periodontal disease the double edge effects of IL-6 are more difficult to pinpoint [13] and cannot therefore be referenced here. However, many studies on obesity presenting with chronic periodontal involvement can be found. The major focus is that they are both found in high levels in chronic periodontal disease and that they are both stored or produced in adipocytes.

Connecting the (Dots) – Cytokines

The possibility of obesity and periodontal disease playing upon each other may be still be under speculation, but it is tempting to connect the two especially with studies being published recently. According to Krejci & Bissada [23] who quoted Ritchie [24] & Yudkin [25], adipose tissue is not only a storage unit for adipocytes. It is believed that adipose tissue acts

How to cite this article: Sabrina Perdita Anthea Heglund. On Lines: Gum Lines and Waist Lines. Ann Rev Resear. 2018; 1(3): 555565.

as a metabolically active "organ" to secrete TNF-α and IL-6. These two inflammatory cytokines further induce liver production of the acute phase hepatic protein, C-reactive protein (CRP) [25].

TNF-α has been shown to increase insulin resistance facilitating obesity and has also been involved in the reduction of adiponectin [26]. Krejci et al. [23] stated that obese individuals were seen to have high levels of TNF-α and IL-6 compared to individuals with normal weight. Another hormone/cytokine produced in adipose tissue, leptin [21,27], -a known appetite suppressor with energy inducing, bone metabolism, reproductive functioning capability that works through a negative feedback control system, was noted in multiple sclerosis and obese individuals. But Margetic [28] postulated that an increase in levels of leptin in those individuals closely paralleled the insulin resistance seen in type-II diabetes.

Essentially, pro-inflammatory cytokines are evident in both obesity and periodontal disease. Interestingly, the inflammatory precursors in type II diabetes come from the same group of cytokines [29]. Genco et al. [15] observed that obesity was a significant predictor of periodontitis and that insulin resistance could mediate the effects due to the high levels of TNF-α. Boesing et al. [30] went further to say that obesity is a risk factor for both inflammatory diseases – periodontal disease and diabetes. Diabetes has long been touted as being a risk factor for periodontal disease basically due to the slower healing in diabetics. Now obesity can be profiled as being a risk factor along with smoking as the overall, number one risk factor. Modifications in neutrophils, the first white blood cells on the scene of tissue trauma, exacerbates the initial acute inflammation into a chronic affair when the original incendiary factor of dental plaque biofilm is allowed to remain on the dentition especially at the gum line and below.

Dental Hygienists' First Tool of Choice – the Toothbrush

Dental hygiene students are introduced to their chosen profession in passionate welcoming speeches of being "the preventive specialist" for disease. Educators may have restricted their messages to being preventive specialists for dental disease. In the last two decades, the dental scientific minds have pursued the inflammation theory behind oral disease alongside the medical scientific minds dissecting the inflammatory role of the immune system in immune diseases. The wealth of knowledge has exponentially grown once the seeds of risk co-factors were planted. As dental hygienists, we see the ravages of gingivitis and periodontitis in varying degrees. We learn that dental plaque biofilm is the root of this evil. Later on in the studies of dental hygiene, at the degree level, cytokines and byproducts of inflammation are introduced as they relate to the immunological side of host protection in the oral cavity.

Immune related path ophysiology studies describe in detail how the innate and adaptive immune systems function in concert to reduce, eliminate, or wage "all out" battles against self or intruders. Homeostasis as learned in early biology classes may not have been taken seriously enough to warrant such a great appreciation of human life and its evolving nature of balancing acts with bacterial and viral onslaughts. The human functions best within narrow confines of acidity and alkalinity, oxygen and deoxygenated states, sodium levels for nerve function and the variability of sugars, hormones and bicarbonate content in the blood. The negative and positive feedback systems themselves are a subject to be studied on their own. The art and science of dental hygiene has never been more profound than it is today. Scientific growth in research has pushed dentistry and dental hygiene to levels far greater than cleaning and fillings. Still, the first tool of choice remains the toothbrush or- getting back to the basics of oral hygiene.

Inflammation –Itis

The suffix "itis" when applied to a body part or tissue means "inflammation or disease of" Merriam-Webster dictionary [31]. In dentistry and dental hygiene we are introduced to gingivitis and periodontitis as being inflammation of the gingiva or the tissues surrounding the teeth. We are also instructed in the capabilities of bacterial adhesions to the dental plaque biofilm on teeth as being the prime motivators for the "itis". Tooth brushing techniques are taught to us along with the measurements of dental plaque biofilm remaining after brushing. The removal of this dental plaque biofilm in an efficient manner has been seen in earlier dental articles to reduce the effects of inflammation. Early studies even induced the biofilm through human studies done on students being asked not to brush for several days. Brushing after a period of time demonstrated that the bleeding and other signs of inflammation were reduced and were then kept at bay with regular use of the brush on the teeth.

In the previous sections of this manuscript, inflammation was discussed in relationship with diabetes, obesity and periodontal disease. The human body reacts in the presence of inflammation with the various cytokines designed for action in different ways. Some attempt to reduce inflammation, others come along to clean up debris and still others when locked out of cells can continue pro-inflammatory actions with or without the irritant. Based on the belief that the body acts through secretion of healing biological mediators via the circulatory system, we have expanded our knowledge to learn that inflammation in the oral cavity can affect other systems in damaging ways. If inflammation is the basic response to obesity, diabetes, periodontal disease, rheumatoid arthritis, cardiovascular disease, and preterm low birth weight babies (among others), we as dental hygienists can reduce the morbidity of other diseases by assisting in the reduction of inflammation in the oral cavity.

Diabetes Control on Gingival Health

To connect the way in which tooth brushing can control an inflammatory disease, a study of the inflammatory condition of

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diabetes was explored. In this study [32], 37 subjects with type 2 diabetes (controlled and still uncontrolled) were followed after they learned the Bass method of tooth brushing using a paste containing triclosan. Triclosan is a broad spectrum antimicrobial agent that has anti-inflammatory effects on the mediators of inflammation [33]. The Bass method of brushing involves the bristles of the toothbrush being used at or slightly into the space between the gum and teeth.

In this study 32 subjects completed the 4 week experiment and were once again evaluated on glycemic control and bleeding on gentle probing. Bleeding was significantly reduced in the group that initially had effective glycemic control. The group that did not have good glycemic control at the start did not reach statistical significance after the paired *t test* was applied for gingival bleeding. Although both groups commenced with similar bleeding scores, the groups did show reduced bleeding at the end with only the controlled group showing greatly different values. In a discussion of the study, the use of triclosan may serve as a distractor to this manuscript.

However, the use of the tooth brushing technique must have accounted for the reduction of bleeding as was seen in both groups. As bleeding is a symptom of inflammation of the gingiva, one may deduce that inflammation was reduced in both groups. The interesting point is that both diabetes and gingival bleeding are thought of as factors in inflammation. The group that did not have glycemic control to the extent the other group did shows that a reduction of inflammation in one area may serve to reduce inflammatory mediators to a reasonable extent in another body system.

Diabetes, Periodontitis, Obesity, Tooth Brushes

From the information provided, a case for the use of toothbrushes in the reduction of oral inflammation can be postulated. Reduction of inflammation at the gum line can reduce inflammatory effects in the rest of the body. If obesity involves higher BMI and possible increase in waist measurements, it may benefit from the reduction of inflammation. If obesity and gum disease are usually present together, the reduction in one should lead to a favourable reduction in the other.

As dental hygienists, we are encouraged to consider evidence based processes and procedures to assist our patients to relatively good health. With all the evidence that is coming out in the research, we are promoting health to greater extents than we did before. We even take the quality of life as a factor that will improve with the retention or replacement of missing teeth. We are called upon for cosmetic tooth whitening, making a patients smile look brighter, suggestions on how to deal with discolored root canal treated teeth, and advice on straightening of teeth. We should consider the cosmetic size of the waist line to the quality of life with a pretty smile and better quality of life when we consider the use of toothbrushes at the gum line. What is the consensus? Do toothbrushes affect lines? Both gums and waists?

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