Periodontal Disease in the Hamann-Todd Osteological Collection

M.A. Thesis Proposal

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I. Abstract:

The purpose of this study is to examine the presence and severity of periodontal disease in the Hamann-Todd Osteological Collection in Cleveland, Ohio. The collection is comprised of individuals from the area during the early 1900's. My hypotheses are: 1) There will be differences in severity between those of different ethnic backgrounds; 2) There will be an increase in severity of the disease in the older groups. This research will provide us with information regarding skeletal health in an impoverished and urban group of individuals. Periodontal disease is a condition that still affects populations around the world and this osteological collection allows us to see the osteological remnants of it.

My methods will consist of completing an inventory for each individual and then calculating the severity for each individual, ethnic group, and sex. All measurements of severity will be recorded on a dental inventory sheet I have designed. I will then test my hypothesis to see which ethnic and age group exhibit a higher degree of severity. I expect to find higher severity index scores for periodontal disease in the Black ethnic group as well as the older age groups.

II. Introduction:

It is not uncommon for an individual to assume that many oral problems such as receding gum lines, tooth loss, and even mandibular/ maxillary bone loss are signs of old age. Research from dental experts, biologists, disease specialists, and physical anthropologists have shown that the processes of these various disease conditions are formed by several composite factors. It is important to acknowledge the significance of bone chemistry, physiology, genetics, and the overall complexity of the immune system. Periodontal disease is a complex condition that can manifest itself in the bony remains of an individual and may then be examined in the deceased. The purpose of this study is to examine periodontal disease in the Hamann-Todd Osteological Collection in Cleveland, Ohio. Because the remains in the collection are mostly those from lower socioeconomic backgrounds, this study will enable me to investigate the severity of the disease in those with essentially decreased access to health care.

Modern dentistry and oral hygiene improvements have made it possible to combat many of these complications. Oral complications have plagued *Homo sapiens* for a considerable amount of time and the severity of some of these cases can be seen in fossilized remains (Molnar 2005). Periodontal disease, one of the most destructive oral diseases, is a condition that can potentially result in the loss of alveolar bone in the maxilla and mandible. It is a disease that affects many around the world with many of the cases seen in the archaeological record. There is a fairly large gradient of severity of the disease with some cases resulting in very little degradation while others show signs of serious damage. In many cases periodontal disease is initiated by an accumulation of bacteria near the gum line or in an existing location in the mouth where bacteria have access to the deep tissue in the gums. These locations can range from the gum line to a damaged tooth with an exposed root. As we will discuss later, the bacteria is non-site specific and results in an immune response from the individual. There are many types of organisms in the mouth that are present in both diseased individuals and those uninfected and in varying numbers (Merchant 2007). The inflammation that follows the build up of organisms as well as other factors such as genetics, can lead to the destruction of oral tissue and bone.

Along with improvements in dental care, research has provided the scientific community with genetic studies and susceptibility patterns for periodontal disease (Merchant 2007). Researchers have examined the occurrence of periodontal disease and considered the effect of hereditary influences (Kinane and Hart 2003). The common cell lineage between those cells responsible for bone breakdown and those pertaining to the immune system has motivated researchers to search for more elevated risk groups. The

possibility of elevated risk groups has been examined in various medical or genetic conditions as well as other important sub-groups such as those related to race and socioeconomic status. Research on periodontal disease is continuing to expand into fields previously not considered relative. The role of bacteria, genetics, bone morphology, diet, lifestyle, and other immunological factors are providing dentists and other researchers with vital data to help better understanding the disease process and potential treatments or precautionary methods (Merchant 2007).

I will test my hypothesis that older age groups will exhibit degrees of severity much higher than younger ones. I will also test the differences in severity between ethnic groups and the sexes. I will travel to the museum in Cleveland, Ohio to conduct the field research. Permission from the Museum of Natural History will be obtained before any severity research is conducted. After the on-site research, I will perform a statistical analysis using my findings to test my hypotheses and illuminate any major differences between my test sub-groups.

III. Background:

Periodontal diseases are infectious diseases caused by bacterial virulence factors and the host responses to those bacterial factors that together can destroy the attachment apparatus of teeth and can ultimately result in osteoclastic resorption of the alveolar bone of the jaw and loss of teeth (Baker and Roopenian 2002). This type of clinical description of periodontal disease is widely accepted, although ideas on more specific issues are sometimes still hotly debated. Bacteria are quite abundant in numbers in the oral cavity, however the types of bacteria are rather limited to streptococcal species and other various gram positive and negative rods. The bacteria can start from a small colony and progress

rapidly to an accumulation of extreme densities. Like many infectious diseases, Periodontal disease is initiated by a less complex microbe and can progress into a chronic condition. Periodontal disease is not a common disease, and 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 that inhabit our mouths and live in a form of symbiosis with the human body has caught the attention of dentists, dental researchers, disease specialists, an various anthropologists. This seemingly harmless and sometimes useful bacteria have proven themselves to be quite destructive towards the human oral tissue and bone. 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 can survive in those particular conditions.

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. Although the rate of bone remodeling varies throughout an individuals life, it can be influenced by factors such as age, nutrition, stress, and other environmental conditions. Bone is carefully broken down and then deposited in continuous cycles and is regulated by several different factors. The first step is for Osteoclasts to adhere to the exposed Hydroxyapatite crystals. Protons are generated by the osteoclast cells, which lower the pH of the Hydroxyapatite surface. This complicated and lengthy process leads to the eventual resorption of bone (Mundy 1991). Inflammation resulting from bacterial build-

up as well as poor dental hygiene can ultimately conclude in alveolar bone loss. Although gingival build of bacteria is not the sole instigator in the disease process, it plays a significant role. The close connection between bacterial colonization, the responses of the immune system, and the resorption of bone has been closely documented and are the focus of countless studies (Baker 2000). It is becoming more apparent that the bacterial build-up causes an immune system response, and that more work is necessary to identify key aspects of the disease progression.

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. Not surprising, both cell types are present in the bone microenvironment (Baker 2000). This association between the immune system and bone tissue is helping researchers better understand the microprocesses involved in the loss of bone. As we will discuss later, the association or link between the immune system and periodontal disease will prove to be significant as it relates to varying disease rates between sub-groups. The study of bone loss is a complex subject with influences such as various diseases, conditions, and genetic disorders that the individual already possesses. These diseases or conditions can range in influence from minor to extremely significant, but are of importance nonetheless. When attempting to identify high-risk groups for the disease, it is important to understand the influence of those same factors that potentially put them at a higher risk. 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 itself.

Robert Jurmain, from San Jose State University, examined overall dental health from the CA-Ala-329 site on the southeast side of the bay area of San Francisco (1990). His assessments included dental attrition, periodontal disease, caries, and other dental health indicators. His results show that some surface periodontal involvement is expressed in almost 75% of adults in the population. In this specific population near San Jose, exposure through the pulp cavity seems to be the major contributor to infection (1990). Moderate and severe lesions were present 46% of the time, and surprisingly, no sex differences were observed for periodontal disease rates. One of the most severe forms of the disease is infection that results in abscesses. For the San Jose site CA-Ala-329, 30% of individuals 11 years old or older had at least one abscess. What this study exposes, is the full range of the effects of the disease. It is important to understand both poles of the severity index of the disease.

Delgado-Darias and colleagues performed a study on the anthropological collection of the Museo Canario and examined a total of 791 individuals. The study focused on the rates and distribution of calculus deposition among individuals from two sites from the island. The sample is a prehistoric population with an observed periodontal disease rate of 66.78% (2006). An interesting note to this study was the higher prevalence of periodontal disease among men (72.47%) than compared to women (62.79%). As expected, the rate was much lower among the youngest individuals. Ante mortem tooth loss was seen in 64.73% of individuals with similar figures for tooth loss and periodontal disease seen in the two sites together. Delgado-Darias provides several statistical

associations between calculus deposition and periodontal disease, and sex and age differences through the use of non-parametric tests such as Kruskall-Wallis and Mann-Whitney tests. This particular study is useful due to its exposure of differences in disease rates between sex groups. This case study found a difference of almost 10% between disease rates of men and women.

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 sub-groups that may show clear distinctions in rates of the disease. One specific example is the comparison of different age sub-groups. Periodontal disease is not specifically a disease of the elderly, although trends do show elevated rates in older sub-groups (Ingle 1975). The role of ageing in the onset and progression of periodontal disease is complicated and difficult to isolate. Much of the alveolar damage that can occur may be due to the cumulative tissue and bone breakdown over an extended period of time. It has also been suggested that elderly individuals are at a much higher risk for onset and progression of the disease for multiple reasons, one of them being their age. Presently, there is conflicting evidence as to the role of ageing and if it is a significant predictive factor for the disease. Papapanou et al. (1989) reported age as a risk factor for alveolar bone loss while Brown et al. (1994) found no association. When considering other characteristics of periodontal disease such as clinical attachment loss, Ismail et al. (1990) and Norderyd et al (1999) found age to be a possible risk factor. It is reasonable at this point to understand that regardless of the

confounding variables, age is a viable factor in the equation of assessing the risk to periodontal disease and should be at least examined in further studies.

Heitz-Mayfield (2005) eloquently put the age variable into context by suggesting that the amount of tissue destruction relative to the patient's age is a good predictor of future disease progression. A much younger patient with a very aggressive form of the disease and showing signs of severe clinical attachment loss should be considered at a much higher risk for additional disease progression when compared to an elderly individual with exactly the same levels of tissue destruction. The younger individual has the potential to lose significantly more bone throughout his life if he has already experienced alveolar bone resorption. If traditional case studies suggest that older individuals show signs of the disease more than younger individuals, attention must be paid to those younger persons who exhibit signs of severe Periodontitis. We are fortunate enough to have survey data on various age groups ranging from sub adults to those almost in their 80's. Age data for periodontal index scores are quite abundant, including those studies from the early 60's.

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. The risks for many diseases, and this includes periodontal disease, is not equally distributed by all individuals in any population (Griffiths, G.S., Wilton, J. 1988). 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. An individual's genetic composition is a vital aspect of a host response-related risk (Kinane 2005). 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. Twin studies are some of the best ways of determining genetic influence for many diseases and conditions. Michalowicz et al (1991) reviewed dizygous twins reared apart and reared together, and monozygous twins reared apart and reared together. The measurements taken from marquis probing were found to differ significantly less for the monozygous twins reared together compared to the dizygous twins reared together. This twin study provides researchers with much more confidence that periodontal disease has a genetic component. Michalowicz (1991a) also examined alveolar bone height among twins and found significant variations related to genotype. Twin studies represent some of the best methods for isolating many outside variables and allows the researchers to better understand the components behind our innate variability in susceptibility to this and other diseases.

Diet and nutrition can be of significance in relation to development of periodontal disease, although it may be difficult considering the multiple factors that are involved in food choice. There is no debate as to if food helps maintain a properly working body, and a fully functioning immune system is not an exception. 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. Patients suffering from immune crushing treatments are made sure to stay healthy in their diets and activities. Diets rich in whole grains, fruits, and vegetables are recommended for maintaining the integrity of the dental tissue (Schifferle 2009). When an immune system becomes jeopardized, it must have access to resources and our nutritional health is partially responsible for keeping those resources at appropriate levels. If the immune system is jeopardized, and resource levels are less than adequate, serious damage could proceed.

Racial or ethnic groups are proving themselves as important sub-groups for the study of potential elevated risk groups. Several studies have reported a higher prevalence of periodontal disease in specific racial or ethnic groups and further studies could prove beneficial in their application to treatment and preventative methods (Brown et al. 1994). The concept of race as a subgroup is complicated however due to its possible associations with other sub-groups such as social and economic background. Certain geographic areas around the nation may be composed of a specific demographic or make up. Just as important is the overall income of each surveyed area. A survey done in Beverly Hills in California may not be demographically representative of the rest of the United States. Less financially stable populations will have fewer resources for health care or lack the insurance to ensure the proper dental hygiene. Less financially stable populations may also lack proper diets or have moderate to severe nutritional deficiencies. Obesity or smoking rates may be higher in specific racial or ethnic sub-groups and may complicate

results. The identification of racial sub-groups is of extreme importance when discussing disease rates in a multi-cultured and multi-ethnic test group.

Early studies on Periodontal Index Scores and race have revealed interesting information regarding the prevalence and severity of the disease. Some 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). A study by Albandar et al. (1997) found Black Americans to have the highest incidence of aggressive Periodontitis among schoolchildren. A later study by Albandar (2002) produced similar results of individuals with African heritage having the highest prevalence of Periodontitis.

Another possible elevated risk group may include one of the sexes compared to the other. Many of the studies done have not only included age, ethnicity, and weight, but also the sex of the individual. Multiple studies suggest significant differences between men and women as groups. Attachment loss in periodontal disease has been shown to occur much more in males compared to females (Hyman and Reid 2003). In this study, males were at an increased risk of attachment loss after all confounding variables were accounted for. Significant attachment loss was found in 23%, 44%, and 55% more males than females. Albandar (2002) suggests it is the hormonal and behavioral differences that may be a factor for increased risk for Periodontitis. These possible differences between the sexes require more research to better understand the hormonal and behavioral differences between the sexes and how it affects periodontal disease rates. Further studies are needed to help conclude this issue and possibly establish better healthcare or preventative measures for those sub-groups at risk.

One of the most significant factors in the initial onset and progression of periodontal disease is the use of cigarettes (Beck et al. 1990, Bergstrom and Preber 1994, Grossi et al. 1994, Machtei et al. 1997, Tomar and Asma 2000, Hyman and Reid 2003). A series of long-term studies have helped convince any skeptics about the elevated risk of smokers compared to non-smokers. Paulander et al. (2004), in a 10-year study examined the incidence of bone loss and periodontal disease in a sample of 50-year olds. It was found that smoking was the strongest predictor for alveolar bone loss for that sample over the time period. Bergstrom (2004) analyzed the effects of smoking on alveolar bone height and found a significant association of smoking on the height of the alveolar bone.

There is also significant research findings that suggest a correlation between smoking and the response to periodontal treatment. Jansson et al., in a 20-year long-term study of 507 individuals, found a significant effect of smoking on longitudinal bone loss (2002). Smoking is a widely accepted "high-risk" behavior for the onset and development of periodontal disease. In an extensive survey of the Australian population, Do and colleagues found that former and current smokers had significantly higher Periodontitis prevalence than those who never smoked (2008). They measured probing pocket depth (PPD) and gingival recession at various locations. This study was of particular importance because of its large sample size and the exposure of younger adults and teens and how their smoking is a significantly risky behavior for developing periodontal disease.

Many individuals smoke due to high stress levels or occupational pressure. The effects of high stress levels have been documented in health studies and clinics for many years. This stress can range from occupational to relationship-induced anxiety and current

studies are suggesting a link to Periodontitis (Ronderos and Ryder 2004). Unfortunately, full results on the link between the host response to bacterial infection, periodontal disease, and stress levels are inconclusive and not fully understood. Stress is a highly sensitive factor and can be confounded with many other additional factors. More research is needed to tease apart the influence stress has on host response to bacterial infection of the oral tissue. Research focused on stress levels and its effect on disease rates may find useful links in those studies focusing on homeless populations experiencing tremendous levels of hardship.

IV. Methods:

Almost all published case studies or articles concerning methodology can trace their roots back to Russell's 1956 article entitled, "A System of Classification and Scoring For Prevalence Surveys of Periodontal Disease" (Russell 1956). This Journal of Dental Research article describes the first method that permits quantitative comparison between individuals, populations, or within either. It is also favored by many field studies considering it requires minimal equipment. The methods used in Russell's study were applied to living tissue and not dry skulls or dry mandibles; however his methodology has been adopted and modified to fit the needs of many physical anthropologists, osteoarchaeologists, etc.

One of the earliest periodontal studies published was done by Davies, Picton, and Alexander at the U. C. H. Dental School in London. In 1969 Davies and colleagues calculated an objective method for assessing periodontal disease in deceased individuals. Their methods involved a complex index of two main measurements and eventually a score that could be assigned to the individual that was correlated with the early 1950's

Russell index (Davies 1969). The concept of a dental index is what I will incorporate most in the design of my own methods.

The methods used in this study of the Hamann-Todd collection will be similar to those used by Lavigne and Molto in their 1995 article published in the International Journal of Osteoarchaeology. Lavigne and Molto's methods are considerably based on research identifying a distance of 1-1.5mm of connective tissue that exists between the epithelial attachment of each tooth and the crest of the alveolar bone (Pattison1992, Gargiulo et al. 1961). It would be unrealistic for this study to pursue a collection of skulls the size of the Hamann-Todd collection, and because of this, a sample size of 200 will be selected randomly instead. Each individual tested will have all existing teeth recorded as well as their sex, age-at-death, and racial affinity. I have designed a Dental Inventory Form to record all measurements and notes. An existing database will provide me with the necessary demographic data such as sex, age-at-death, and racial affinity due to possible time constraints.

Before measurements of any type are recorded, a brief overview of each skull needs to be performed. This allows for notation of any loose teeth or noteworthy dentition. All existing teeth are accounted for and recorded in the appropriate section of the Dental Inventory Form. Each tooth will be measured using a periodontal probe at six different locations: Distobuccal, buccal, mesiobuccal, mesiolingual, lingual, and distolingual. Similar to Lavigne and Molto's methods, I will be subtracting 1.5mm to all measurements, as the alveolar crest is actually 1-1.5mm below the CEJ. After all six measurements are recorded, the largest of the six will be matched to an appropriate Degree of Severity using the criteria necessary and recorded as the "Individual Tooth

Score." The criteria for matching the measurements with the appropriate degree of severity will be provided at the bottom of the Dental Inventory Sheet. Once all "Individual Tooth Scores" have been calculated, a Periodontal Disease Index score (PDI) is assigned for the individual. The sum of the "Individual Tooth Scores" is divided by the number of teeth present resulting in a PDI of 0-3. This method accounts for the loss of soft tissue and attachments in dry skulls and classifies the disease into decipherable and comparable scores. Each PDI score is assigned a general description to aid in the fast identification of the severity of the disease.

A major benefit of having a PDI score is the ability to use this index in any statistical analysis for comparing sub-groups. The PDI will allow me to test each sub-group for statistically significant differences. I will specifically test for significant differences between males and females, black and white racial affinity, and various age groups. Testing of age groups will require 4 groups or age ranges to be outlined.

V. Expected Findings:

The expected findings of this study are that older age groups will express degrees of severity higher than those of younger groups. Rates of periodontal disease are expected to be higher in older age groups compared to younger ones. I also expect to find rates and/or degree of severity higher in Blacks as well as females due to the history of those groups during the time. Housing and socioeconomic conditions play an important role in health, so I expect to find data to show that individuals of black ethnicity and females, historically oppressed groups, show more signs of the disease.

VI. Schedule:

I plan to collect and record my data during the fall 2010. I will gather approval before making my final plans to visit the Cleveland Museum of Natural History. I have researched sections such as the Literature Review and the Materials and Methods, and have begun writing their respected portions of the thesis. I expect to complete all my data collection and finish the thesis in the spring of 2010.

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