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LITERATURE REVIEW

Introduction

Bacterial Influence/ Studies

Like many infectious diseases, Periodontal Disease is initiated by a less complex microbe and can progress into a chronic disease with a mess of symptoms and conditions. Periodontal Disease is not a rare disease with contagious bacteria, but it is of importance to researchers because of how the human body responds to the presence and accumulation of bacteria on the oral surface. Periodontal disease and its chronic effects are the results of the combined actions of the virulent bacteria and the body's response to the infection (Baker and Roopenian 2002). The normal bacterial flora which inhabit our mouths live in a form of symbiosis with the human body and has caught the attention of dentists, dental researchers, scientists, and various anthropologists. Studies have provided dental researchers and anthropologists alike with a better understanding of the infection process that occurs in the oral cavity and the specific flora that survive in those particular conditions. We have been able to reject certain hypotheses on issues pertaining to the type of bacteria causing the infection and ultimately resulting in alveolar bone loss. Bacteria is quite abundant in the oral cavity, however the types of bacteria are rather limited to streptococcal species and other various gram positive and negative rods.

Current theory holds that the types of bacteria involved in the infection of the periodontium are the same indigenous bacteria that are used in the protection of the oral tissue (Hirsch and Clarke 1989, Van Palenstein 1981). The protective role of the bacteria is inherently conflicting with the idea that the same normal flora can cause severe inflammation and eventual alveolar bone loss. These species of streptococcus can proliferate at an extremely high rate and colonize rapidly. In some cases where oral hygiene is poor, the colonies of different streptococcal species can develop into dense plaques. As the bacteria accumulate into dense plaques, the ecology of those colonies becomes quite complex. The ratios of bacterial species change and continue to build-up under the changing environment (Hardie and Bowden 1976). Past hypotheses have suggested infection was a result of possible foreign pathogens or from an exogenous source of bacteria. This non-specific theory of the progression of infection to Periodontitis has been reviewed extensively and shows that the subgingival colonization by the normal flora has the virulence factors to cause severe inflammation. The bacteria causing the infection and inflammatory response of the gingival tissue are not foreign invaders, but the same normal species that populate even a healthy individuals mouth (Theilade 1986). If a foreign invader is to blame in this situation, it would be helpful to secure a method which targets that specific invader and importance of routine oral healthcare would be undermined.

Bone Morphology and Processes

The Periodontium is the coined term that covers all of the oral tissue from the attachment sites to the support tissue. The types of tissue range from gingival tissue, teeth, and even bone (Hirsch and Clarke 1989). It is necessary to understand the differences between these tissues and how the bacterial infection inflicts damage to all of them. Gingivitis, as many dentists or researchers claim, is sometimes referred to as a disease; however, it can also be seen as a host response (Clarke and Carey 1985). The inflammation of the surrounding tissue is a response to help ensure that further complications do not arise. The gingival tissue is an important section of the human body due to its defensive position it must uphold to maintain the integrity of the internal environment when bacteria builds up to dense plaques (Clarke and Carey 1985, Page 1986). Just as the HCL in the human stomach serves to help kill invading pathogens and other elements that may potentially make us ill, the oral tissue adapts and attempts to prevent bacterial build-up from progressing into other more complicated conditions. In severe cases, this infection and inflamed tissue can spread to surrounding bone and become problematic to the individual's health. Periodontitis, the extension of gingivitis into the surrounding bone, may appear in the form of horizontal or angular bone loss, but is not an automatic later stage (O'Leary 1988). Just as it is important to fully understand the importance of the human response to the bacterial build up, it is also important to understand the link between the immune system responses and bone.

One of the most significant aspects of severe cases of Periodontal Disease is the loss of alveolar bone; however, bone is remodeled continuously in our everyday experiences.

Bone remodeling occurs throughout the majority of an individual's life through a meticulous process involving the activity of osteoblasts and osteoclasts (Baker 2000).

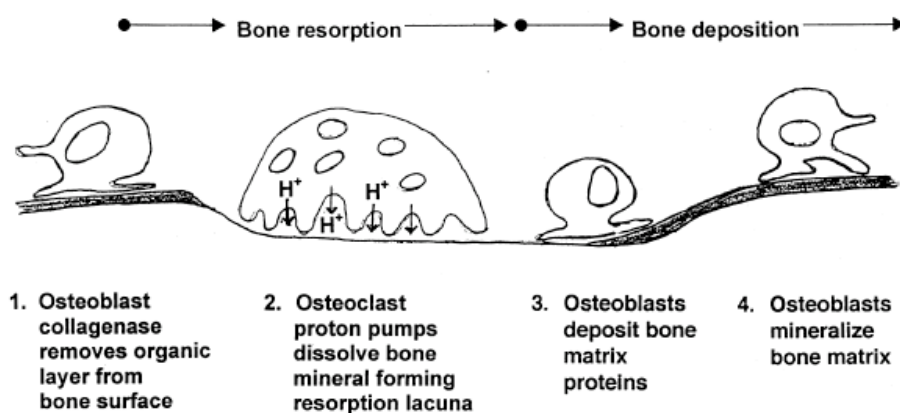


Figure 1. One cycle of normal bone remodeling (Baker 2000).

Bone is carefully broken down and then deposited in continuous cycles and is regulated by several different factors. Inflammation resulting from bacterial build-up as well as poor dental hygiene can ultimately conclude in alveolar bone loss. The close connection between bacterial colonization, the responses of the immune system, and the resorption of bone has been closely documented and grabbing the attention of many scientists (Baker 2000). It is becoming more apparent to scientists that the bacterial build-up causes an immune system response, which is closely linked to processes of bone loss. In a 2000 study by Pamela Baker from Bates College, she expressed how the immune system is intimately associated with bone regulatory factors. Osteoclasts, the cells responsible for bone resorption, share a common lineage with immune cells. Both cell types are present in the bone microenvironment as well (Baker 2000). This association

between the immune system and bone tissue is helping researchers better understand the microprocesses involved in the loss of bone. The study of bone loss is a complex subject with aspects reaching into various diseases, conditions, and genetic disorders. The loss of bone is one of the most important features of chronic Periodontitis and can vary between individuals as well as local sites within the same individual (Manson 1976). The two main factors to consider when discussing the effects of Periodontal Disease are the rates and form of the bone loss.

Case Studies

[This section will include some good examples of studies that are comparable to what I plan on accomplishing. I felt this section would be important to include despite its similarity to the Methodology section. This section will probably discuss less about the methods and more on how the researcher derived specific results. I have not researched this section nearly enough to discuss specific case studies, but I certainly plan on it. I most likely will rearrange this section to be closer to the Methodology section out of the close relationship they possess.]

Genetics/ Risk Groups

Ongoing research is helping inform experts on the roles of the human genome in the susceptibility and eventual progression of many diseases and conditions. The immunological and morphological aspects have been discussed, but other factors can have influential roles as well. There are multiple factors as well as problems in

determining the extent and severity of Periodontal Disease and many studies are suggesting the reality of a genetic susceptibility. Because much of the variance we see in populations is due to genetics, the issue of genetic susceptibility has become a hot topic for biologists and other researchers. For years, studies on monozygotic and dizygotic twins have revealed that about half of the population variance in disease can be attributed to genetic factors (Michalowicz 2000). There are a few main types of clinical studies that have helped suggest a genetic risk for diseases like chronic Periodontitis. These studies include those from monozygotic and dizygotic twins, genetic markers, and the incidence of Periodontal Disease in inherited diseases. The genetic factor is determined by allelic variation of host genes that confer susceptibility and resistance to the disease etiology (Baker and Roopenian 2002). Although Periodontal Disease is often considered a disease of the elderly, it can show severe signs in those much younger. Much of the evidence suggests a possible genetic foundation as to why at times there is an early-onset of the disease (Hart 1996). The discussion of this genetic factor to susceptibility of Periodontal Disease has led researchers to consider the possibility of certain groups of people where there is a noticeably higher risk (Hart and Kornman 1997).

Disease	MIM no.	Gene map locus	Inheritance	Comments
Leukocyte adhesion deficiency type 1	116920	21q22.3	Autosomal recessive	Disorder of neutrophil function resulting from deficiency of beta-2 integrin subunit of the leukocyte cell adhesion molecule.
Familial disseminated atypical mycobacterial infection	209950	2q33-37	Autosomal dominant resistance allele; autosomal recessive susceptibility allele	<i>Bcg</i> gene is expressed in 2 allelic forms, a dominant resistance allele and parasitic infection, and affects macrophage function.
Chédiak-Higashi syndrome	214500	1q42.1-42.2	Autosomal recessive	Clinical findings include neutropenia and abnormal susceptibility to infection.
Chronic granulomatous disease	306400	Xp21.1	X-linked recessive	In this disorder, neutrophils can phagocytose bacteria but cannot kill them in the phagocytic vacuoles. The cause of the killing defect is an inability to increase the cell's respiration and consequent failure to deliver activated oxygen into the phagocytic vacuole.
Specific granule deficiency (also called lactoferrin-deficient neutrophils)	245480	3q21-q23	Autosomal recessive	Lactoferrin has strong bacteriostatic properties and can deprive bacteria of the iron essential for growth. It may also protect cells from free radical damage. Patients with specific granule deficiency have normal neutrophil counts, but have a tissue (neutrophil)-specific absence of lactoferrin secondary to an abnormality of RNA production, possibly due to a defective granule-packaging gene.
Myeloperoxidase deficiency	254600	17q12-21	Autosomal recessive	Absence of myeloperoxidase, a dimeric protein that catalyzes production of intermediates with microbicidal activity against a wide range of microbes. Exaggerated superoxide production; several allelic variants have been identified.
Localized juvenile periodontitis	170650	4q11-113	Autosomal dominant	A major gene locus has been mapped to chromosome 4q. The genetic defect is unknown.

Figure 2. Disease conditions attributed to immune responses. As shown in Hart and Kornman 1997.

Individual responses to subgingival colonization can vary and these higher risk groups may provide researchers with a probable explanation (Griffiths 1988). Elevated risk groups are not limited to those with genetic disorders or simple variance. Some of the identified risk groups can include those who participate in activities or lack of proper hygiene. In many studies, smoking has been shown to be as big a contribution to the disease as the accumulation of bacteria (Offenbacher 1996). It's extremely important to not underestimate the importance of genetic variance or susceptibility; however, we must consider all factors. Some of these risk factors can include temporary physiological disturbances such as pregnancy, menstruation, and oral contraceptives (Hugoson 1980,

Lindhe and Attstrom 1967, Pankhurst et al. 1981). It is quite possible that certain drugs may interfere with the normal functioning immune system. As we have discussed earlier, there is an association between the immune system and bone resorption. Designer drugs and medications are notorious for possessing an array of side effects as well as various interactions with other drugs or medications. Diet and nutrition can be of significance in relation to development of Periodontal Disease, although it may be one of difficulty considering the multiple factors that are involved in food choice. Food intake can be intimately related to socioeconomic status among other things. Since ultimately all tissue growth, development, and maintenance depends on proper nutrition, the Periodontium is no exception (Wilton 1988). Immune systems can also be compromised without proper dietary intake and macronutrient levels, and a malfunctioning immune system may increase the likelihood of the development of Gingivitis.

Methodology

[For this section, I plan on diving into the main methods used in current periodontal research. I know there are several different indices that may be used, and I would like to examine as many as possible. I want to understand the main arguments for and against each method. This will come in handy when deciding which method would work best for my project. A lot of the research out there and published articles pertain to living subjects making it difficult to find appropriate research.]

Socioeconomic Studies

The idea of certain subgroups as more genetically susceptible to infection and alveolar bone loss has led many researchers to consider the possibility of socioeconomic subgroups. We have discussed the issues related to higher risk groups that may not be genetically determined; however, there may be an association between Periodontal Disease rates and distributions and socioeconomic groups. These social and economic subgroups would be more susceptible to Periodontal Disease or exhibit elevated rates of infection and alveolar bone loss due to a number of reasons. Until recent decades, studies or information in general relative to certain social or economic groups and rates of Periodontal Disease were scarce (Henry and Sinkford 1974). Before examining Periodontal Disease rates among different populations or samples, it is important to consider some of the main subgroups that may show clear distinctions in rates of the disease. One specific example is the comparison of different age subgroups. Periodontal Disease is not specifically a disease of the elderly, although trends do show elevated rates in older subgroups (Ingle 1975). Periodontal Index Scores for younger subgroups can vary from .13 to .54 while those in older groups may reach scores of over 2.5 (Ingle 1975).

<i>Age group and years</i>	<i>Mean PI</i>
Children, 6–11 years, 1963–65	0.13
Youths, 12–17 years, 1966–1970	0.31
Adults, 18–79 years, 1960–63	1.13
18–24	0.54
25–34	0.75
35–44	1.04
45–54	1.42
55–64	1.84
65–74	2.05
75–79	2.92

Figure 3. Average Periodontal Index score for children, youths, and adults as shown by Ingle and the National Center for Health Statistics.

These results are not uncommon among the vast clinical studies that have been done. Early studies in the late sixties and early seventies were already showing rates, distribution, and demography of Periodontal Disease. The relationship between groups of sex, age, and racial affinity are interesting demographic factors to consider when analyzing risk factors or susceptibility. In a Public Health Report from 1974, various demographic subgroups were analyzed and the appropriate Periodontal Index Scores were compared (Henry and Sinkford 1974). It is clear that there is a relation between specific subgroups and Periodontal Index scores, but further longitudinal studies are needed to best analyze demography and future preventative or precautionary measures. Early studies on Periodontal Index Scores and race have revealed interesting information regarding the prevalence and severity of the disease. Results have shown 27.8 percent of the “White” subgroup not showing signs of having Periodontal Disease, while only 15.8

percent of the “Black” subgroup not having the disease (Ingle 1975 and National Center for Health Statistics 1967).

<i>Race and sex</i>	<i>Without periodontal disease</i>	<i>With periodontal disease</i>	
		<i>Without pockets</i>	<i>With pockets</i>
White, both sexes	27.8	48.3	23.9
Men	22.4	48.7	28.9
Women	33.0	47.8	19.2
Negro, both sexes	15.8	48.2	36.0
Men	12.2	48.9	38.9
Women	19.1	47.6	33.3

Figure 4. Percentage of adults without Periodontal Disease as shown by Ingle and the National Center for Health Statistics.

Similar studies also show significant differences between the men and women as groups. Further studies are needed to help conclude this issue and possibly establish better healthcare or preventative measures for those subgroups at risk.

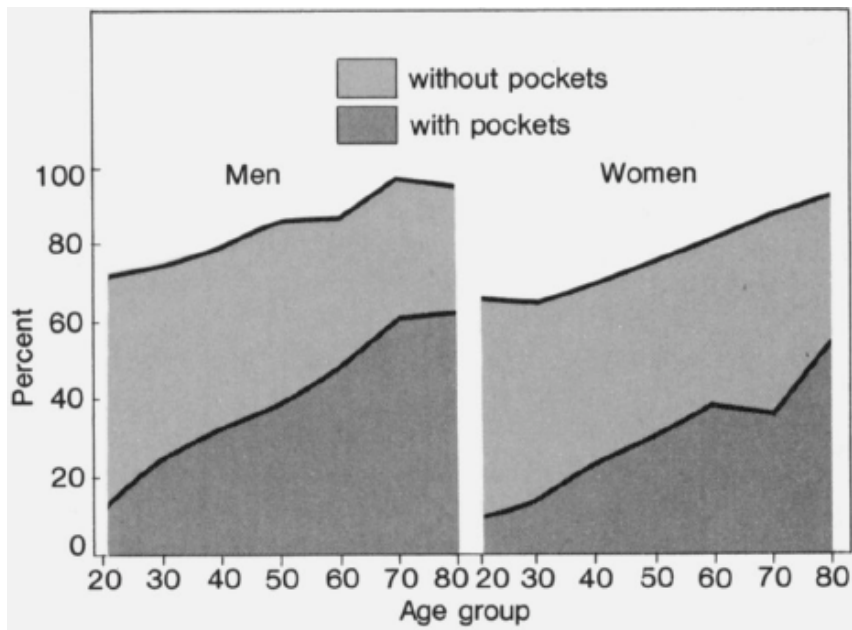


Figure 5. Percentage of men and women with Periodontal Disease, with and without pockets, and by age groups. As shown by Ingle and the National Center for Health Statistics.

Conclusions

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