Linear Enamel Hypoplasia and Dental Disease: Implications of Health and Lifestyle Behaviors of the Urban Enslaved from Two Burial Grounds in Bridgetown, Barbados

Jennifer Yamazaki

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LINEAR ENAMEL HYPOPLASIA AND DENTAL DISEASE: IMPLICATIONS OF HEALTH AND LIFESTYLE BEHAVIORS OF THE URBAN ENSLAVED FROM TWO BURIAL GROUNDS IN BRIDGETOWN, BARBADOS

by

Jennifer Yamazaki

A Thesis
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
requirements for the
Degree of Master of Arts
Department of Anthrolopology

Western Michigan University
Kalamazoo, Michigan
August 2006
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2006
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Jennifer Yamazaki
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Jennifer Yamazaki, M.A.
Western Michigan University, 2006

This project investigates aspects of diet, health, and lifestyles of enslaved Africans of Bridgetown, Barbados during the 17th-19th centuries. The dentition of 10 Pierhead and Fontabelle individuals was examined to provide evidence of stress and coping capacities experienced within the urban context of slavery. Linear enamel hypoplasias (LEH), dental disease, and cultural modifications were noted. The frequencies of the pathologies were calculated and the peak age of stress occurrence for LEH were timed and compared with other New World enslaved populations in order to determine if differences existed in overall health and lifestyle experiences between those enslaved in cities and those enslaved in rural plantations.

The results demonstrate that the Pierhead and Fontabelle individuals expressed less non-specific systemic stress in regard to malnutrition and disease compared with most other groups of New World enslaved Africans from rural plantations. This is reflected in the low frequencies of linear enamel hypoplasia. However, analysis of other dental disease frequencies including carious lesions and periodontal disease exhibit similarities to those found on other New World enslaved populations suggesting similarities in diet, oral hygiene, and lifestyle behaviors.
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CHAPTER I

INTRODUCTION

The enslavement of Africans in the New World was a unique human experience that has engaged the interests of anthropologists and historians. The enslavement of Africans in the New World created unique conditions where the entire environment has been considered an all encompassing “life stress” (Kelley and Angel 1987) which are considered by many to be unparalleled in promoting human suffering. In attempts to reconstruct the lifeways of the enslaved, modern researchers have postulated the physical suffering experienced by enslaved Africans. The institution of Afro-American and Afro-Caribbean slavery have produced voluminous literature stemming from diverse academic disciplines but few studies have attempted to examine the human skeletal remains to provide direct information regarding the hardships they faced (Corruccini et al. 1982).

The study of teeth from archaeological settings provide valuable insight into the interpretation of past populations as teeth are the most durable part of human skeletal remains and they do not remodel once they are formed (Alt et al. 1998). Recent demolition of historical buildings in Barbados have uncovered two unmarked burial grounds from the Pierhead and Fontabelle sections of Bridgetown. The burial grounds were utilized by enslaved Africans and their descendents which were used during the mid-seventeenth to early nineteenth centuries (Farmer 2004). Barbados offers unique opportunities to examine slavery within the urban context and how the life experiences of the urban enslaved may manifest in the dentition according to the
degree of demographic hardships they encountered. The goal of this study is to analyze enamel hypoplasias, dental disease, and unintentional cultural modifications in the dentition from two 17th to 19th urban burial grounds from Bridgetown, Barbados and to compare these results with osteological and historical research on contemporaneous New World rural enslaved populations.

Osteological Analysis and Bioarchaeology

The application of human osteology in anthropology can make important contributions to our understanding and reconstruction of human’s past. Whether the context is focused on forensic osteology, paleontology, or part of an archaeological record, the study of human skeletal remains can reveal a variety of pathologies from which the individual experienced (White 2000). Osteology has also been used extensively to reconstruct diet and subsistence patterns, disease and demography, and assess biological distance to estimate the degree of genetic relatedness between human populations making the transition from hunting and gathering to subsistence-based agriculture (Goodman and Armelagos 1984).

The study of human skeletal remains from archaeological settings can be a valuable source of information for interpreting lifeways of past people (Larsen 1997). Bioarchaeology offers opportunities to raise questions pertaining to human biological mechanisms and responses to inequality and exploitation (Goodman and Rose 1991). Inferences regarding diet and nutrition, disease, demography, and physical behavior and lifestyle can be drawn at the individual and population levels (Larsen 1997). Skeletal and dental tissues from archaeological settings can provide more insight into the human
condition and human social behavior and biology. Since human skeletal tissues are sensitive to the environment, they serve as a rich source of information into an individuals’ past (Alt et al. 1998).

Dental Analysis and Bioarchaeology

For anthropologists studying archaeological and fossil remains, teeth provide a valuable source of evidence in understanding the biology of past communities as they do not remodel once they are formed as well as exhibiting low susceptibility to post-depositional degradation (Kelley and Larsen 1991). The dentition is one of the most informative parts of excavated human skeletal remains (Alt et al. 1998). Dental research offers insight into evolutionary morphology, anatomy and histology, and an individuals’ physical development as well as informing us regarding cultural behaviors associated with wear, attrition, and degradation in later life (Larsen 1997). Specifically, anthropologists analyzing dental defects, plaque related diseases, and tooth modifications can reveal much about a population’s dietary intake, general health, and cultural behaviors.

Since teeth develop early in life and do not remodel once they are formed, they are protected from many external environmental influences and provide information regarding the genetic constitution and development of an individual (Alt et al. 1998). At the same time, microscopic and macroscopic observations of worn teeth disclose much information regarding food acquisition and processing (Alt et al. 1998). The anthropological study of teeth has revealed much information regarding nutrition and diet (Goodman and Rose 1991). The close relationship between dentition and diet has
revealed dental structures that incorporate certain characteristics that reflect what type of food was eaten (Goodman and Rose 1991).

A pervasive interest for bioarchaeologists is the study of dental defects and dental disease (Alt et al. 1998). The recording of enamel hypoplasias provide insight into childhood morbidity as enamel hypoplasias are symptomatic of extended periods of stress indicative of disease, malnutrition, or other factors experienced during the formative years of development (Goodman and Rose 1991). In addition, assessing different aspects of plaque related diseases including periodontal disease and carious lesions play an important role in reconstructing past human behaviors in terms of dental health and diet. Periodontal disease and carious lesions in archaeological populations often result from dietary shifts, poor oral hygiene, attrition, and nutritional deprivation (Larsen 1997). Thus, the overall study of frequencies related to enamel hypoplasias, periodontal disease, carious lesions, and unintentional tooth modifications in past human populations allows for an assessment of overall health and behavior patterns.

Teeth may also reflect an individuals’ or mothers’ nutritional status during tooth development, with the latter exhibited in deciduous dentition (Alt et al. 1998). An individuals’ nutritional status is certainly affected by dietary intake (Goodman and Rose 1991), but nutritional status also interacts with factors such as work load, and disease. Thus, nutritional status is a “reflection of an individuals’ physiological balance and it is a function of a variety of factors such as political-economic, and ecological conditions” (Goodman and Rose 1991: 256). Therefore, measuring nutritional status within contemporary and prehistoric populations can raise questions pertaining to overall quality of life.
Bioarchaeology and the Caribbean

Historians (Higman 1984, Welch 2003) note the consequences experienced due to demographic differences between past populations of urban and rural enslaved Africans in the British Caribbean. The differences were mostly exhibited through material conditions and demographic variation (Higman 1984). Osteological analysis within the urban context of slavery is limited, with comparisons between New World rural enslaved populations being slightly more abundant. Osteological and dental analysis provided by Corruccini et al. (1982), conducted on rural plantation enslaved Africans and their descendents from the Newton Plantation provided life expectancy at birth rates and compared them with other contemporaneous enslaved Africans from the United States. Corruccini et al. (1982) also utilized the available historical data from the Newton Plantation in comparison with the skeletal evidence and found higher rates of infant deaths and lower life expectancy at birth than noted in plantation journals.

In addition to the demographic findings, Corruccini et al. (1982, 1985) found high rates of enamel defects and dental disease relating to nutritional deprivation, diet, and non-specific systemic disease. Their analysis of dentition raised questions to nutritional inadequacies of those enslaved in sugar plantations as they found high frequencies of linear enamel hypoplasias associated with long bouts of nutritional crises, and non-specific systemic disease. Dental analysis conducted on other New World enslaved populations have revealed similar results. The dental analysis on the New York African Burial Ground found evidence of malnutrition and disease that occurred during childhood suggesting a population that was highly stressed (Blakey 1998).
Project Goals

The aim of this study is to document frequencies of enamel hypoplasias and the peak age of stress occurrence, periodontal disease severity and frequencies, and unintentional tooth modifications within an historic urban enslaved population from the Pierhead and Fontabelle sections of Bridgetown, Barbados, and to examine the Pierhead/Fontabelle data in light of other past New World African enslaved populations within and outside of the British Caribbean.

The remainder of the study will be organized as follows. Chapter II discusses the historical context of slavery in the British Caribbean and focuses on comparisons between the urban and rural context of slavery in Barbados. Chapter II then focuses on archaeology of New World slavery and bioarchaeology of New World slavery and dentition. Chapter III reviews dental etiology and histology, highlighting dental development necessary for understanding enamel defect and dental disease studies. Chapter IV discusses the methodologies used in obtaining and interpreting the data for the Pierhead/Fontabelle sample. Chapter V presents the results obtained from the dental analysis of the Pierhead/Fontabelle sample and comparative populations. Chapter VI discusses conclusions and suggestions for further research.
CHAPTER II

LITERATURE REVIEW

Caribbean Slavery

The majority of literature written on New World slavery, particularly slavery in the British Caribbean has been concerned with historical documents (as cited by Handler and Corruccini 1983). Although the historical literature is important in the understanding and interpretation of New World slavery, the use of archaeology and osteology can add dimension to the depth of our interpretation. There are important implications of using osteology to assess New World slavery and the health of a population. White (2000) states that although the interaction between nutrition and skeletal pathology is complex and stress markers in bone are nonspecific, patterns and trends of nutritional stress can be ascertained on the populational level. The majority of literature concerning New World slavery is from historical documents and is focused within the plantation context but historical and osteological literature of the urban context of slavery is lacking. As more enslaved burials are uncovered from urban settings, further analyses can expand our knowledge concerning the urban context of slavery and test assumptions made about life-ways, hardships, and coping capacities of enslaved Africans in the New World.

The British Caribbean

The birth of the Atlantic slave trade was a system manifested out of socio-economic exploitation (Shepherd and McD. Beckles 2000). The Atlantic slavery project evolved slowly but quickly gained momentum during the second half of the fifteenth
century, with its principal elements being established by the mid-sixteenth century (Shepherd and McD. Beckles 2000). States and entrepreneurs drove their interests aggressively and long-distance trade generated a distinct socio-cultural order (Shepherd and McD. Beckles 2000).

The "variations in the demographic experience of West Indian slaves were determined principally by their material conditions of life" (Higman 1984:1). The material conditions resulted as the product of economic forces rather than any particular religion, laws, or state systems (Higman 1984). The exploitation of enslaved Africans in the New World took certain characteristic forms which were rooted in distinct combinations of productive forces (Higman 1984). But even in the British Caribbean distinct situations occurred where a significant proportion of the enslaved population lived and worked in situations beyond the plantation boundaries (Higman 1984). Higman (1984) argues that the demographic experience between urban and rural plantation enslaved differed in consequence.

**Barbados**

The period of slavery in Barbados existed from 1627 until emancipation in 1834. In the early years of slavery, the cash economy existed on tobacco production, cotton, ginger, and indigo. These small farms were mainly cultivated by free and indentured Europeans, along with small numbers of Africans captured along the sea voyage (Handler and Lange 1978). Amerindian populations including Caribs and Arawaks, were known to inhabit the island during pre-Columbian times but the island appeared to be mostly depopulated by 1541 (Handler and Lange 1978). As the costs of indentured servants had
risen relative to the reduced costs of enslaved, a shift to predominantly African labor was utilized, and by the mid 1650s, the number of enslaved Africans doubled that of indentured servants (Welch 2003). Between 1643-1645, the Public Record Office estimated Barbados’ population to consist of 5,680 to 6,400 enslaved Africans and 18,300 to 18,600 European males (Handler and Lange 1978). At this time, the commercial growth of sugar was encouraged due to the problems associated with marketing and difficulties of growing tobacco and cotton crops (Handler and Lange 1978).

During the mid-1640s, the emphasis on sugar cultivation increased and the plantation system began to expand, thus replacing many of the small scale plantations (Handler and Lange 1978). By the end of the 17th c., Barbados had become England’s “richest colony” in the New World (Corruccini et al. 1982). From the 1700s until 1834, the plantation system was the major institution that shaped the island’s society (Handler and Lange 1978). The by-products of sugar, including molasses and rum accounted for over 95 percent of exports during the eighteenth century (Sheridan 1974), with Britain receiving most of the imports.

The introduction of sugar lead to a social and economic transformation resulting in the fast-paced development after the 1640s. Barbados’s rise in population and density increased at a rapid rate. By 1680, the first major census indicated there were 23,000 “whites” and 38,000 enslaved (Welch 2003). The majority of enslaved Africans brought to Barbados were imported during the late seventeenth to the mid-eighteenth century from the coastline of West Africa. British traders acquired them from a large section of coast that spanned from the Windward Coast, through the Gold Coast, and to the Bights
Handler and Lange (1978) estimate that from 1651 to 1807, over 352,000 enslaved Africans were brought to Barbados primarily by British traders, yet in 1834 there were only 82,000 on the island to receive liberation (Kiple 1984). Kiple (1984) suggests the implications of plantation enslaved living at the mercy of their holder's pocket. When times were flush, plantation holders discouraged reproduction as they could afford to replace the sick or infirmed enslaved instead of trying to rehabilitate them (Higman 1984). During hard periods such as hurricanes, droughts, and war, which would limit trade and ruin crops, they encouraged reproduction in order to keep the cost of purchasing new enslaved Africans to a minimum (Higman 1984). In 1817, Barbados was witnessing the natural growth of the enslaved population, resulting in only 7 percent of the enslaved being African born. It wasn’t until the late 18th c. that amelioration was adopted. It was designed to enforce plantation holders to give the enslaved specific amounts of land to raise their own provisions in an effort to decrease adult and infant mortality and increase reproduction (Kiple 1984) Thus, “the more intensive the system, the worse the enslaved were treated, and the less they reproduced, resulting in a greater need for imports” (Kiple 1984:107).

The profits of the sugar industry resulted in increasing importance of the Caribbean island economies to the English treasury (Welch 2003). Protectionist policies were designed to maximize the benefits of colonial production to the metropolitan economy and the growth of transatlantic linkages required ports or administrative centers which could facilitate the extractive process (Welch 2003). Bridgetown was established as the capital and chief port in Barbados which contained the chief administrative and
commercial institutions which governed the island. Although there were three smaller port towns, Bridgetown contained a sizable population consisting mainly of Europeans and enslaved Africans. The relationship between enslaver and enslaved hold important implications in the examination of slavery within the urban context. Although the relationship does not fit easily into a specific model, the urban enslaved experienced conditions of life different from those enslaved on plantations (Higman 1984). The weakening of bonds of dependence undermined important elements in the social control of the enslaved (as cited by Welch 2003).

Barbados: Rural Versus Urban Caribbean Slavery

The differences between rural and urban slavery in the Caribbean become apparent in the contrasts between workload, disease, and diet, between the populations. Although there is not the extent of written literature regarding urban slavery as compared with rural plantation slavery, documents written before emancipation suggest a number of differences in lifestyle and treatment of the enslaved. As plantation managers kept detailed written accounts (also known as levy books) in regard to demographics (Handler and Lange 1978), there are few records pertaining to slave demographics in an urban setting in the Caribbean (Higman 1984).

According to Higman, (1984) the main differences expressed between Caribbean rural and urban enslaved concerned occupational structure and the material conditions of enslaved life. Planters recognized plantations as small communities whereas the occupational structure of the towns represented a much more fluid network where labor was extracted by broader methods (Higman 1984). The patterns of enslaved
ownership played a significant role in the material conditions of enslaved life such as food supplies, housing, and clothing (Higman 1984). Although the quality of urban enslaved housing conditions depended on the enslaver’s wealth, it is accepted by Higman (1984) that the general quality of urban enslaved housing, especially for domestics living in the enslaver’s quarters, protected them from rain, wind, and fire. Those who were under the hiring out system may have had to find their own accommodations and were unable or unwilling to spend money for temporary homes, thus making their living conditions comparable to the worst housing found on plantations (Higman 1984).

According to Welch (2003), many of the urban enslaved in Bridgetown, Barbados were often engaged in lighter workloads compared to the enslaved from rural plantations. Most urban enslaved women worked as domestics (Higman 1984). In 1817, 50% of Bridgetown’s enslaved population were female domestics. These occupations included cooks, chambermaids, attendants, washerwomen, and housemaids. In addition, the towns also contained more sellers and transport workers than in the rural populations (Higman 1984). Thus in Bridgetown, the majority of occupations included domestics, skilled tradespeople, transport workers, laborers (manual laborers performing non-agricultural tasks), fishermen, and sellers (Higman 1984). The absence of field laborers in Bridgetown accounted for the absence of drivers. This resulted in the lack of organized gangs which were enforced under the driving system (Higman 1984).

The differences in workloads between the rural and urban enslaved in the Caribbean are exemplified by Welch (2003) and Higman (1984). Although the urban enslaved engaged in long hours of stressful, arduous labor, the fluidity of the networks made it possible for the enslaved to create a system of greater interdependence between
the enslaved and their holders (Farmer et al. 2005). The driving system of rural plantations reinforced a strict divide between enslaver and enslaved. The enslaved were under rigorous supervision and were forced into divisions of labor where even young children were classified into gangs (Higman 1984, Welch 2003). Many urban skilled tradesmen worked on an individual basis under immediate supervision of their holders and were more often employed under the self-hire system than rural plantation enslaved (Higman 1984). Female domestics may also have been able to hire themselves out and leave their holders supervision for some periods (Higman 1984). Although it is not certain if the majority of employers gave self-hired enslaved rations or accommodations, they may have been able to acquire a surplus to help accommodate them for several days (Higman 1984).

Historians state that before amelioration was enforced, planters often set out to make a quick fortune in order to return to Great Britain (Kiple 1984). This often resulted in unwillingness to give the enslaved land and/or time to grow their own provisions (Dirks 1978). In addition, planters often refused to spend their own profits on the enslaved provisions, and even those with the best intentions left the enslaved malnourished and underfed for the amount of energy spent (Higman 1984). The plantation enslaved occasionally supplemented their rations with small gardens containing small livestock, poultry, sugar cane, and food crops raised on small plots of land sometimes made available by plantation managements (Handler and Corruccini 1983). The variety was helpful in relieving the constant diet of Guinea and Indian corn but not enough to compensate for the loss of nutrients needed to be healthy (Kiple 1984). Even after amelioration, it cannot be certain that plantation managers were engaging this
policy (Higman 1984) as it was poorly enforced (Dirks 1978).

Within the urban context, urban enslaved who were unable to be hired out may have been at more of a disadvantage compared with self-hired enslaved (Higman 1984). These individuals consisted mainly of domestics and may have had reduced means to supplement their diet (Welch 2003). However, enslaved domestics may have performed a variety of tasks, requiring them to leave their holders homes, which was not an option for plantation enslaved (Higman 1984). In addition, meat was a rare commodity on plantations and the diet of urban enslaved may have been more diverse (Farmer, Smith, Watson forthcoming). 2005). Land crabs were numerous in Bridgetown’s semi-marsh environment and may have been a supplement to the lack of protein provided in their diet (Farmer, Smith, Watson forthcoming). In urban areas, the enslaved would have had more access to imported fresh fruits and vegetables as well as fish since Bridgetown was located on a port (Welch 2003). In addition, those near enough to towns could buy flesh foods with the money earned in the marketing trades and theft of food was not uncommon (Higman 1984, Welch 2003).

Sugar estates generally had the highest morbidity rates than other types of rural enslaved holdings (Higman 1984). In 1817, 0.6 percent of urban enslaved living in Bridgetown were registered as invalids, compared to 1.1 percent in plantation parishes of St. John and St. Andrew, Barbados. Morbidity levels on plantations also varied in association with seasonal cycles. The number of enslaved reported ill reached its peak at the beginning of the sugar harvest and fell during the crop time, and rose significantly about a month or two after crop-over (Higman 1984). The out-of-crop period was often referred to as the “hungry-time”, as during the crop season the enslaved were able to
supplement their diet with sugar and molasses (1984). Higman (1984) asserts that minor illnesses were associated with malnutrition and weather, while debilitating diseases depended on the rigor of labor regimes. The most common ailments, illnesses, and contagious diseases reported among Barbados plantation enslaved were body sores, cold, “consumption”, coughs, diarrhea, dysentery, dropsy, yaws, yellow fever, influenza, tooth aches, and leprosy (Handler and Lange 1978).

The relatively lighter workloads, of urban enslaved and more diverse diets may have implications in their overall dental health. As research was conducted on caloric intake of rural plantation enslaved, Dirks (1987) attempted to reconstruct the nutritional needs of agricultural laborers based on their daily caloric intake and amount of food allowances most planters offered. Dirks (1987) found their caloric intake extremely insufficient for the amount of energy expended in regard to their arduous labor but containing enough protein as demonstrated in the taller heights of many first generation creoles.

Archaeology of New World Slavery

The interpretation of New World slavery in the British Caribbean has drastically changed throughout the past (Orser and Funari 2001). Prior to a small number of archaeologists and ethnohistorians who emerged in the late 1960’s and early 1970’s, most reconstruction of the life of enslaved populations was acquired from a wide variety of published sources provided by European writers (Handler and Lange 1979). These documents emerged from traveler’s descriptions of island life, journals and private letters, and official letters and reports (Handler and Lange 1979, Higman 1984). In
addition to the fact that there are few documents written by the enslaved themselves, the European documents are considered to be highly prejudicial (Handler and Lange 1979). Handler and Lange (1979) challenged the difficulties in interpreting historical material in terms of overcoming class, racial and ethnocentric biases in these sources. These previously published sources were often fragmentary and highly variable in quality and completeness (Handler and Lange 1979).

In response to overcoming some of these biases produced in the literature, a number of archaeologists began to examine aspects of slavery through material culture (Handler and Lange 1978). The archaeologists of the 1960’s and 1970’s who examined characteristics of slavery were primarily concerned with cultural identification (as cited by Orser and Funari 2001). They performed the required tasks of determining the nature of enslaved material culture and had few expectations about their findings at abandoned slave settlements since these types of settlements had never been excavated before in a thorough manner (Orser and Funari 2001). According to Orser and Funari (2001), these preliminary studies forced archaeologists to refute the commonly held belief that the enslaved peoples in the New World did not have an active, expressive material culture.

Research conducted by Handler and Lange (1978) was designed to yield information within a rural plantation context in terms of domestic life, and mortuary behavior, which was scarce from the literature. Their research on the Newton Plantation was the largest archaeological sample of human skeletal remains of enslaved individuals in the British West Indies (Handler and Corrucinni 1983). Handler and Lange (1978) wanted to demonstrate how archaeological materials could give an unbiased assessment of how Africans and their descendents would directly communicate aspects of their social
and cultural life through their material cultural and human remains in the New World (Handler and Lange 1978). The ultimate goal of Handler and Lange’s (1978) research was to compare the archaeological research with the available historical information to shed more light on plantation slavery in Barbados.

Dentition and Bioarchaeology

The potential role of bioarchaeology for understanding humanity’s past has only recently become realized (Larsen 1997). Currently, a growing number of archaeologists are incorporating osteological analyses into their research designs (Blakey et al. 1994, Corruccini et al. 1985, Handler and Corrucinni 1983, Kelley and Angel 1987, Rathbun 1987), as larger numbers of physical anthropologists move beyond simple descriptive analyses of human skeletal remains (Larsen 1997). In the past three decades, there have been a growing number of anthropologists reconstructing the daily life past populations through osteological observations. These researchers have utilized the available human skeletal material for analysis regarding agricultural intensification (Blakey and Armelagos 1985, Goodman et al. 1980, Lukacs 1992,) but only more recently have Afro-American human skeletal remains from historic sites become available for analysis (Rathbun 1987).

Osteological analyses can allow for inferences to be made about the health and coping capacities of past populations. Analysis of a rural enslaved South Carolina population conducted by Rathbun (1987), have found skeletal indicators of health and disease including dental pathologies, anemia as evidenced by cribra orbitalia, infection, growth interruption evidenced by Harris lines and linear enamel hypoplasia, and trace
elements in bone related to diet. Rathbun (1987) also adds that osteological research of enslaved populations provides additional evidence as them being subject to arduous labor and exposure to infectious disease (Rathbun 1987). Analysis on the same population revealed skeletal indications resulting from strenuous physical labor and stress such as degenerative changes in shoulders, hips, and lower vertebrae (Rathbun 1987). Similar research conducted by Kelley and Angel (1987) comparing different United States plantation enslaved found high levels of anemia indicative of low protein levels, and parietal depression and ulna fractures indicative of possible violence-related trauma.

The osteological analysis conducted on rural enslaved populations from Barbados and elsewhere in the Caribbean and the New World has provided additional insight into cultural and behavioral systems manifested in plantation slavery as a result of European colonialism (Corruccini et al. 1982, 1985). Previous research conducted on New World enslaved populations have been concerned primarily with written literature alone, partly due to the rarity of human skeletal material (Rathbun 1987). In addition, the qualitative or literary historical sources were often considered ethnocentric and variable in quality as they took the form of traveler’s accounts, private letters and journals, official letters, and reports written by Europeans (Handler and Lange 1978). Information regarding health and lifestyles of urban enslaved populations has been even more limited as information from historical documents is scarce (Higman 1984) and few urban burials have been recovered.

Shortly after the initial archaeological investigation of the Newton Plantation, Handler and Corrucinni (1983) corroborated to conduct osteological analysis on the enslaved burial ground as Corrucinni was a physical anthropologist who focused in dental
morphology. Their osteological analysis from an archaeological setting offered a fundamentally new way to approach archaeology in terms of raising new questions pertaining to various problems associated with the life of the enslaved. Questions concerning quality of life associated with slavery could be more thoroughly addressed in terms of disease, workload, and malnutrition as the consequences of these can manifest in the human skeleton.

The research conducted by Handler and Corrucinni (1983) focused on dental defects and dental disease of enslaved Africans as the dentition was the most abundant skeletal tissue recovered, and is a valuable indicator of cultural practices and physiological stress. The primary focus of the dental analysis consisted of the frequency and chronology of enamel hypoplasias. The focus on enamel hypoplasias and periodontal disease were perhaps the best indicators of malnutrition, and disease (Handler and Corruccini 1983) which historians note were commonly experienced by many enslaved Africans (Shepherd and McD. Beckels 2000). This analysis combined with the historical literature confirmed that the majority of enslaved individuals from the Newton Plantation suffered from severe physiological disturbances during their growth.

A number of other anthropologists were also incorporating a biological and historical approach in regard to their analysis instead of relying primarily on the historical data. Researchers including Rathbun (1987), and Blakey (1994) looked at the sociocultural factors with the biological factors that have influenced the health, nutrition, mortality, and morbidity of enslaved African Americans within an historical context. The research conducted by Handler and Corrucinni (1978) began to raise new questions on the life of enslaved plantation workers in the New World within a sociocultural
context as it sometimes complemented and refuted the historical demographic literature.

Archaeologists focusing on New World slavery in the United States (Blakey et al. 1994, Mack and Blakey 2004, Kelley and Angel 1987, Owsley et al. 1987) addressed many of the same issues that Handler and Corrucinni (1983) had addressed. These studies focused on enamel defect frequencies and many aspects of dental disease as well. This information was used to infer overall health and lifestyle in association with disease, diet, and malnutrition. With the excavation of the New York African Burial Ground (Blakey 2001), comparisons between slavery in the British West Indies and the United States were addressed as well as comparisons between enslaved populations in the United States. In addition to making inferences of childhood stress, Blakey et al. (1994) used methods proposed by Massler and colleagues (1941) and Goodman et al. (1980) to record the chronology of enamel hypoplasias to infer aspects of timing and weaning practices of the mothers to their children. The studies in bioarchaeology with a biohistorical approach of African enslaved populations have continued to grow as more burials are uncovered, thus allowing for comparisons within the rural-urban context.

Lanphear (1990) also addressed the frequency and changes in LEH from the 19th c. Monroe County Poorhouse Cemetery, Rochester, NY. As European and American demographers associated increases in mortality during industrialization from agricultural subsistence. The Monroe County Poorhouse contained individuals who could no longer care for themselves, the mentally and physically disabled, the aged, and chronically ill. They found that of the number of mandibular and maxillary canines and incisors present, 70 to 73% contained at least one enamel hypoplasia with peak age at stress for the incisors occurring between 2.5 to 3 years and for the canines between 3.5 and 4 years.
When comparing these differences in peak age at stress with other subsistence groups, Lanphear (1990) found that age at stress occurs earlier in the 19th c. sample. For example, prehistoric and historic agricultural populations display a peak age at stress between 2 and 6 years (Goodman et al. 1984). Lanphear (1990) associated the stress events to indicate the time of weaning.

Enamel Hypoplasia Research

Sarnat and Schour (1941) cite the earliest references to enamel defects date back to 1743, when Bunon described that “erosion” of teeth occurs as a result from rickets, measles, and scurvy, and in 1785, Sanchez described dental alterations in correlation with syphilis. Research conducted in the late 1900s by Hutchinson correlated certain dental alterations to be characteristic of hereditary syphilis, thus confirming Sanchez’s earlier claims of relating dental defects to specific diseases (as cited by Goodman and Rose 1990). Research conducted by Parrot considered the possibility of the combination of syphilis and rickets as principal agents causing enamel defects (Sarnat and Schour 1941). Simultaneously, researchers including Berten, Black, and Calteux focused on nutrition as a causative agent and made broader assumptions stating that any severe disturbance that interfered with nutrition would result in defects of the tooth (Sarnat and Schour 1941).

The term “erosion” was first used by the French (as cited by Sarnat and Schour 1941) and other terms such as enamel aplasia, premature caries, atrophied teeth, and notched incisors were used in conjunction, and in place of the term enamel hypoplasias (Goodman and Rose 1990). Zsigmondy advocated for the term “enamel hypoplasia” to
describe dental defects in 1893 and while others disregarded the term, it is the most generally accepted term today (Sarnat and Schour 1941). According to Goodman and Rose (1990), the early studies prior to the 1930s regarding developmental defects tended to focus on two issues: the relationship between enamel quality and subsequent susceptibility to dental caries, and, that the quality of enamel reflects underlying physiological disturbances due to periods of malnutrition and disease. In 1906, Black compared histological observations with surface observations to suggest that surface defects result from systemic and localized insults (Goodman and Rose 1990).

While these studies did not settle the debate supporting any relationship between carious lesions and enamel quality, they did set the stage for histological methods to be utilized on archaeological teeth. The early studies also demonstrated that enamel surface and microstructural defects of the enamel and dentine were associated with disturbances to the developing enamel as a result of some type of event that occurred in an individual’s life (Goodman and Rose 1990). Sheldon et al. (1945) state the importance of these early studies as they gave rise to the concept that sickness in early life had an adverse affect on the structure of the tooth. The relationship between the location of the surface defect and age of occurrence was not systematically examined until the 1930s.

Enamel hypoplasia studies flourished in the succeeding decades and the advancement in the study of enamel defects were largely in part due to the research of Sarnat and Schour (1941, 1942), and Schour and collegeagues from 1932 to 1945 (Goodman and Rose 1990). Sarnat and Schour (1941, 1942) produced a series of literature reviews on enamel formation and tooth development to expound on the etiology of “chronological enamel aplasia” or enamel hypoplasias. As indicated by Sarnat and
Schour (1941), much of the previous research on enamel hypoplasias lead researchers to believe they formed from a specific etiology. In contrast, they looked for correlations with specific disease histories in the patients studied, but found no specific etiology. In fact, all of the individuals with hypoplasias studied had no previous exanthematous diseases (scarlet fever, measles, chickenpox, and smallpox) which were once thought to be the principal etiological factors causing enamel hypoplasias. They concluded that the possible etiologic factors resulting in enamel hypoplasia included but were not limited to rickets, hypoparathyroidism, and fluorosis (Sarnat and Schour 1941).

Sarnat and Schour (1941, 1942) also produced chronological charts to delineate tooth crown formation times as well as assess the attributes of chronologic enamel hypoplasia to disease periods during growth. They asserted that knowledge of the chronology of tooth development which was assessed on a ‘roentgenographic’ (unerupted and erupted teeth) basis, along with clinical and histological bases was necessary to establish the time that the tooth was developing during a disease period. In their series of literature, they provided summaries of tooth development, histological analysis, chronology of tooth development, case studies based off of patients with known disease histories, and possible etiologic factors involved in enamel hypoplasia development.

Although the etiology of enamel hypoplasias can be multicausal, the majority of studies since Sarnat and Schour’s (1941) research indicate they are a sensitive reflection of systemic physiological stress produced by a variety of factors including malnutrition, systemic disease (including childhood fevers), fluorosis1, psychologically stressful

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1 Although fluorosis is a well-known factor causing enamel hypoplasias in humans (Hillson 1986), fluorine ions which are present in most drinking water usually occur in very low concentrations in which enamel defects rarely result from fluorosis (Hillson 1986).
situations (Guatelli-Steinberg 2001), and in rare instances, hereditary factors (Hillson 1986, Lovell and Whyte 1999). Schour and Massler (Goodman and Rose 1990) produced experiments focused on nutritional analysis in conjunction with oral defects. These studies aimed to correlate changes in enamel structure due to deficiency in calcium, Vitamins A, C, and D, and fluorine levels. The importance of Schour and Massler's research was that they found enamel to be uninfluenced by alterations in nutrition after the enamel had already been formed.

Many of the early researchers analyzing hypoplasia etiologies were concerned with using laboratory animals to further knowledge of enamel defects in humans (Kreshover 1944). Studies experimenting with induced stress in laboratory animals demonstrated the direct link between enamel deficiency and stress that produced histologic and surface defects from disrupted enamel formation (Kreshover 1944). The researchers of this time had not made distinctions between the human and non-human teeth they were making comparisons with. Kreshover (1945) looked at ameloblastic injury in rats and guinea pigs infected with injections of tubercle bacilli in order to demonstrate the earliest, second, and terminal stages of hypoplasias. His research was set up to construct the changes in enamel formation in association with the onset of disease.

By the 1960s a number of different researchers had published a vast amount of literature regarding the variety of etiologies associated with enamel hypoplasias. Extensive series of histological studies examined relationships between the different stressors such as tuberculosis, alloxan diabetes, induced fever, hormonal imbalances, nutritional deficiencies, and hereditary factors associated with abnormal amelogenesis,
microdefects, and surface defects. Although this information was being established prior to Kreshover's 1960 publication (as cited by Goodman and Rose 1990), his reports on experimental evidence showing the non-specific nature of enamel defects brought on by stress influenced the research for the next generation of researchers.

Goodman and Rose (1990) compile the history of enamel defects research back to the first observations of tooth defects or "erosion" in the late 1800s. They state that although the stage had been set by the 1960s for the general methods employed in researching enamel hypoplasias, two assumptions that may have been derived from Sarnat and Schour's work (1941, 1942) hindered the discovery of disruption patterns and prevented studies in population variations of stress. For example, the first assumption was that teeth were universally susceptible to enamel defects. This assumption also assumed that all developing teeth were equally exposed and responsive to stress, thus the quality of enamel is the same across all tooth classes.

The second assumption was that the chronological pattern of enamel defects was due to a "universal constitutional change in susceptibility to disruption" (Goodman and Rose 1990: 79). According to Goodman and Rose (1990), this second assumption prevented researchers from studying the variations between different populations in regard to differences in chronologies of stress. Not taking differences in population variances was also reflected in Kreshover's (1944) research which focused on using laboratory animals to infer the etiology of enamel defects of humans.

The advances in dental histology of the 1960s through the late 1970s allowed more understanding of embryology and histology of enamel (Goodman and Rose 1990). Connected with this time period was an increase in archaeological and human skeletal
populations with a focus on the relationship between nutritional and disease stressors in relation to enamel defects. In addition, studies were now being conducted on extant populations in underdeveloped nations. Important research to follow included public health surveys constructed to correlate poor living conditions and enamel defect prevalence in contemporary human populations from underdeveloped nations (Sweeney and Guzman 1966, Sweeney 1969).

Comparisons between children of three Guatemalan villages exemplified large differences of enamel hypoplasia prevalence between populations and lead Sweeney (1969) and Guzman (1966) to draw on correlations between enamel hypoplasia formation with nutritional status and infectious disease. As they noted vast differences in enamel hypoplasia frequencies between villages, they also explored multiple causes of defect etiology including low serum vitamin A levels, birth weight, gestation length, types of infectious diseases, birth order and family size, while emphasizing social, or genetic predispositions. Although they did not find significant correlations between hypoplastic lines and infectious disease at time of birth, the significance of their research highlighted the correlations they made between infectious disease and nutritional status with linear enamel hypoplasia.

While systematic studies focusing on enamel defects between populations in developing countries continued, paleoepidemiological research was also developing. With the development of bioarchaeology, a growing number of anthropologists were researching histological changes in teeth from archaeological populations. Swardstedt (1966) is attributed as being the first to conduct systematic studies of linear enamel hypoplasias on archaeological populations (as cited by Goodman and Rose 1990). His
research focused on a medieval population from the province of Westerhus, mid-Sweden. Comparisons were now being made between males and females, socioeconomic classes, and the chronologic nature of linear enamel hypoplasias to infer annual cycles of stress. The model of “better-off” versus “less well-off” individuals and increased stress was strengthened as he found significant differences in prevalence of linear enamel hypoplasia in individuals from lower economic classes (Swardstedt 1966).

One of the biggest innovations attributed to Swardstedt was the ability of using the tooth crown formation charts provided by Massler and co-workers (1941) and exhibiting the age of the occurrence of enamel defect based on measurements from the cement enamel junction (CEJ) to the defect. This method provided a relative age of occurrence at the time of the enamel hypoplasia formation. Swardstedt (1966) is also recognized for noticing that defects should be matched across teeth that were developing concurrently at the time of the disturbance (Goodman and Rose 1990). Other researchers (Hillson 1979) were employing similar methods and finding similarities in peak age of occurrence in Egyptians and Nubians from 2 to 4 years of age. Similar research conducted by Scuilli (1977) compared deciduous to permanent teeth of agriculturalists of prehistoric Ohio Valley Native Americans to decipher if the prenatal environment was “safer” than the postnatal environment.

The paleoepidemiological studies of this period established general methods to estimate the developmental age of the individual at the time of the defect formation. Research conducted by El-Najjar et al. (1978) analyzed populations from the Hammon-Todd skeletal series in an attempt to compare enamel hypoplasia frequencies from an early 20th c. urban population with contemporary groups of “white” and “black”
children from Cleveland. Their research suggested that the frequencies of enamel hypoplasias were significantly higher in the early 20th c. populations compared with the modern populations from Cleveland. Although they noted no specific etiology could be found, they proposed that increased nutritional conditions played a significant role in the decrease in frequencies over time. When they compared prevalence of enamel hypoplasias between tooth classes, they noticed significant differences in prevalence between teeth, with anterior teeth exhibiting higher frequencies. These results suggested that other factors were involved other than time of development.

Few studies in the late 1970s and early 1980s continued to correlate or focus on enamel quality and carious lesions (Goodman and Rose 1990). As histological methods vastly improved, researchers began to further understand enamel etiology and thus, separated etiological research of carious lesions from enamel hypoplasias. In addition, researchers were realizing other mammalian teeth (rodents) were not good indicators of human enamel etiology and experiments in enamel defects with animals slowed. Clinical studies continued to flourish and focus on dental fluorosis and the relation to enamel defects (as cited by Goodman and Rose 1990). Clinical studies also focused on the relationship between neonatal complications and abnormalities such as hearing impairments, to defects of deciduous teeth.

Research in the 1980s continued looking at enamel surface defects in human skeletal populations. Studies conducted by Cohen and Armelagos (1984) traced the differences in frequencies of enamel hypoplasias between hunter-gatherer groups and agricultural groups. They documented the health costs of subsistence shifts such as nutritional deprivation and increased diseases, and provided evidence of increased
enamel hypoplasia frequencies occurring in early agriculturalist groups. Other researchers conducting similar studies found similar patterns in enamel hypoplasia expression.

Goodman et al. (1984) focused on morbidity and mortality measures of the Native Americans who lived at the Dickson Mounds site, Illinois during the preagricultural, transitional, and agricultural phases. These studies in paleoepidemiology used stature and other anthropometric measures such as porotic hyperostosis, and enamel defects as general indicators of nutritional stress during infancy and childhood.

One of the most notable studies conducted on enamel hypoplasias was conducted on enslaved Africans from a rural sugar plantation in Barbados (Handler and Corruccini 1983). Their research contained the largest number of enslaved Africans in the New World and has allowed for comparisons between other New World enslaved populations from rural and urban contexts. Handler and Corruccini (1983) reported 98% of the enslaved Africans from the Newton plantation to express severe linear enamel hypoplasias. They attributed the high frequency and severity of enamel hypoplasias resulting from weaning stress, periods of near starvation, a diet lacking in vital nutrients in the pre and immediate post-natal environment (Handler and Corruccini 1983). Further research also suggested high rates of congenital syphilis which contributed to morbidity, infant mortality, and infertility in this population, thus demonstrating high frequencies of enamel hypoplasias (Jacobi et al. 1992).

Anthropologists have utilized and expounded upon these types of studies throughout the 1980’s and continue today (Alt et al. 1998). Anthropological studies of dental defects and dental disease primarily focus on contemporary and prehistoric humans, and non-human primate dentition (Hillson 1996). The various methods
employed for recording and analyzing dental defects are not always agreed upon by various anthropologists (as cited by Hillson 1996). Although this makes it difficult to summarize results of the studies, there are a number of recurring themes for interpreting increases in dental defects in archaeological samples. Examples are the transition from hunter-gatherer subsistence to agriculture (limiting nutrient intake), a modal age of growth disruption associated with weaning, and the arrival of Europeans to the New World (e.g., introduction of new pathogens to indigenous populations) (Hillson 1996).

Caries Research

Although caries have been found in ancient human populations, the frequency has not dramatically increased until the adoption of agriculture (Hillson 2000). Early studies on caries that emerged prior to the 1930’s conducted on ancient and prehistoric people attempted to determine why modern people exhibited higher rates of carious lesions while most ancient people did not (Goodman and Rose 1990). These studies focused on the relationship between enamel quality and caries susceptibility and utilized teeth from archaeological settings to section for histological observations (Goodman and Rose 1990). As Goodman and Rose (1990) point out though, clinical studies on extant populations were not being conducted at this time. Hypotheses that stated the teeth of ancient people were superior to modern people due to the lack of carious lesions were challenged by Berten (as cited by Sheldon et al. 1945) as he provided evidence showing that disturbances in metabolism are reflected in the microstructure of a tooth.

As this set the stage for furthering enamel hypoplasia studies, research was still being conducted on ancient teeth in regard to the superior enamel quality and lack of
carious lesions compared with modern populations. Sectioning of archaeological teeth by Sognnaes (Goodman and Rose 1990) tried to determine the postmortem changes in the enamel and dentin with burial and fossilization. The enamel however, remained unchanged in even the oldest teeth and helped researchers to determine that enamel of ancient people was not superior to enamel of modern people despite the high frequencies of carious lesions in the modern humans.

More importantly, Goodman and Rose (1990) state that Schuman and Sognnaes concluded defective enamel is not necessarily the result of having inferior enamel but rather, defects were a result of nutritional and other metabolic disturbances. An increase in histological defects in Aluet teeth were attributed to the decline in the quality of diet associated with Western acculturation (Moores 1957). Caries rates were now being associated with diets high in refined carbohydrates (Goodman and Rose 1990). These studies eventually established the basis for using microstructure of enamel to the adaptive success of prehistoric people and enabled enamel histology to be employed in anthropological research.

Today, there is an abundance of published data on dental caries in the archaeological record and in living populations because most lesions are macroscopically observable (Larsen 1997). Although caries etiology is not completely understood (Larsen 1997) several factors are now known to contribute to the development of caries. These include: “1) the exposure of tooth surfaces to the oral environment, 2) the presence of aggregates of complex indigenous oral bacterial flora, salivary glycoproteins, and inorganic salts adhering to the tooth surfaces; and 3) diet” (Larsen 1997:65). During the last century, sugar consumption has been attributed as the major cause of caries rates
Hillson 2000). This has been inversely demonstrated through decreased caries rates due to sugar rationing in Japan, Norway, and the Island of Jersey during World War II (Hillson 2000).

Caries rates are often reported in the archaeological literature and reflect the nature of the diet (Hillson 2000). The prevalence of caries rates in samples of New World enslaved populations have been documented within the rural and urban context (Handler and Corruccini 1983, Owsley et al. 1987, and Rathbun 1987). Owsley et al. (1987) report moderately high frequencies of caries from an urban enslaved population from New Orleans and attribute the high frequencies to the overwhelming consumption of corn meal, fine flour, and molasses. Handler and Corruccini (1983) report high frequencies of interproximal caries in enslaved Africans from a Barbados sugar plantation, but relatively low frequencies of occlusal surface caries comparable to non-industrialized groups. Thus, asserting that the diet was largely carbohydrate (which corroborated with the historical data) but caries rates were low compared with gum and root pathologies (Handler and Corruccini 1983). These studies have been useful in complimenting the available historical and demographic data, and will allow comparisons of diet, oral hygiene habits, and attrition to be made between different New World enslaved populations.

Periodontal Disease Research

Antemortem loss of teeth and periodontal disease has been an interest of study for anthropologists, dentists, and public health investigators (Costa 1980). The prevalence and severity of periodontal disease can reveal much about human health, nutritional status, and lifestyle behaviors in the archaeological record. Periodontal disease and dental
disease of various types has long been considered to plague Homo sapiens since the earliest times (Molnar and Molnar 1985).

The frequency of occurrence has varied widely throughout prehistory but there has been a general increase in frequency in more recent periods (Molnar and Molnar 1985). Numerous studies have exposed rates of tooth decay among ancient and modern human populations but few studies address the rates of periodontal disease. Among these studies however, there is a noted trend toward an overall increase in dental disease including periodontal disease (Molnar and Molnar 1985). The factors associated with periodontal disease etiology in oral health surveys of modern human populations include changes in lifestyle, particularly diet and oral hygiene (Molnar and Molnar 1985). Clark (1997) notes that recent populations undergoing shifts from traditional to Western diets experience a higher rate of periodontal disease and carious lesions, and archaeological populations demonstrate high levels of dental disease with consumption of plant carbohydrates and processed foods.

Some researchers (Costa 1983, Clarke et al. 1986) challenge the conventional concept of the pathogenesis of periodontal disease (Clark et al. 1986). Clark et al. (1986) suggests that periodontal disease is overestimated in the archaeological record as other factors can be responsible for tooth loss and alveolar bone resorption. Clark et al. (1986) made attempts to discriminate between alveolar modifications resulting from bacterially related crestal bone resorption (periodontal disease) and anatomical, developmental, physiological, and other pathological defects that may be responsible for the increased distance between the cemento-enamel junction (CEJ) and the alveolar crest (AC).

Previous research of prehistoric Eskimo skeletal remains conducted by Costa
(1982) considered the relationship between diet and periodontal disease in societies consuming a non-Western diet (high in protein/fat and low in carbohydrates). While it is generally agreed diet is a major contributor to periodontal disease, other factors such as heavy mastication and poor oral hygiene attribute to periodontal disease and tooth loss (Larsen 1997). For example, Costa (1982) determined that high rates of periodontal disease from Ipiutak and Tigara populations was caused by a diet unlike modern Western diets and the etiology may be more closely associated with high rates of wear and attrition.

Dental disease studies on enslaved populations offer insight into possible periodontal disease etiology and are useful for reconstructing dental health in past populations. The majority of research conducted on New World enslaved populations reveal high rates and severe stages of periodontitis (fourth stage of periodontal disease) and attribute causes such as calculus build up, lack of systemic oral hygiene, and factors associated with diet and malnutrition (Corruccini et al. 1987). Previous research conducted by Handler and Corruccini (1983) on enslaved Africans from a rural sugar plantation in Barbados showed high rates of severe periodontitis where nearly all of the adequately preserved individuals expressed the disease. Since they found few peripical abscesses and little evidence of trauma, they asserted that periodontal disease was more closely associated with lack of systemic oral hygiene and natural stimulating and cleaning elements such as fiber found in the diet. In addition, Corruccini et al. (1987) relate periodontal disease in the Barbados sample to possible factors including iron and protein deficiencies, malnutrition, and vitamin C deficiency.

Other research conducted on an urban enslaved population from New Orleans
(Owsley et al. 1987) found 31.6% of all adult tooth sockets affected by active periodontal or periapical abscesses at the time of death, or antemortem tooth loss and corresponding bone resorption. Although they did not make attempts to assess the etiology of periodontitis among the population, they suggested that dental caries and antemortem tooth loss owing to periodontal disease abscess formation were common health problems, and that a diet high in refined carbohydrates and sugars was consumed (Owsley et al. 1987). Although dental wear and attrition should not be discounted in assessing periodontal disease in ancient populations, diets of past New World enslaved populations were relatively similar, consisting of high amounts of plant carbohydrates and refined sugars (Handler and Corruccini 1983, Rathbun 1987, Owsley et al. 1987). The high rates in dental defects, dental pathologies, and growth interruption suggest diets high in these types of foods (Rathbun 1987).

Premortem Dental Alterations

Different cultural behaviors may alter dentitions in a variety of ways (Buikstra and Ubelaker 1994). These may include deliberate modifications such as filing and inlays, or unintentional modifications resulting from habitual practices such as using the mouth as a ‘third hand’ or pipe smoking. A classic example is the resulting circular notch created by gripping clay pipe stems (Hillson 1996). Although the preemancipation historical record rarely mentions the use of pipes by the enslaved, several sources indicate they valued and smoked tobacco (Handler and Lange 1978). Most of the tobacco was imported and distributed by plantation managements as a reward or incentive for good behavior of the enslaved (Handler and Corruccini 1983). Barbados imported pipes from
Europe during the slave period in exceedingly high amounts and they appear often in the archaeological record as grave goods interred with slave burials (Handler and Lange 1978, Handler and Corruccini 1983).

Because premortem dental alterations, enamel defects, and dental disease can be easily observed in dental samples with the latter two representative of nutritional stress and systemic disease, they are often used as health indicators of past and present human populations (Guatelli-Steinberg 2001). Thus, many archaeologists and physical anthropologists have been utilizing enamel hypoplasia studies and aspects of dental disease such as carious lesions, and periodontal disease from archaeological samples in order to compare past human populations in terms of stress, deprivation, and changes in diet (Larsen 1997). The study of dentitions from paleontological and archaeological settings adds dimension to the understanding of the history of human stress and the complexity of how it links with the environment, culture, and biology (Larsen 1997).

Site Descriptions

Pierhead Burial Ground

In July, 1996 an unmarked African burial ground was discovered in Bridgetown, Barbados by construction workers while renovating the Dacosta Mannings department store. The construction workers notified the Barbados Museum and Historical Society of the burial ground and altered their work schedules in order for archaeologists to conduct an emergency excavation (Crain et al. 2004). Dr. Karl Watson from the University of West Indies, Dr. Frederick Smith of Western Michigan University, and a team of archaeology students from the University of West Indies investigated and
recovered information about the human skeletal remains and artifacts that lay around the construction site (Crain et al. 2004). The grave goods, associated artifacts and human skeletal remains that were able to be recovered were then taken and stored at the archaeology laboratory at the University of West Indies, Cave Hill Campus (Crain et al. 2004).

The grave goods, associated artifacts, and osteological analysis conducted by Christopher Crain (Crain et al. 2004) indicate that the Pierhead burial ground was of enslaved Africans and their descendents. Ceramic fragments found at the site including Staffordshire slipware, delftware, and Westerwald stoneware suggest the cemetery was utilized from the late seventeenth century to the mid-eighteenth century (Crain et al. 2004). In addition, the bowl configuration and shape of an English tobacco pipe made of white kaolin clay add further evidence of the Pierhead burial ground being used in the late seventeenth to mid-eighteenth century though it is uncertain how long the burial ground was in use (Crain et al. 2004).

Fontabelle Burial Ground

In the year 2000, another unmarked cemetery consisting of human skeletal remains and grave goods was located in the Fontabelle section of Bridgetown, Barbados during the construction of the Barbados Small Business Development Center (Farmer 2004). Emergency excavation was carried out under the supervision of Kevin Farmer. The associated grave goods and a maker’s mark found on the bowl of a tobacco pipe within a burial indicate that Fontabelle is of the same time period as the Pierhead cemetery. Preliminary osteological analysis, historical documents, and associated grave
goods exhibit characteristics of an enslaved Afro-Caribbean burial ground from the Fontabelle section of Bridgetown, Barbados (Farmer, Smith, and Watson forthcoming).

A number of factors were considered to determine that the Pierhead and Fontabelle burials contained enslaved Africans of Barbados. Historical documents written by Robert Poole, a visiting English physician in 1748 record the events of an infant burial for enslaved inhabitants of the Fontabelle section of Bridgetown (Farmer, Smith, and Watson forthcoming). Poole described the burial ground as facing the sea with sandy beach before it (Farmer 2004). The site excavation of Fontabelle uncovered yellow-beach sand and is consistent with the description of Poole’s account and is also similar to the Pierhead burial ground (Farmer 2004). Farmer (2004) also note the marginal location of the two burial grounds which were located close to the sea and just beyond the beach. The Pierhead and Fontabelle section of Bridgetown were located in marginal coastal lands which were mostly undeveloped until the mid-eighteenth century (Farmer 2004).

By 1654, the West End was the location of the main church, St. Michael. Crain et al. (2004) considered the possibility of the burial grounds belonging to Christian Europeans residents but they were buried nearby in St. Michael’s churchyard. Crain et al. (2004) also considered the possibility of an Amerindian burial ground but ruled it out based on the presence of historic period materials buried with some of the individuals. Crain et al. (2004) considered the possibility of a Quaker burial but they were buried in a burial adjacent to the synagogue. The possibility of the burial grounds belonging to freedmen and freedwomen was also ruled out as they made up a small percent of the population in the second half of the eighteenth century and were often buried in
churchyard grounds as freedmen and freedwomen were often baptized (Crain et al. 2004).

The internment practices and body orientation of both burial grounds were similar to those found at the Newton Plantation (Crain et al. 2004, Farmer, Smith, and Watson forthcoming). The excavation of the Newton Plantation, a rural sugar estate in Barbados revealed a lack of headstones along with west-headed and east-headed burials (Handler and Lange 1978). Handler and Lange (1978) stated the possibility of east-headed burials showing an appreciation for African-oriented beliefs and a return to Africa in the afterlife. In addition, they state that the earlier burials from the Newton Plantation demonstrated more east-headed burials when the majority of enslaved were African born, while later burials were more west-headed oriented and probably indicated a higher number of Creoles adopting Western burial practices (Handler and Lange 1978). The Pierhead burial ground site only exhibited two east-headed burials while all of the Fontabelle burials exhibited west-headed burials (Crain et al. 2004).

The biological profile to determine the ancestry of the human skeletal remains from both burial grounds was determined by Christopher Crain (Crain et al. 2004). Crain (Crain et al. 2004) used methods proposed by Bass (as cited by Crain et al. 2004). The primary traits used for determining ancestry included “rectangular eye orbits, receding zygomatics, rectangular dental arcade, spatulate incisors, simple vault sutures, s-shaped zygomaxilary sutures, a post-bregmatic depression, wide nasal opening, nasal guttering, notable facial prognathism, and a round auditory meatus” (Crain et al. 2004:8). Crain et al. (2004) determined that when combined, these values and traits represented those of African ancestry.
Burial Ground Location

Barbados is the most eastern of the Caribbean islands. Bridgetown, Barbados is located on the south-west portion of the island. During the seventeenth through nineteenth centuries, the Fontabelle and Pierhead areas of Bridgetown were located in marginal coastal areas on the periphery of Bridgetown. The Pierhead section of Bridgetown marked the southern boundary of Bridgetown, about 40 meters south of the Constitution River and about 60 meters southwest of the Swing Bridge (Crain et al. 2004). The Fontabelle burial ground was located on an undeveloped stretch of white-sand beachhead on the western edge of Bridgetown (Farmer 2004), just three miles south of the Pierhead site (Farmer 2004). The Pierhead section was also undeveloped shoreline surrounded by a marshy swamp (Farmer, Smith, and Watson forthcoming).
CHAPTER III

ENAMEL ETIOLOGY AND HISTOLOGY

Teeth hold an important place in anthropological studies of past human populations (Hillson 1996). For anthropologists studying archaeological and fossil remains, the teeth provide valuable insight into the understanding and interpretation of the biology of past populations (Hillson 1996). Teeth are the most resistant parts of archaeological and fossil remains and their durability allow their preservation when other parts of human skeletal remains often do not survive (Hillson 1979). In order to discriminate between, and interpret dental diseases and dental defects of past populations, it is essential to be able to recognize the role of physiological variables that influence teeth. The study of dental defects and dental diseases of New World enslaved populations can provide more insight into the human condition and human social behavior and biology.

Tooth Composition and Anatomy

A tooth has two externally visible components, the crown and root, and is made up of three distinct, inorganic, calcified tissues; enamel, dentine, and cementum (Scott 1967). The pulp is the only soft tissue, which provides the blood and nerve supply to the crown and root. The tooth crown projects into the mouth whereas the root is fixed in the jaws (Hillson 1996). In humans and other mammals, the tooth is secured by one or more roots, the periodontal membrane, and periodontal ligament into a socket formed by the alveolar bone of the upper or lower jaw. These root surfaces are covered by
cementum, which is deposited as a thin layer. The dentine forms the bulk of the tooth as it surrounds the pulp cavity, and is normally covered in one region by the enamel and in another by the cement (Scott 1967). Enamel covers the crown (Figure 3.1).

Figure 3.1 Representation of a permanent canine (after Hillson, 1996:11).

Of the calcified tissues of the teeth, the dentine and cement are of a collagenous nature, and thus, reveal similarities to bone (Scott 1967). As discussed by Scott (1967) the cement is most similar to bone, both chemically and histologically and is similar in its physiological behavior. Dentine is harder than cement or bone, and contains a greater proportion of inorganic material. The formation of dentine and cement continue throughout life, ultimately increasing their bulk. Where additions to the dentine are confined to the interior or pulpal surface, the additions to the cement are contained to its external surface. In addition, the cement has the ability to repair any loss of its substance produced by resorption, by deposits of fresh cement. Dentine, however is incapable of restoring any loss of its substance.
Enamel, the hardest tissue of the body, is a highly specialized calcified tissue “well suited to take the heavy wear involved in mastication” (Goodman and Rose 1990: 61). Archaeological settings often provide abundant samples of teeth as the high mineral content of enamel preserves teeth more so than the properties of bone (Alt et al. 1998). As teeth are also more resistant than bone to chemical and physical destruction, they are abundantly represented in the archaeological and paleontological record (White 2000). Unlike dentine, cement, or bone, enamel formation is not continuous throughout life. Therefore, once the thickness for the tooth has been produced, no more enamel is deposited (Scott 1967). Enamel is unique among the calcified tissues as it does not possess the ability of repairing itself to injury by means of cellular tissues.

During life, humans develop two sets of teeth, the deciduous and permanent dentitions. Much of the deciduous dentition is formed by birth and begins to erupt in the oral cavity around six months after birth. The deciduous eruption process is complete at about two and a half years. The permanent dentition begins to erupt in the oral cavity around six years of age, thereby replacing the deciduous dentition and continues until around twenty years of age (Scott 1967, Hillson 1996). Teeth are arranged into dental arches with teeth on the opposing side of the same arch (antimeres) seen as mirror images (Figure 3.2) and usually vary only slightly in size and form (Scott 1967).
Dental landmarks are used to describe the adjoining tissues and can be used as reference points when making observations. Longitudinal sections through a tooth help illustrate the boundaries separating distinct dental tissues. These boundaries are known as the cement-enamel junction (CEJ), the cement-dentine junction (CDJ), and the enamel-dentine junction (DEJ). The boundary separating the crown from the root is called the cervix. The inside of the tooth below the tooth surface contains the pulp chamber which holds soft pulp tissue (Figure 3.3).
Enamel Development and Histology

The two major processes involved in the development of enamel are matrix formation and the succeeding calcification (mineralization) of the matrix. Initially, matrix is completely organic, but begins to calcify soon after formation (Scott 1967). In mammalian tissues, mineralization is the process of deposition of inorganic material (Scott 1967). The overall structure of enamel occurs in the matrix formation stage and enamel mineralization when the matrix becomes calcified. The calcification process incorporates large amounts of mineral salts that produce changes in
the organic matrix. The later stages of enamel formation are referred to as the maturation stage. Fully formed enamel is totally acellular and almost completely composed of inorganic salt (Scott 1967).

While questions persist, the general pattern of enamel development and formation are well understood (Goodman and Rose 1990). The early stages of enamel formation are characterized by dome-like layers that lie at the core of future cusps. The domes are then surrounded by increasingly larger layers which are deposited on top, eventually forming the cusps (Hillson 1996). When each cusp has grown to its full height and width, the layers open out at the tip to complete the sides of the cusps. The ridges are formed by additional folds in the enamel organ that result in indentations in the layers (Hillson 1996). Sleeve-like layers form down the crown sides towards the cervix once the occlusal surface is formed. These layers become shorter and narrower until enamel formation ceases.

Amelogenesis

Ameloblasts are important cells in enamel formation as they secrete the enamel matrix (Guatelli-Steinberg 2001). Amelogenesis, or enamel formation and development has two different stages; the secretory stage (or matrix formation) and the maturation stage. The secretory stage consists of the formation of enamel, and initiates the beginning of mineralization, and the maturation stage is where mineralization is completed (Guatelli-Steinberg 2001, Hillson 1986). During the secretory stage, the ameloblasts secrete a matrix which consists of the proteins amelogenins and enamalins, as well as calcium and alkaline phosphates (Guatelli-Steinberg 2001). The high frequency (90%) of
amelogenins in the enamel matrix allow them to control the growth and orientation of enamel crystallites (Guatelli-Steinberg 2001). These hydroxyapatite crystals of the enamel form from minerals in the matrix. The crystals in this enamel forming stage are packed randomly within the dentine crystals. By the end of the secretion stage, the enamel is 30% mineralized and then the maturation stage begins (Guatelli-Steinberg 2001).

During the maturation stage, the ameloblasts are involved in a process where inorganic material replaces the water and organic material (mainly the amelogenin proteins) with more apatite in order to produce the heavily mineralized mature enamel (Guatelli-Steinberg 2001). Ameloblasts secrete materials needed for calcification as well as remove or break down the organic component of the matrix. This stage continues until 96% of the enamel tissue is calcified. "In a developing tooth crown, the forming edge is therefore characterized by a zone of enamel matrix" (Hillson 1996: 149).

Enamel Developmental Defects

Macroscopic enamel defects are characterized by enamel hypoplasias, which refer to a deficiency in the amount or thickness in enamel (Goodman 1990). Enamel hypoplasias can take the form of single or multiple pits, small furrows, to deep and wide troughs of decreased enamel thickness. The most severe type of enamel hypoplasia consists of entirely missing enamel. The formation of enamel hypoplasias are widely thought to occur as a result from physiological disturbances or perturbations to ameloblasts during matrix secretion (Hillson and Bond 1997, Goodman and Rose 1990). The macroscopic defects related to generalized disturbances occur in three different types
of hypoplasias that remain the basic classification: 1) Furrow-type defects or linear enamel hypoplasia (LEH), 2) Pit-type defects, and 3) Plane-type defects. These defects are often arranged in a band around the circumference of the crown following the trend of the perikymata\(^1\) resulting in missing enamel (Hillson 1996).

Enamel defects result from disruptions in the process of amelogenesis (Hillson 1996, Hillson and Bond 1997). The disruption during amelogenesis causes groups of ameloblasts in the forming enamel matrix to cease secreting matrix prematurely (Hillson and Bond 1997). Thus, enamel hypoplasias represent "disruptions to matrix secretion throughout the growing dentition" (Hillson 1996: 165), resulting in some amounts of missing enamel. Disruption during matrix formation can result in a hypoplastic line in the enamel which is sometimes accompanied by hypocalcification or opacity (Hillson 1996). There are several different types of developmental defects that occur in enamel. These include opacities (changes in enamel translucency), accentuated striae of Retzius (found in certain types of histological sections of enamel), and enamel hypoplasias (macroscopic defects appearing on the enamel surface) (Hillson 1996). Where opacities are caused by hypomineralization, the latter two result from disruptions to enamel-producing cells (ameloblasts), as they secrete the enamel matrix.

Hypoplasias can be reliably diagnosed resulting from hereditary anomalies, localized traumas, or systemic metabolic stress (Hillson and Bond 1997). Hereditary defects have only been noted in the archaeological record once (Cook 1980) and are rarely noted in clinical studies (Hillson and Bond 1997). In addition, hypoplasias due to

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\(^{1}\) Perikymata are surface manifestations of underlying striae of Retzius (Guatelli-Steinberg 2001). LEH result when a wider than expected margin of each brown striae of Retzius plane is exposed at the tooth surface, thus the disturbance affecting the Retzius planes will leave a furrow in the enamel surface larger than the perikymata groove (Guatelli-Steinberg 2001).
localized severe trauma are extremely rare in the archaeological record, and among modern human populations (Goodman and Rose 1990). Furrow-type enamel defects also referred to as linear enamel hypoplasias (LEH) are the most common form of hypoplasia macroscopically observed (Hillson and Bond 1997) and have long been associated with systemic physiologic disruption during tooth development (Goodman and Rose 1990). LEH is most commonly distinguished by a horizontal area of decreased enamel thickness down the crown side of the tooth. They are typically found on the sides of anterior tooth crowns but are also seen in the cervical half of posterior tooth crowns (Hillson 1996).

Pit-type defects vary in size with some representing interruptions to hundreds of ameloblasts and others representing a small group (Hillson 1996). Pit-type defects may occur as isolated pits and often two or more pits with different depths will represent a single growth disruption (Hillson and Bond 1997). Pit-type defects may be arranged in bands around the crown sides when associated with furrow-form defects, and will be observed as widely separated and quite large or small and close together, depending on the size of the furrow (Hillson 1996). Exposed-plane-form defects result when several planes of brown striae of Retzius are left partially or fully exposed. These defects can be identified by the resulting Tome’s process pits in which there is a marked step at the cervical margin where normal enamel and correlated perikymata resume (Hillson and Bond 1997).

The etiology of opacities can be multicausal as they are often associated with diagenetic changes, altered water content, and a direct result from hypoplasias (Hillson 1996). Hillson (1996) suggests that opacities result from both matrix secretion and maturation as poorly mineralized zones sometimes occupy the entire enamel thickness
indicating maturation was interrupted permanently, and other times poorly mineralized zones do not reach the surface implying the affected ameloblasts must have returned to normal. The etiology of enamel hypocalcifications are debated as research by Suckling (as cited by Goodman and Rose 1990) has suggested that hypocalcifications can form as a result from a disruption in matrix formation. Other research suggests that hypocalcifications typically result from disruptions in enamel maturation (Goodman and Rose 1990).

Dental Pathologies: Plaque Related Diseases

Dental plaque is responsible for the majority of common disease to affect fully erupted teeth (Hillson 1996). After teeth erupt into the mouth they are colonized by bacteria which adhere to dental surfaces, aiding to metabolize food entering the oral cavity (Hillson 1979). Teeth are vulnerable to microbial buildup due to the lack of a non-shedding mucosa surface as seen in the lips, palate, cheek, tongue and gums (Hillson 1996). If teeth are not frequently cleaned, these bacteria build up on the tooth surface, forming a layer extending around the cervical part of the crown. In human remains the presence of plaque is recognized in the form of mineralized plaque, or calculus (Hillson 2000) (Figure 3.4). Thus, calculus accumulates at the base of a living plaque deposit and attaches to the surface of the tooth (Hillson 1996). For a complete review of calculus etiology see Hillson (1996), Lieverse (1999).

Mineralization of plaque is required for calculus to form. The initiation of mineralization is correlated with the extent of plaque build up and depends on factors leading to plaque accumulation including poor oral hygiene and carbohydrate consumption (Hillson 1996). Two types of calculus are recognized: supra-gingival and
sub-gingival calculus. Supra-gingival calculus generally attaches to the enamel of the cervical crown as a band above the sub-gingival margin. Supra-gingival calculus deposits can be associated with healthy and periodontally diseased teeth whereas sub-gingival calculus (located within the gingival pocket) is always associated with periodontal disease (Lieverse 1999).

Figure 3.4 The physiology of dental plaque (after Hillson, 1979:148).

Periodontal Disease

Periodontal disease in human dental remains is identified when the surface cortical bone is lost exposing the porus cancellous structure of the supporting bone (Larsen 1997). This alveolar bone loss is progressive in periodontal disease and if left untreated, can result in tooth loss. Carious lesions and periodontal disease represent the
majority of tooth loss in the United States (Costa 1982). Periodontal diseases increases in severity with age and is the major source of tooth loss for persons over 40 (Costa 1982). The etiology of periodontal disease is complex as there are a number of factors involved in its on-set and progression. Although it is agreed upon that many different bacteria found in plaque influence the on-set of periodontal disease, other modifying factors include poor oral hygiene, cariogenesis, malocclusion, and nutritional status (Larsen 1997, Handler and Corruccini 1983).

The long-term presence of microorganisms in dental plaque deposits in the mouth gives rise to a range of conditions such as carious lesions and periodontal disease, which cause damage to the tissues and loss of supporting bone in the jaws (Hillson 2000). Gingivitis can occur as a result of plaque buildup which is demonstrated by an inflammation of the gums (Larsen 1997). Gingivitis does not necessarily affect the underlying alveolus which makes it impossible to detect in archaeological remains. However, it can intensify to the point where it manifests into a deeper lesion that involves all the periodontal tissues. This final stage of periodontal disease classified as periodontitis (Hillson 1996) is generally characterized by a loss of alveolar bone and is frequently demonstrated in the archaeological record (Larsen 1997).

The function of the periodontal tissues is to surround and support the teeth (Hillson 1996). These include the bone of the jaws, the periodontal ligament, cement, gingivae and mucosa. The alveolar process is the arch of bone which holds the dentition and consists of alveolae or tooth sockets. The periodontal ligament contains fibers that surround each tooth root and binds them into bone and cement. The mucosa (layer of soft tissue) covers the alveolar process and surrounds the base of each tooth crown. This area
is referred to as the gingivae.

The presence of large colonies of bacteria and micro-organisms next to the gingivae stimulates a response in the tissues of the jaw (Hillson 1996). The body has two defense mechanisms designed to fight plaque micro-organisms; these include innate and acquired immunity. Innate immunity includes factors always present such as phagocytes, bactericides in saliva, and the barrier of oral mucosa. Thus innate immune responses are designed to ingest and destroy plaque forming bacteria (Hillson 1979). Acquired immunity is a response of specific cells to produce antibodies to individual antigens.

Inflammation is characterized by the succession of changes occurring in tissues in response to injury or irritation. Inflammation that is normal or slight involves little tissue damage. However, when plaque deposits are extensive, it can trigger inflammation which damages body cells (Hillson 1979). This results in detachment of the collagen fibres which attach the gingivae and teeth to the underlying bone of the jaw. In periodontal inflammation, the initial site of irritation is known as a lesion (Figure 3.5) and its development is divided into four stages: initial lesion, early lesion, established lesion, and advanced lesion (Hillson 1996). The fourth stage (periodontitis) involves all of the periodontal tissues and it is at this stage where progressive resorption of alveolar bone occurs. Thus, as bone is resorbed around the tooth sockets, the tooth eventually falls out and the bone remodels to a smooth socketless surface. The effect of alveolar resorption and remodeling is referred to as progressive periodontal disease.
Caries

Carious lesions are characterized by destruction of enamel, or the focal mineralization of dental hard tissues by organic acids produced by bacterial fermentation of dietary carbohydrates by some plaque bacteria (Delgado-Darias et al. 2005, Hillson 2000). The corrosive action of these acids leads to progressive destruction of the tooth surface, ranging from enamel opacities, to the formation of an extensive cavity (or hole).
involving partial or complete loss of tooth crowns and roots (Larsen 1997, Hillson 1996). If the hole reaches the pulp chamber it may induce resorption of the neighboring tooth (Delgado-Darias et al. 2005) although in most cases, tooth loss associated with caries is the deliberate extraction of the tooth to treat tooth pain (Hillson 2000). The factors involved in caries etiology include the chemical composition of food, composition of saliva, oral hygiene, and the degree and rate of wear (Delgado-Darias et al. 2005). The disease process is slowly progressive and is characterized by arrested or remineralizing phases alternating with active phases resulting in a cavity that may remain stable for long periods of time (Hillson 1996).

Two main forms of carious lesions can be distinguished (Hillson 2000): coronal caries and root surface caries. Coronal caries are found in the enamel of the crown and if exposed by wear, can be seen in the dentine (Hillson 2000). Root surface caries take the form of shallow craters, are initiated at the cemento-enamel-junction (CEJ) and extend around the circumference of the root. Root surface caries only occur on root surfaces exposed above the margin of the gingivae. Whereas “root surface caries is associated with root exposure through periodontitis” (Hillson 1996: 274) coronal caries, especially pit and fissure caries are common in populations with a westernized diet (Hillson 2000). Root surface caries, interproximal, and cervical caries become more common with age and periodontal disease (Buikstra and Ubelaker 1994).

Carious lesions can also be divided into different categories depending on where they develop in the tooth crown and root (Hillson 1996). Pit and fissure site caries (or occlusal caries) occur most often in molars and premolars on the occlusal surface of the crown and smooth surface caries occur outside the fissures or pits on the sides of the
crown. Interproximal caries include the mesial and distal cervical regions of the tooth. Smooth surface caries rarely occur on the buccal and lingual sides of the crown as the level of gingival attachment recedes with age and leaves the lesion with an open area conducive for effective natural cleaning (Hillson 1996).

The role of diet and oral hygiene play an important factor in caries etiology. Poor oral hygiene leads to bacterial overgrowth which affects the amount of bacteria in dental plaque (Hillson 1979). Dietary carbohydrates such as sugar are metabolized more rapidly than other carbohydrates which causes more lactic acid to be produced more quickly. The pH of plaque varies depending on the amount of protein as opposed to carbohydrate in the diet (Hillson 1979). When plaque bacteria metabolizes carbohydrate, they produce lactic acid as a waste product whereas plaque bacteria metabolizing protein produces alkaline waste products (Hillson 1979). The balance between these periods of alterations of alkalinity and acidity determines whether the disease dental caries will occur (Hillson 1979).

The pH level of plaque fluid depends on the nutrients available to the plaque bacteria (Hillson 1979). The available nutrients are responsible for pH fluctuations throughout the day and long periods of low pH phases are accompanied by demineralization. Repeated administration of foods lowering plaque pH depresses plaque pH so that it cannot fully recover, thus contributing to demineralization of the enamel underneath the teeth (Hillson 1996, 1979). During acid phases the mineral of the enamel is dissolved but is replaced during the alkaline phases obtained from the saliva (Hillson 1979). When acid phases outnumber and last longer than the alkaline phases, the mineral is not replaced. Thus, carbohydrates such as sucrose is particularly responsible for
development of carious lesions because it involves rapid production of plaque and it is the most common sweetening agent in foods (Hillson 1996).

Summary

The understanding of enamel etiology and histology are essential in the interpretation of enamel defects and dental disease from archaeological populations. In order to reconstruct dietary and nutritional inadequacies, and overall dental health within the urban context of Caribbean slavery, a strong base in dental anatomy, and enamel development and histology must be obtained. Anthropologists studying human dental remains must be able to discriminate between different dental diseases and dental defects in order to properly record, interpret and reconstruct past dental health and gain insight into how past populations lived.
CHAPTER IV

MATERIALS AND METHODOLOGY

Research Problem

The purpose of this study is to analyze linear enamel hypoplasia rates and peak age of stress occurrence, dental disease, and unintentional tooth modifications in two historic human skeletal samples from two burial grounds of enslaved Africans and their descendents in Bridgetown, Barbados, and to compare enamel hypoplasia frequencies and peak age of occurrence with those from other populations of rural and urban enslaved Africans from the New World, ranging in time from the early 17th to the late 19th centuries. Overall frequencies of dental pathologies and unintentional tooth modifications will be compared with frequencies established by Corruccini et al. (1982) on a 17th to 19th c. burial ground of enslaved Africans from the Newton Plantation, Barbados.

Sample Population

The Pierhead skeletal collection, associated artifacts, and grave goods are currently stored at the archaeology laboratory at University of West Indies, Cave Hill campus. The Fontabelle skeletal collection, associated artifacts, and grave goods are currently stored at the Barbados Museum and Historical Society and were given accession numbers. Five were adult females, 2 were adult males, and one was an adult of unknown sex. Two were sub-adults with an unknown sex and were not used in the analysis as they would yield little information regarding dental disease. The Pierhead skeletal sample consisted of one adult female and one probable adult male. The two
samples were combined for dental analysis. The two combined samples used in this analysis yielded a total of 10 relatively complete individuals yielding 100 teeth and a total of 175 teeth for the entire tooth count sample.

Dental Sampling

The dentition from the Fontabelle burial ground sample consists of secondary burial contexts, the loose teeth associated with the secondary burials, and isolated dental remains. Secondary burials consist of incomplete human skeletal remains that have been “displaced from their original context by erosion, later intrusive burials” (Lukacs 1992: 136) or from other factors. Primary burial contexts include undisturbed burials which contain a complete skeleton in its original burial position (Lukacs 1992). The Pierhead skeletal sample consisted of secondary burial contexts, and loose associated teeth with the secondary burials.

The dental analysis of the two burial grounds employs two primary reporting methods previously demonstrated by Lukacs (1992): prevalence of dental disease/conditions on the basis of the number of relatively complete individuals affected by each disorder (individual count method), and prevalence and distribution of specific dental conditions by tooth type and class (tooth count method). The individual count method (the number of affected individuals/number of observable individuals) was used to represent the population prevalence of a given dental defect, disease or cultural modification. The tooth count method (number of affected teeth/number of observable teeth) was also used so as to permit a larger sample size which facilitates the comparison of disease frequencies between different tooth classes.
The tooth count consists of all of the affected teeth as a proportion of the number of observable teeth. The individual count consists of the number of affected individuals as a proportion of the observed individuals. These standards follow previous research conducted by Lovell and Whyte (1999). Lovell and Whyte (1999) state that the individual count method gives a sense of the proportion of the population that experienced the stress episode, and the tooth count frequencies allow a broader comparison among sub-samples and/or with other study samples. This method also allows all teeth to be observed for each sample.

In addition to the tooth-count and individual-count method, the dentitions were pooled to record the total sample, including the better preserved individuals, isolated, and loose teeth. This method allowed for an MNI to be configured and does not significantly inflate the sample size. The total sample was configured by separating the better preserved individuals from the loose teeth and assessing the loose teeth to specific better preserved individuals. The assignment of loose teeth to better preserved individuals was based on overall tooth morphology, size, aspects of dental disease such as severe hypercementosis of all roots, and similarities in discoloration of teeth. If loose teeth could not be assigned to a specific individual, it was categorized as isolated. The MNI and recording of specific dental diseases was constructed by assessing how many teeth were in each tooth class. For example, when counting the number of maxillary lateral incisors, all of the teeth from the left side of the arcade of the better preserved individuals and the associated loose teeth were counted. If the left maxillary incisor was missing, the right antimere was substituted if present.
Dental Analysis

All methodology of general dental inventory follows standard osteological procedures presented by Buikstra and Ubelaker (1994). For the purpose of this study, only individuals over the age of 16 were observed in the assessment of dental diseases and defects as there was an under representation of sub-adults and they yielded little information regarding dental disease. Dental inventory forms modeled after Buikstra and Ubelaker (1994) were used to record the presence or absence of permanent dentition, dental development, pathology and wear, and enamel defects. An additional form modeled after Reid and Dean (2000) was used to calculate the chronology of linear enamel hypoplasias of the anterior dentition (Appendix J).

Tooth Measurements

All of the dentition was observed macroscopically under diffused lighting with the aid of an illuminated hand-held 20x lens. Measurements of the dentition were taken with Helios needle-nose digital calipers calibrated to 0.05 mm. Three measurements of all observed permanent maxillary and mandibular dentition from the better preserved individuals were taken from both sides of the arcade. Measurements were taken using the dental measurements and morphology form modeled after Buikstra and Ubelaker (1994). Measurements consist of the mesiodistal diameter (crown length), the buccolingual diameter (crown width), and the crown height (Appendix A). The mesiodistal diameter is defined as the maximum width of the tooth crown in the mesiodistal plane. The buccolingual diameter is defined as the widest diameter of the tooth (on molars this is usually half the distance between the cemento-enamel junction, or
CEJ, and the occlusal surface). Crown height is measured from the occlusal surface to the CEJ on incisors, canines, and premolars. Molar crown height was taken from the mesiobuccal cusp (when present) to the CEJ (Figure 4.1).

Dental Inventory

A detailed dental inventory must be created in order to observe the presence and condition of all teeth (Buikstra and Ubelaker 1994). The visual coding system (Appendix B) assigns numbers 1-32 to the permanent dentition, starting from the maxillary right third molar (1), to the maxillary third left molar (16). The mandibular dentition started from the left third molar (17), to the right third molar (32). This form is useful as a reference for recording pathologies such as carious lesions, missing teeth, or cultural modifications. Additional charts were created to more fully represent tooth presence, pathology, dental development, dental attrition (Appendix C), supernumerary teeth and abscesses (Appendix D, Appendix E), and cultural modifications (Appendix F).

Figure 4.1 Measurement of crown height: a) posterior teeth; b) anterior teeth (after Buikstra and Ubelaker 1994:63).
Caries and Antemortem Tooth Loss

Carious lesions were recorded as zero (no lesion present), to seven, depending on the location of the caries on the tooth or in the root (below the cemento-enamel junction) (Appendix C). Distinctions were also made between the location of the carious lesion in order to decipher different types of lesions. For example, carious lesions present on the root below the CEJ are considered to be root-surface caries whereas carious lesions on the occlusal surface of the tooth indicate occlusal surface caries (Hillson 1996). The number of carious lesions per tooth and location were also recorded. In some instances, the origin of lesion could not be established due to the large size of the lesion (Figure 4.2). These carious lesions were recorded as large caries; which indicate that the cavities have destroyed so much of the tooth that point of origin is unknown. Carious lesions were scored on all permanent isolated teeth and of better preserved individuals, and were marked present if the lesion had invaded the dentin. This method reduces the amount of enamel pits that may be recorded as carious lesions (Cucina and Tiesler 2003).

Figure 4.2 Pierhead female exhibiting caries. Female, aged 21-35 years, from the Pierhead burial ground exhibiting large carious lesion of unknown origin. Photo with permission from Christopher Crain.
Three different indexes were used to determine caries rates within the two combined populations. The first index demonstrated by Kelley et al. (1991) is the most widely employed caries index:

Total number of carious teeth x 100/total number of teeth available for study

The second method employed is modeled after Kelley et al. (1991) which “increases the sensitivity of assessing dietary and other environmental sources of carious lesion formation” (Kelley et al. 1991:205). This is referred to as the total caries index: Total number of lesions per mouth x 100/total number of teeth available for study per mouth. The third index employs a rapid recording system demonstrated by Hillson (1996) initially proposed by Kerr (1990). This method allows for postmortem tooth loss, where caries frequency can be expressed as a percentage of the total number of those sites surviving, whether they are carious or non-carious. This index separates the anterior from posterior dentition so the overall percentage is not influenced by the pattern of postmortem tooth loss (Appendix G).

In anthropological studies, tooth loss, carious lesions, abscesses, and periodontitis have been utilized to assess general oral health of a population (Cucina and Tiesler 2003). Antemortem tooth loss (AMTL) has been observed in association with dental caries as once a carious lesion enters the pulpal chamber, “bacteria enter the systemic circulation, causing inflammatory responses that may eventually result in the loss of the tooth during the individual’s lifetime” (Cucina and Tiesler 2003:2). AMTL and periodontal disease are closely connected. AMTL can occur after a chronic severe case of gingival inflammation extends to the alveolar bone underneath the gum (Figure 4.3). This process causes the alveolar bone to retract and can lead to the eventual loss of the tooth. In
addition, AMTL can be a result of continuous eruption of the tooth caused by attrition. The physiological process related to the biting capacity in heavily worn teeth causes a tooth to continuously erupt.

Data on AMTL was recorded from alveolar bone. The tooth was considered to be absent when its alveolar bone was resorbed up to complete or almost complete closure of the tooth socket. A tooth was considered present when the corresponding tooth socket was present. Loose teeth that were not represented by their corresponding sockets were not considered for the assessment of AMTL.

Alveolar Resorption and Remodeling

Alveolar resorption was recorded using modified methods employed by Costa (1982). Four different levels of resorption were recorded on a scale from 1-4 with level 1 indicating no resorption, level 2 indicating slight resorption, level 3 indicating moderate resorption, and level 4 indicating extreme resorption. These levels are determined by the observer as measurements were not taken from the alveolar crest to the cement-enamel-junction (CEJ). This method allows for missing teeth to be determined missing as either ante or post mortem.
Periodontitis was recorded only when the bone crest of the alveolar margins showed either loss of the cortical surface, revealing porous cancellous spaces, or an altered morphology of the alveolar crest (Clark et al. 1986) (Figure 4.4). These changes resulted in the appearance of a porous bony surface or a "shelflike" margin instead of the normal "knife-edged" configuration. Larsen (1997) reports that there is a progressive increase in the CEJ to AC (alveolar crest) distance throughout life, and some evidence exists to indicate that this physiologic bone loss corresponds with the degree of compensatory process of continuous eruption. Therefore, no measurements were made
between the CEJ to AC when periodontal disease was assessed and observations were based on the criteria listed above.

Figure 4.4  Fontabelle female exhibiting horizontal alveolar bone loss. Accession # 30 BU 54. Female, aged 21+ from Fontabelle burial ground exhibiting horizontal alveolar bone loss over posterior dentition.

Hypercementosis

Hypercementosis was assessed by visual examination and was recorded as absent, slight, and severe in loose and isolated teeth, and in the mandibles or maxillae of relatively complete individuals where alveolar bone loss was so severe that the roots were exposed due to loss of bone. Slight hypercementosis included teeth with any sign of root cementum apposition and swelling; severe included lumpy appositions, bulbous apices, or complete root fusion (Figure 4.5).
Unintentional Cultural Modifications

Dentition was examined for unintentional cultural modifications, specifically, evidence of tobacco smoking through pipes as demonstrated by pipewear facets. The presence of habitual pipe-stem smoking is indicated by a semi-circular notch in the apical portion of the tooth (Figure 4.6). When a tooth has the corresponding tooth across antimeres or in dentition next to each other in the same arcade, the semi-circular notch of one tooth will correspond with the other tooth creating a half-circular notch.
Dental Defects: Enamel Hypoplasias

Analysis and recording of enamel hypoplasias, as well as all other macroscopic observations, were based on standard criteria outlined by Buikstra and Ubelaker (1994). Enamel defects were recorded by type of defect, severity of defect, location (which tooth it occurs on), location of defect on each tooth (labial or lingual side), frequency of defects, and distance from the defect to the CEJ (cemento-enamel junction). Enamel defects were scored on a scale from “0” (absent), to “7” (hypocalified enamel). Three types of defects were scored if observed: Linear enamel hypoplasia (LEH), enamel pitting, and hypocalcification linear band of discoloration.

Although three different defects can be scored, specific attention was focused on LEH as these types of hypoplasias are not hereditary (Larsen 1997), and are representative of physiological stress experienced in past and contemporary human populations (Hillson 1996) (Figure 4.7). The width of any observed LEH, and distance of LEH to the cemento-enamel junction was recorded using a pair of Helios digital calipers.
and measured to the nearest .02mm. To control for intraobserver error, a sample of teeth was measured with the same methods and the mean difference between repeat measures was calculated.

Figure 4.7  Fontabelle male exhibiting LEH and enamel pitting. Accession # 30 bu 54. Male, age 21+ from the Fontabelle burial ground exhibiting linear enamel hypoplasias (white arrow) and vertical pit-form hypoplasias (black arrows) on mandibular teeth. Photo with permission from Christopher Crain.

All observations were taken macroscopically (assessed visually) on the labial/anterior side of all permanent teeth and confirmed by the use of a 20X hand-held illuminated lens. Teeth with attrition extending beyond the incisal/occlusal third of the crown were not included in the study as minimal incidence of hypoplasias occur on the first third of crown development (Hillson 1996).

Sample Population

Research on LEH often consists of examining defects that span across two or more teeth (as cited by Guatelli-Steinberg 2003). This method helps eliminate defects
that may have been caused by localized stressors (Guatelli-Steinberg 2003, Goodman and Rose 1990). Due to the fragmentary nature and the size of the sample, it was not always possible to match defects across the tooth classes or across antimeres. Individuals were sometimes represented by a few teeth. Therefore, methodology used by (Guatelli-Steinberg 2003) was utilized for the analysis of enamel hypoplasias. This methodology consists of two reporting methods: the prevalence was first recorded for a subsample of individuals represented by two or more anterior/posterior teeth and that have crown lapping formation spans. The second method records defect prevalence by using the entire sample to report the frequency of affected teeth across the different tooth classes.

The under representation of males in the two combined samples did not allow for comparisons between males and females in assessing enamel defect frequencies between sexes. However, Lanphear (1990) reports little statistical significance in enamel hypoplasia frequencies between males and females in archaeological settings. Therefore, the male and female data are combined for analysis.

Tooth Selection

All permanent anterior and posterior dentition was observed for enamel defects. Teeth not included in this sample consisted of ones that had a large amount of enamel chipping, exhibited excessive tarter or calculus on the labial/buccal side of the tooth crown, or were missing more than one third of the tooth crown due to postmortem breakage, or attrition. Dental calculus was not cleaned from the teeth in order to hinder possible breakage of enamel or loosening of teeth from the sockets. This may interfere with the observations of defects although severe dental calculus was not exhibited in high
Enamel hypoplasias observed on teeth were classified as linear bands/lines, pit hypoplasias, or hypocalcifications. When a series of pit hypoplasias form in a linear plane, they were classified as a band/line since they were symmetrical across the lingual plane (Goodman and Rose 1990). Bands/lines were defined according to their severity in form. For example, bands in their milder form (often referred as “fever lines”) are represented by faint encircling bands. The other form of band/lines were represented by growth arrest lines (deeply indented horizontal depressions running completely around the tooth (Handler and Corrucinni 1983). Growth arrest lines are often associated with extreme dietary deficiency or starvation (Handler and Corrucinni 1983) or from long periods of non-specific systemic stress possibly associated with disease (Goodman and Rose 1990) which causes the body to stop growing because it lacks nutritional reserves.

Recordation

Observations of the labial/buccal and lingual surface were made by gross visual examination and confirmed with a hand-held 20x illuminated lens. All disturbances that were felt by running a dental probe along the surface of the tooth from the crown to the cemento-enamel junction (CEJ) were recorded. Total crown height measurements were taken along with measurements from the CEJ to the occlusal portion of the defect (Appendix H). The type of enamel defect was determined by codes previously determined by Buikstra and Ubelaker (1994) (Appendix I). Defects and absence of
defects were recorded on a number system ranging from 0 (no defect) to 7 (diffuse boundary opacity). Color codes for opacities (hypocalcifications) were also determined using standards provided by Buikstra and Ubelaker (1994).

Chronology of LEH

The method by Reid and Dean (2000) has been used to calculate the age of hypoplastic stress events for all anterior teeth exhibiting LEH (Appendix J). This method employs the total time taken to form the crowns of each anterior tooth by cuspal enamel formation, and dividing each percentile of total tooth height. Each anterior maxillary and mandibular tooth was previously assessed by Reid and Dean (2000) and assigned crown formation times based on histological estimates for the time of initiation of mineralization of each tooth. This method was used as it does not assume a linear relationship between crown height and the total time taken to form enamel.

Permanent anterior teeth exhibiting enamel defects were measured from the CEJ to the most occlusal portion of the defect. This method utilizes anterior teeth and estimates the mean chronological age at crown completion of each anterior tooth individually as considerable variation exists in mineralization time between each anterior tooth (Reid and Dean 2000).

Comparison Populations

A number of enamel hypoplasia, and dental disease studies have been selected for comparison with the Pierhead/Fontabelle sample. Data has been gathered for comparison of frequencies of enamel hypoplasias and dental disease occurrence and of chronology of
enamel defects of anterior teeth. In addition, comparison between non-intentional cultural modifications of teeth will be compared between the selected studies where information is available.
CHAPTER V

RESULTS

Two methods of analysis are utilized for recording dental defects, dental disease, and cultural modifications among the Pierhead/Fontabelle sample. These consist of the individual count sample, and the total sample (tooth count). The individual count sample yielded 10 relatively complete individuals, with a total of 100 observable teeth. The total sample consists of relatively complete individuals, the associated loose dentition, and isolated teeth yielding a total of 175 observable teeth. The male and female data are combined for analysis in assessing LEH and carious lesion frequencies due to the under representation of adult males in the sample, and absence of LEH and carious lesions exhibited in the males. Male and female data are separated for analysis when assessing antemortem tooth loss, alveolar bone loss, hypercementosis, and cultural modifications.

Tables 5.1-5.2 summarize the overall frequencies and percentages of dental defects, pathologies, and cultural modifications within the Pierhead/Fontabelle sample. Table 5.1. summarizes frequencies by individual count for the relatively complete individuals. Among the relatively complete individuals from the Pierhead/Fontabelle sample, five individuals (50%) exhibited carious lesions in at least one tooth. The highest rate of dental pathologies consisted of antemortem tooth loss and alveolar bone loss. Six individuals (60%) exhibited antemortem tooth loss and seven (70%) out of ten individuals exhibited horizontal alveolar bone loss exposing at least one half of the tooth roots. Enamel defects and pathologies in the form of linear enamel hypoplasias (LEH), hypercementosis, and cultural modifications in the form of pipe-stem facets were
exhibited in 3 individuals (30%).

Table 5.2 summarizes frequencies by the total sample. Carious lesions were exhibited in 47 teeth out of 175, affecting 27% of the total tooth sample.

Hypercementosis was exhibited in 15 teeth out of 175, affecting 9% of the total tooth sample. Enamel defects in the form of LEH were exhibited in 24 teeth out of 175, affecting 14% of the total tooth sample. Cultural modifications in the form of pipe-stem facets were exhibited in 5 teeth out of 175, affecting 3% of the total tooth sample.

Table 5.1 Individual count: Frequencies and percentages of dental pathologies, defects and cultural modifications among the Pierhead/Fontabelle (n=10; total number of teeth=100)

<table>
<thead>
<tr>
<th></th>
<th>Number of individuals affected by pathology or alteration</th>
<th>Percentage of individuals affected by pathology or alteration</th>
<th>Number of instances</th>
<th>Percentage of teeth affected by pathology or alteration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caries</td>
<td>5</td>
<td>50</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>AMTL</td>
<td>6</td>
<td>60</td>
<td>38*</td>
<td>24</td>
</tr>
<tr>
<td>Alveolar Bone Loss</td>
<td>7</td>
<td>70</td>
<td>79*</td>
<td>49</td>
</tr>
<tr>
<td>Hypercementosis</td>
<td>3</td>
<td>30</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Enamel Defects</td>
<td>3</td>
<td>30</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Cultural Modifications</td>
<td>3</td>
<td>30</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

*Note that AMTL and Alveolar bone loss percentage of teeth affected was assessed by averaging the number of observable tooth sockets for the relatively complete individuals. Thus, the estimated observable number of tooth sockets per individual yielded 16.
Table 5.2  Total sample (tooth count sample): Frequencies and percentages of dental pathologies, defects, and cultural modifications among the Pierhead/Fontabelle (total number of teeth= 175)

<table>
<thead>
<tr>
<th></th>
<th>Number of instances</th>
<th>Percentage of teeth affected by pathology or alteration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caries</td>
<td>47</td>
<td>27</td>
</tr>
<tr>
<td>Hypercementosis</td>
<td>15</td>
<td>9</td>
</tr>
<tr>
<td>Enamel Defects</td>
<td>24</td>
<td>14</td>
</tr>
<tr>
<td>Cultural Modifications</td>
<td>5</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 5.3 presents enamel defects, dental pathologies, and cultural modification frequencies for the individual count sample from the Newton Plantation (Corruccini et al. 1982). The data presented in table 5.3 is summarized from Corruccini et al. (1982) and is presented in order for comparisons of dental defects, dental pathologies, and cultural modification frequencies to be made with the data analyzed in this study.

Table 5.4 summarizes the overall frequencies of dental pathologies and cultural modifications for the individual count of the Pierhead/Fontabelle sample. Carious lesions are broken into two types of lesion consisting of occlusal surface caries and interproximal carious lesions. Occlusal carious lesions were exhibited in 5 out of 10 individuals whereas interproximal carious lesions were only exhibited in 1 out of 10 individuals. Enamel hypoplasias occurring on anterior teeth were exhibited in 1 out of 10 individuals and enamel hypoplasias occurring on the posterior teeth were exhibited in 2 out of 10 individuals.
Table 5.3 Occurrence of pathologies and cultural modifications of teeth from the Newton Plantation, Barbados

<table>
<thead>
<tr>
<th>Trait</th>
<th>Occurrences/n\textsuperscript{a}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occlusal surface caries</td>
<td>19/94</td>
</tr>
<tr>
<td>Interproximal caries</td>
<td>54/94</td>
</tr>
<tr>
<td>Tooth Loss</td>
<td>28/55</td>
</tr>
<tr>
<td>Hypercementosis</td>
<td>11/21</td>
</tr>
<tr>
<td>Anterior tooth enamel hypoplasia</td>
<td>66/68</td>
</tr>
<tr>
<td>Posterior tooth enamel hypoplasia</td>
<td>93/94</td>
</tr>
<tr>
<td>Pipewear facets</td>
<td>25/60</td>
</tr>
</tbody>
</table>

\textsuperscript{a} is the number of adequately preserved individuals for the trait (after Corruccini et al. 1982)

Table 5.4 Occurrence of pathologies and cultural modifications of teeth among the Pierhead/Fontabelle sample

<table>
<thead>
<tr>
<th>Trait</th>
<th>Occurrences/n\textsuperscript{a}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occlusal surface caries</td>
<td>5/10</td>
</tr>
<tr>
<td>Interproximal caries</td>
<td>1/10</td>
</tr>
<tr>
<td>Antemortem tooth loss</td>
<td>6/10</td>
</tr>
<tr>
<td>Hypercementosis</td>
<td>3/10</td>
</tr>
<tr>
<td>Anterior tooth enamel hypoplasia</td>
<td>1/10</td>
</tr>
<tr>
<td>Posterior tooth enamel hypoplasia</td>
<td>3/10</td>
</tr>
<tr>
<td>Pipewear facets</td>
<td>3/10</td>
</tr>
</tbody>
</table>

\textsuperscript{a} is the number of adequately preserved individuals for the trait
Enamel Defects

Presence or Absence of LEH

In the Pierhead/Fontabelle sample, out of 10 individuals sampled, the total number of individuals affected by enamel hypoplasia in the form of LEH is 3 (Table 5.1). By dividing the number of individuals affected by the total number of individuals in the sample, a percent was able to be calculated for the percentage of individuals affected by LEH, 30%. Among the 3 individuals, 30 BU 54, a 21+ year-old male expressed LEH across all observed mandibular teeth with multiple stress episodes exhibited in the canine. The total number of instances for the individual count sample, where LEH affected the teeth was 10% (10 teeth affected with LEH, out of 100 observable teeth).

When examining the dentition for the total sample (individuals, the associated loose dentition, and isolated dentition), the total frequency of LEH is 14% (24 teeth with at least one instance of LEH, out of 175 observable teeth (Table 5.2). The average frequency of LEH for the maxilla is 11.3% (10 teeth with at least one incidence of LEH out of 88 observable teeth (Table 5.5). The average frequency for the mandible is 16% (14 teeth with at least one incidence of LEH out of 87 observable teeth) (Table 5.6). Tables 5.5-5.8 also display enamel hypoplasia frequency by left and right sides of the dental arcade. When the teeth are separated by left and right sides (mandible and maxilla combined) the right side has 19.76% of teeth affected with LEH (17 teeth out of 86 with at least 1 enamel hypoplasia), while the left side has only .07% of teeth affected with LEH (7 teeth out of 89 with at least one hypoplasia). When the data is broken down by tooth class, the canine has the highest LEH frequency of 40% (10 teeth with LEH, out of
25 total teeth), the third premolar at 18.5% (5 teeth with LEH, out of 27 total teeth),
the first molar at 15.4% (4 teeth with LEH, out of 26 total teeth), and the central and
lateral incisors at .08% (2 teeth with LEH, out of 26 total teeth).

Age at Occurrence of LEH

Age at occurrence is calculated for individual hypoplastic events, the
mandibular canine exhibits consistent age at formation times (Table 5.9). The peak time
of occurrence for all mandibular canines is between 3.6 and 4.2 years. One individual
experienced multiple hypoplastic events in the canine where the first event occurred
between 2.7 to 3.1 years, and the last episode occurred between 3.6 and 4.2 years. Peak
age at occurrence for the maxillary incisors was between 2.9 and 3.4 years. No
mandibular incisors exhibited any hypoplastic events that could be measured. The
maxillary canines exhibited 2 different age ranges for peak age at occurrence. The
maxillary left canine exhibited a stress event occurring between 2.7 and 3 years, and the
maxillary right canine exhibited a stress event occurring between 3.8 and 4.3 years. When
the peak age of occurrence for hypoplastic events is combined, the peak age at
occurrence is approximately between 3 to 4 years.
Table 5.5

Frequency of enamel hypoplasia by tooth class among the Pierhead/Fontabelle sample: Maxillary dentition

<table>
<thead>
<tr>
<th>Left Tooth Class</th>
<th>Total # of Teeth</th>
<th>Number with at least one hypoplasia</th>
<th>Right Tooth Class</th>
<th>Total # of Teeth</th>
<th>Number with at least one hypoplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>I1</td>
<td>5</td>
<td>1 (20%)</td>
<td>I1</td>
<td>2</td>
<td>1 (50%)</td>
</tr>
<tr>
<td>I2</td>
<td>3</td>
<td>0</td>
<td>I2</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>C</td>
<td>6</td>
<td>1 (16%)</td>
<td>C</td>
<td>5</td>
<td>3 (60%)</td>
</tr>
<tr>
<td>P3</td>
<td>9</td>
<td>0</td>
<td>P3</td>
<td>6</td>
<td>2 (33%)</td>
</tr>
<tr>
<td>P4</td>
<td>10</td>
<td>0</td>
<td>P4</td>
<td>5</td>
<td>1 (20%)</td>
</tr>
<tr>
<td>M1</td>
<td>7</td>
<td>1 (14%)</td>
<td>M1</td>
<td>5</td>
<td>1 (20%)</td>
</tr>
<tr>
<td>M2</td>
<td>8</td>
<td>1 (12%)</td>
<td>M2</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>M3</td>
<td>4</td>
<td>0</td>
<td>M3</td>
<td>5</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 5.6

Frequency of enamel hypoplasia by tooth class among the Pierhead/Fontabelle sample: Mandibular dentition

<table>
<thead>
<tr>
<th>Left Tooth Class</th>
<th>Total # of Teeth</th>
<th>Number with at least one hypoplasia</th>
<th>Right Tooth Class</th>
<th>Total # of Teeth</th>
<th>Number with at least one hypoplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>I1</td>
<td>3</td>
<td>0</td>
<td>I1</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>I2</td>
<td>2</td>
<td>0</td>
<td>I2</td>
<td>4</td>
<td>1 (25%)</td>
</tr>
<tr>
<td>C</td>
<td>7</td>
<td>0</td>
<td>C</td>
<td>7</td>
<td>2 (29%)</td>
</tr>
<tr>
<td>P3</td>
<td>4</td>
<td>1 (25%)</td>
<td>P3</td>
<td>8</td>
<td>2 (25%)</td>
</tr>
<tr>
<td>P4</td>
<td>5</td>
<td>0</td>
<td>P4</td>
<td>6</td>
<td>1 (17%)</td>
</tr>
<tr>
<td>M1</td>
<td>7</td>
<td>0</td>
<td>M1</td>
<td>7</td>
<td>2 (29%)</td>
</tr>
<tr>
<td>M2</td>
<td>6</td>
<td>1 (17%)</td>
<td>M2</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>M3</td>
<td>4</td>
<td>0</td>
<td>M3</td>
<td>7</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 5.7  Frequencies and percentages of enamel hypoplasias in mandibular dentition among the Pierhead/Fontabelle sample (total number of teeth = 87)
Table 5.8  Frequencies and percentages of enamel hypoplasias in maxillary dentition among the Pierhead/Fontabelle sample (total number of teeth =88)

<table>
<thead>
<tr>
<th>Tooth Type</th>
<th>One defect</th>
<th>&gt; than one defect</th>
</tr>
</thead>
<tbody>
<tr>
<td>I1</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>I2</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>P3</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>P4</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>M1</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>M2</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>M3</td>
<td>9</td>
<td></td>
</tr>
</tbody>
</table>

Enamel Hypoplasia: Total Sample
Table 5.9  Timing of linear enamel hypoplasia in anterior teeth among the Pierhead/Fontabelle sample

<table>
<thead>
<tr>
<th>Tooth</th>
<th>Crown Height</th>
<th>Distance to CEJ</th>
<th>Time of Occurrence in Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>RI</td>
<td>9.12 mm</td>
<td>2.78 mm</td>
<td>2.9-3.4 years</td>
</tr>
<tr>
<td>LI$^1$</td>
<td>9.07 mm</td>
<td>3.41 mm</td>
<td>2.9-3.4 years</td>
</tr>
<tr>
<td>LC</td>
<td>9.12 mm</td>
<td>5.16 mm</td>
<td>2.7-3.0 years</td>
</tr>
<tr>
<td>RC</td>
<td>9.02 mm</td>
<td>2.59 mm</td>
<td>3.8-4.3 years</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LC</td>
<td>9.48 mm</td>
<td>3.10 mm</td>
<td>3.6-4.2 years</td>
</tr>
<tr>
<td>RC</td>
<td>9.02 mm</td>
<td>3.07 mm</td>
<td>3.6-4.2 years</td>
</tr>
<tr>
<td>RC</td>
<td>9.04 mm</td>
<td>3.70 mm</td>
<td>3.6-4.2 years</td>
</tr>
<tr>
<td>RC</td>
<td>9.39 mm</td>
<td>3.38 mm</td>
<td>3.6-4.2 years</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4.21 mm</td>
<td>3.1-3.6 years</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4.94 mm</td>
<td>2.7-3.1 years</td>
</tr>
</tbody>
</table>
Comparison Populations

Presence or Absence of LEH

The frequency of LEH occurrence between comparison populations is demonstrated in Table 5.10. The frequencies of four New World rural enslaved populations were compared with the Pierhead/Fontabelle population. The comparison populations include a 19th c. site from Maryland and Virginia, the Catoctin Furnace and Clifts Plantation Sites dating between the 18th -19th c., the Newton Plantation Site of Barbados, dating between the 17th -19th centuries, and 38CH778, a South Carolina plantation 19th c. site.

The frequencies of LEH occurrence between comparison populations ranges from 26.0 % to 89.0%. The Pierhead/Fontabelle sample, the only urban enslaved population, has a much lower frequency of LEH compared with 3 of the 4 comparison populations. When assessing percentages of LEH among the comparison populations, the highest percentage of LEH was exhibited in the Catoctin Furnace and Clifts Plantation Site, accounting for 89% of the individuals. Frequencies of LEH were also high in the South Carolina Plantation Site, 38CH778, accounting for 77.4% of the individuals. Based on the comparisons, the Pierhead/Fontabelle population most closely resembles the 19th c. Maryland and Virginia population with a frequency of 26.0% and the Pierhead/Fontabelle population frequency at 30.0%.

Age at Occurrence of LEH

The results of comparisons of peak age of LEH occurrence between the Pierhead/
Fontabelle population and other populations reveal a similarity between the Caribbean rural plantation and urban enslaved populations (Table 5.11). The New World Caribbean samples exhibit peak age at occurrence ranges between 3 and 4 years of age, while the peak age at occurrence for the Catoctin Furnace Site and Virginia Cliffs Plantation Site range from 1.5 to 4.5 years. However, the agricultural site from Pasion River, Guatemala AD 900 also exhibits peak age at occurrence between 3 and 4 years of age.

Caries

Presence or Absence of Caries

In the Pierhead/Fontabelle sample, out of 10 individuals sampled, the total number of individuals affected by carious lesions was 5 (Table 5.1). By dividing the number of individuals affected by the total number of individuals in the sample, a percent was able to be calculated for the percentage of individuals affected by carious lesions, 50%. Among the 5 individuals, 4 individuals exhibited multiple lesions across tooth classes consisting primarily of the mandibular molars. The total number of instances for the individual count sample, where carious lesions affected the teeth was 20% (20 teeth affected with carious lesions out of 100 observable teeth).
Table 5.10

Frequency of LEH occurrence in comparison populations and the Pierhead/Fontabelle sample

<table>
<thead>
<tr>
<th>Population/Site</th>
<th>Period</th>
<th>Subsistence</th>
<th>No.</th>
<th>LEH</th>
<th>%</th>
<th>Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maryland &amp; Virginia</td>
<td>AD 19(^{th}) c.</td>
<td>Agricultural enslaved</td>
<td>34</td>
<td>9</td>
<td>26.0</td>
<td>(Kelley and Angel 1987)</td>
</tr>
<tr>
<td>Catoctin Furnace, Maryland</td>
<td>AD 18-19(^{th}) c.</td>
<td>Agricultural enslaved</td>
<td>27</td>
<td>24</td>
<td>89.0</td>
<td>(Blakey et al. 1994)</td>
</tr>
<tr>
<td>Cliffs Plantation, Virginia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Newton Plantation, Barbados</td>
<td>AD 17-19(^{th}) c.</td>
<td>Agricultural enslaved</td>
<td>103</td>
<td>56</td>
<td>54.5</td>
<td>(Corruccini et al. 1985)</td>
</tr>
<tr>
<td>Pierhead/Fontabelle, Barbados</td>
<td>AD 17-19(^{th}) c.</td>
<td>Urban enslaved</td>
<td>10</td>
<td>3</td>
<td>30.0</td>
<td>(this study)</td>
</tr>
<tr>
<td>Population/Site</td>
<td>Period</td>
<td>Subsistence</td>
<td>Peak age at stress</td>
<td>Sources</td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------------------------------------------</td>
<td>--------------</td>
<td>----------------------</td>
<td>--------------------</td>
<td>-----------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Catoctin Furnace, Maryland Clifts Plantation, Virginia</td>
<td>AD 18-19th c.</td>
<td>Agricultural enslaved</td>
<td>1.5-4.5</td>
<td>(Blakey et al. 1994)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Newton Plantation, Barbados</td>
<td>AD 17-19th c.</td>
<td>Agricultural enslaved</td>
<td>3-4</td>
<td>(Corruccini et al. 1985)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pierhead/Fontabelle, Barbados</td>
<td>AD 17-19th c.</td>
<td>Urban enslaved</td>
<td>3-4</td>
<td>(this study)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>South Carolina Plantation, Site: 38CH778</td>
<td>AD 19th c.</td>
<td>Agricultural enslaved</td>
<td>2-4</td>
<td>(Rahtbun 1987)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pasion River, Guatemala</td>
<td>AD 900</td>
<td>Agriculturalists</td>
<td>3-4</td>
<td>(Wright 1997)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
When anterior teeth for the individual count are examined, no carious lesions are present (Tables 5.12-5.13). The posterior teeth contain 100% of carious lesions in the Pierhead/Fontabelle sample. Among the posterior teeth, the second and third left-mandibular molars contain the highest rate of carious lesions, accounting for 45% of carious teeth (9 teeth out of 20 carious teeth). The right maxillary second and third molars contain the next highest frequency at 20% (4 teeth out of 20 carious teeth).

When examining the dentition for the total sample (individuals, the associated loose dentition, and isolated dentition), the total frequency of carious lesions is 27% (47 teeth with at least one lesion out of 175 observable teeth) (Table 5.2). This is the minimum number of carious lesions for the total sample. The average frequency of carious lesions for the maxilla is 17.0% (15 teeth with at least one incidence of carious lesions out of 88 observable teeth (Table 5.14). The average frequency for the mandible is 36.7% (32 teeth with at least one incidence of carious lesions out of 87 observable teeth) (Table 5.15). When the teeth are separated by left and right sides (mandible and maxilla combined) the right side has 18.8% of teeth with at least one carious lesion (16 teeth with one or more lesions out of 85 total teeth), while the left side has 34.4% of teeth with at least one carious lesion (31 teeth with one or more lesions out of 90 total teeth) (Tables 5.12-5.13).
Table 5.12

Frequency of carious lesions by tooth class among the Pierhead/Fontabelle sample: Maxillary dentition

<table>
<thead>
<tr>
<th>Left Tooth Class</th>
<th>Total # Teeth</th>
<th># with one lesion</th>
<th># with 2 or more</th>
<th>Right Tooth Class</th>
<th>Total # Teeth</th>
<th># with one lesion</th>
<th># with 2 or more</th>
</tr>
</thead>
<tbody>
<tr>
<td>I1</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>I1</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>I2</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>I2</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>C</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>C</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>P3</td>
<td>9</td>
<td>1 (11%)</td>
<td>0</td>
<td>P3</td>
<td>6</td>
<td>1 (16%)</td>
<td>0</td>
</tr>
<tr>
<td>P4</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>P4</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>M1</td>
<td>7</td>
<td>4 (57%)</td>
<td>1 (14%)</td>
<td>M1</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>M2</td>
<td>8</td>
<td>3 (38%)</td>
<td>1 (12%)</td>
<td>M2</td>
<td>5</td>
<td>1 (20%)</td>
<td>1 (20%)</td>
</tr>
<tr>
<td>M3</td>
<td>4</td>
<td>1 (25%)</td>
<td>1 (25%)</td>
<td>M3</td>
<td>5</td>
<td>1 (20%)</td>
<td>1 (20%)</td>
</tr>
</tbody>
</table>
Table 5.13

Frequency of carious lesions by tooth class among the Pierhead/Fontabelle sample: Mandibular dentition

<table>
<thead>
<tr>
<th>Left Tooth Class</th>
<th>Total # Teeth</th>
<th># with one lesion</th>
<th># with 2 or more</th>
<th>Right Tooth Class</th>
<th>Total # Teeth</th>
<th># with one lesion</th>
<th># with 2 or more</th>
</tr>
</thead>
<tbody>
<tr>
<td>I1</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>I1</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>I2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>I2</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>C</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>C</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>P3</td>
<td>4</td>
<td>1 (25%)</td>
<td>0</td>
<td>P3</td>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>P4</td>
<td>5</td>
<td>1 (20%)</td>
<td>0</td>
<td>P4</td>
<td>6</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>M1</td>
<td>7</td>
<td>2 (29%)</td>
<td>0</td>
<td>M1</td>
<td>7</td>
<td>3 (42%)</td>
<td>0</td>
</tr>
<tr>
<td>M2</td>
<td>6</td>
<td>4 (67%)</td>
<td>2 (29%)</td>
<td>M2</td>
<td>6</td>
<td>2 (33%)</td>
<td>0</td>
</tr>
<tr>
<td>M3</td>
<td>4</td>
<td>3 (75%)</td>
<td>3 (75%)</td>
<td>M3</td>
<td>7</td>
<td>4 (57%)</td>
<td>2 (29%)</td>
</tr>
</tbody>
</table>
The Total Caries Index for the Pierhead/Fontabelle individuals is presented in table 5.16. Five out of 10 individuals in the Pierhead/Fontabelle sample exhibiting carious lesions were females. The Total Caries Index ranges from 14.8% to 50% of all observable teeth. The female exhibiting the highest total caries index exhibited 50% and was aged between 30-50 years old. The female exhibiting the lowest caries index exhibited a caries index of 14.8% and was aged between 20-30 years.

Table 5.14  Frequencies and percentages of carious lesions in maxillary dentition among the Pierhead/Fontabelle sample (total number of teeth =88)
Table 5.15  Frequencies and percentages of carious lesions in mandibular dentition among the Pierhead and Fontabelle sample (total number of teeth = 87)
Table 5.16  Total caries index for the Pierhead/Fontabelle sample

<table>
<thead>
<tr>
<th>Sample</th>
<th>Age Group</th>
<th>Caries Index</th>
<th>Caries Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pierhead</td>
<td>Female, age 20-30:</td>
<td>14.8%</td>
<td>4 caries out of 27 observable teeth</td>
</tr>
<tr>
<td>30 BU 61:</td>
<td>Female, age 35-45:</td>
<td>25%</td>
<td>1 caries out of 4 observable teeth</td>
</tr>
<tr>
<td>30 BU 94:</td>
<td>Female, age 21+:</td>
<td>46%</td>
<td>6 caries out of 13 observable teeth</td>
</tr>
<tr>
<td>30 BU 147:</td>
<td>Female, age 21+:</td>
<td>23.1%</td>
<td>3 caries out of 13 observable teeth</td>
</tr>
<tr>
<td>30 BU 89:</td>
<td>Female, age 30-50:</td>
<td>50%</td>
<td>5 caries out of 10 observable teeth</td>
</tr>
</tbody>
</table>

When carious lesions were assessed by initiation sites for Pierhead/Fontabelle individual count (relatively complete individuals), pit and fissure sites accounted for 18% of all 51 sites present. Approximal sites exhibited only 2% out of 88 sites present, and cervical sites as demonstrated by root-surface lesions were exhibited in only 1% of 23 sites present in the individual count sample (Table 5.17).
Table 5.17  Initiation sites and recording for carious lesions in posterior dentition among the Pierhead/Fontabelle sample. Rapid scoring sheet for counting caries in relation to initiation sites for the individual count

Divide dentition into permanent anterior, and permanent posterior teeth. For each division, record counts of sites present and sites carious as follows:

<table>
<thead>
<tr>
<th>Sites</th>
<th>Present</th>
<th>Carious</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fissure and pit sites</td>
<td>51</td>
<td>18</td>
</tr>
<tr>
<td>Approximal Sites</td>
<td>88</td>
<td>2</td>
</tr>
<tr>
<td>Cervical Sites</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>enamel caries</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CEJ caries</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>root surface caries</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>gross cervical caries</td>
</tr>
<tr>
<td>Tooth Count</td>
<td>51</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>gross crown caries</td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>gross gross caries</td>
</tr>
</tbody>
</table>

Lesions are expressed as percentages of sites present. Count 1 fissure & pit per cheek tooth, if any vestige of fissure system present. Count 2 approximal sites (mesial and distal) per tooth and 1 for most distal tooth in quadrant. Count 1 cervical site per tooth, if any vestige of CEJ is present

(After Hillson 1996: 281)
Antemortem Loss

Antemortem loss was determined by counting the number of observable sockets per relatively complete individual and counting the number of teeth lost before death. A total of 38 teeth out of 99 observable tooth sockets were marked as antemortem tooth loss. Six individuals out of 10 (60%) exhibited antemortem tooth loss. Among the Pierhead/Fontabelle females, 3 were missing a total of 14 teeth due to antemortem loss while 2 males were missing a total of 21 teeth due to antemortem loss. The sex of the sixth individual was not able to be assigned. This individual was missing 3 teeth. The highest frequency of missing teeth occurred in the mandible, accounting for 30 out of 38 teeth marked as being lost antemortem. The 6 individuals exhibiting antemortem tooth loss had 99 present tooth sockets; 75 mandibular and 24 maxillary. The high frequency of antemortem tooth loss of the mandibular dentition may be attributed to the small number of maxillae present in the sample. Table 5.18 demonstrates the age and sex distribution, and percentage of teeth missing antemortem among the Pierhead/Fontabelle sample.

Alveolar Resorption

Since previous methods measuring the distance from the CEJ to the AC have proved erroneous (Molnar and Molnar 1985, Costa 1982) only moderate to severe horizontal alveolar bone loss was marked in order to limit over representation in recording frequencies. Moderate bone loss was demonstrated by one-half root exposure, and severe bone loss was demonstrated by two-thirds root exposure.

Horizontal alveolar bone loss of moderate to severe was noted in 6 (60%) of
relatively complete individuals. Among the 60% demonstrating horizontal alveolar bone loss, 30% were males and the other 30% were females. Two of the males exhibited severe horizontal bone loss where 2/3 of the roots were exposed over all observable teeth. Of special note is the male from Pierhead. This individual exhibited an almost complete edentulous mandible with only 3 teeth present. In addition, the teeth revealed severe attrition, leaving a flat occlusal surface and exposed dentin.

Among the 3 females exhibiting horizontal bone loss, 2 were aged around 40 years with 1 female exhibiting severe bone loss but only slight dental wear, while the other female exhibited moderate bone loss, slight attrition, and hypercementosis of the roots. Table 5.19 demonstrates age and sex distribution, along with frequency of horizontal bone loss and severity of occlusal attrition among the Pierhead/Fontabelle individuals.

Table 5.18  Age distribution, number of individuals and frequency of AMTL among the Pierhead/Fontabelle sample

<table>
<thead>
<tr>
<th>Accession #</th>
<th>Age</th>
<th>Sex</th>
<th># of observed tooth sockets</th>
<th># missing teeth (AMTL)</th>
<th>% AMTL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pierhead</td>
<td>21+</td>
<td>male</td>
<td>16</td>
<td>13</td>
<td>81.2%</td>
</tr>
<tr>
<td>30 BU 89</td>
<td>30-50</td>
<td>female</td>
<td>21</td>
<td>6</td>
<td>28.5%</td>
</tr>
<tr>
<td>30 BU 85</td>
<td>25-35</td>
<td>female</td>
<td>32</td>
<td>4</td>
<td>12.5%</td>
</tr>
<tr>
<td>30 BU 61</td>
<td>35-40</td>
<td>female</td>
<td>9</td>
<td>4</td>
<td>44.4%</td>
</tr>
<tr>
<td>30 BU 54</td>
<td>21+</td>
<td>male</td>
<td>16</td>
<td>8</td>
<td>50.0%</td>
</tr>
<tr>
<td>30 BU 56</td>
<td>adult</td>
<td>N/A</td>
<td>5</td>
<td>5</td>
<td>100.0%</td>
</tr>
</tbody>
</table>
Table 5.19 Age distribution, number of individuals and frequency of horizontal alveolar bone loss among the Pierhead/Fontabelle sample

<table>
<thead>
<tr>
<th>Accession #</th>
<th>Age</th>
<th>Sex</th>
<th># of observed tooth sockets</th>
<th># of instances of mod./severe bone loss</th>
<th>% bone loss</th>
<th>Rate of attrition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pierhead</td>
<td>21+</td>
<td>male</td>
<td>16</td>
<td>16</td>
<td>100%</td>
<td>Severe</td>
</tr>
<tr>
<td>30 BU 12</td>
<td>21+</td>
<td>male</td>
<td>3</td>
<td>3</td>
<td>100%</td>
<td>Mod./Severe</td>
</tr>
<tr>
<td>30 BU 54</td>
<td>21+</td>
<td>male</td>
<td>16</td>
<td>16</td>
<td>100%</td>
<td>Severe</td>
</tr>
<tr>
<td>30 BU 89</td>
<td>30-50</td>
<td>female</td>
<td>21</td>
<td>10</td>
<td>47.6%</td>
<td>Slight</td>
</tr>
<tr>
<td>30 BU 147</td>
<td>21+</td>
<td>female</td>
<td>13</td>
<td>10</td>
<td>76.0%</td>
<td>Moderate</td>
</tr>
<tr>
<td>30 BU 61</td>
<td>35-45</td>
<td>female</td>
<td>16</td>
<td>16</td>
<td>100%</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Hypercementosis

Hypercementosis was noted in 3 relatively complete individuals comprising 30% of the Pierhead/Fontabelle sample and 9 of the 100 teeth (Tables 5.1-5.2).

Hypercementosis was exhibited in 6 out of 175 isolated teeth. The teeth most affected by hypercementosis were the maxillary and mandibular molars. All teeth affected by hypercementosis were noted as exhibiting severe hypercementosis where complete root fusion of the tooth roots occurred. Out of 13 instances of hypercementosis, 9 molars exhibited root fusion affecting 69.2% of all observed instances of hypercementosis. In addition, 2 premolars, 1 canine, and 1 lateral incisor demonstrated hypercementosis. Hypercementosis was seen in teeth with slight or no attrition to severe.
Cultural Modifications

Unintentional tooth modifications were noted in the overall dental inventory of all isolated dentition, and of relatively complete individuals. The unintentional tooth modifications exhibited in the Pierhead/Fontabelle sample consisted of pipe-stem facets in the maxillary and mandibular canines and incisors. A total of 3% of the total sample exhibited pipe-stem facets in the maxillary and mandibular canines and incisors (5 teeth affected out of 175) (Table 5.2). Two out of the 3 individuals exhibiting pipe-stem facets were females; 30 BU 94, and 30 BU 61. The male; 30 BU 54 exhibited pipe-stem facets on the mesial side of RI1 and the distal side of the adjacent RC. The facets formed a semi-circle between the two sides of the adjacent teeth, demonstrating a half-circle where the top of a pipe would be clenched. Among all of the observed instances of pipe-stem facets, the teeth were very discolored and stained, suggesting the habitual use of tobacco smoking.
Teeth provide valuable information in reconstructing the overall health in archaeological settings. The dentition is one of the most informative parts of excavated human skeletal remains (Alt et al. 1998) as they provide a valuable source of evidence in understanding the biology of past communities as they do not remodel once they are formed as well as exhibiting low susceptibility to post-depositional degradation (Kelley and Larsen 1991). Specifically, anthropologists analyzing dental defects, dental diseases, and unintentional tooth modifications can reveal much about prehistoric and historic population’s dietary intake, general health, and cultural behaviors.

This study analyzed the dentition from two combined populations of enslaved Africans and their descendents from the Pierhead and Fontabelle sections of Bridgetown, Barbados in an attempt to reconstruct the physical hardships and coping capacities experienced by these groups as manifested in the dentition. The Pierhead/Fontabelle sample consisted of 10 adult enslaved Africans who worked within the urban context of West Indian slavery, which vastly differed from those enslaved on plantations (Higman 1984). The analysis was compared with contemporaneous New World enslaved populations consisting of Africans and their descendents. Comparisons were centered on the Newton Plantation, a rural sugar estate from Barbados which was typical of most medium to large scale West Indian plantations (Handler and Lange 1978).

In most cases of dental analysis, the demography of the Pierhead/Fontabelle sample did not allow for comparisons between males and females or between age groups.
Although this may hinder certain interpretations of the data and limit future comparisons, research on other New World enslaved populations have also combined the male and female data due to the fragmentary nature of the human skeletal remains and the limited amount of associated post-cranial remains (Corruccini et al. 1982). Investigators note only small differences of LEH expression between male and female data from archaeological settings (Handler and Corruccini 1983, Lanphear 1990). In addition, the under-representation of males in the sample could compliment the historical literature and demographics that note the heavy proportion of females enslaved as domestics in Bridgetown, Barbados (Higman 1984, Welch 2003).

Enamel hypoplasia frequencies in the form of linear enamel hypoplasia (LEH) are low for the Pierhead/Fontabelle sample. The low frequencies are surprising when compared with other populations of New World enslaved Africans from rural contexts. The largest difference of LEH occurrence is demonstrated in comparisons with the Catoctin Furnace and Clifts Plantaton Sites of Virginia and Maryland, which reported LEH frequencies occurring in 89% of the population sampled (Blakey et al. 1994). When LEH frequencies are compared with the Newton Plantation, Corruccini et al. (1982) report 54.6% of the population exhibiting LEH in the anterior teeth. However, when they combined the anterior and posterior dentition, they note that almost 98% of individuals exhibited some form of enamel hypoplasia (Handler and Corruccini 1983).

Some caution must be warranted as the origins of birth are not known for the specific individuals expressing LEH in Pierhead/Fontabelle population. Blakey (1998) reports relatively low frequencies of LEH occurrence from the enslaved Africans from the New York African Burial Ground compared with research on other New World
enslaved populations and suggests that the majority of the enslaved in New York were African born. Blakey (1998) further notes that the individuals expressing LEH were most likely those born in New York during the slavery period and the individuals not expressing LEH were most likely African born. Blakey (1998) also suggests that African societies provided a higher physical quality of life compared with American slavery.

One explanation for the low frequencies of LEH in this study may be the small sample size for this population. However, these low frequencies might also infer that the enslaved population in Bridgetown experienced fewer episodes of nutritional and disease stress during their formative years of development, or were able to cope with stress differently than other New World enslaved populations. An important issue to address is the place of origin for Bridgetown's enslaved population. While Higman (1984) asserts that the majority of urban enslaved in the Caribbean were African born, the population of African born enslaved in the British Caribbean had greatly declined by 1807.

The majority of Bridgetown's female enslavers of European descent generally owned fewer African born enslaved (Higman 1984). If the individuals from the Pierhead/Fontabelle population were African born, then the frequency of LEH would suggest that West African populations were more highly stressed compared with the enslaved from Bridgetown, Barbados. Further research addressing LEH frequencies of New World enslaved populations should use DNA analysis to determine place of birth as well as address LEH frequencies of contemporaneous West African populations who did not experience slavery in the British Caribbean.

When assessing the peak age at occurrence for LEH, the Pierhead/Fontabelle sample was most similar to the peak age at occurrence for the Newton Plantation. The
peak age at occurrence for the Pierhead/Fontabelle population occurred between approximately 3 to 4 years of age. This time coincides with Corruccini et al. (1985), where the central age tendency at formation of hypoplasias is 3-4 years. Six other populations ranging in time from the Middle Mississippian Period to the 20th c. were chosen for comparisons with the Pierhead/Fontabelle population. These were chosen as they represent different modes of subsistence and exhibit a wide time range. The twentieth century sample demonstrates an earlier age at occurrence between 1-3 years, and the Terminal Classic Maya Period has the latest age at occurrence at 3-6 years (based on the third premolar). The rural plantation and urban enslaved samples overlap with each other in age at occurrence of LEH. These sites demonstrate a later age at occurrence compared with most non-industrial populations which show a hypoplasia peak at 2-3 years (Corruccini et al. 1985).

The combination of peak age at stress and LEH frequencies from the Pierhead/Fontabelle sample may suggest that the negative factors associated with weaning stress such as malnutrition and disease were responsible for causing LEH. Most of the Pierhead/Fontabelle teeth exhibiting LEH only contained one stress episode per tooth with the majority of stress episodes occurring between 3 and 4 years. In Barbados, planters reported weaning practices typically occurring around 24 months (Higman 1984). Initial assessment of LEH peak age at occurrence on the Newton Plantation enslaved also demonstrated the same age at occurrence with most teeth exhibiting LEH with only one stress episode (Corruccini et al. 1985). However, later investigation of the Newton Plantation population found 3 individuals with characteristics in the dentition directly associated with syphilis who also expressed multiple episodes of LEH (Jacobi et
al. 1992). This suggests that the cause of LEH in the 3 individuals could be a direct result of congenital syphilis (Jacobi et al. 1992) and therefore the association between syphilis and LEH cannot be ruled out in the Pierhead/Fontabelle sample. In sum, the consistent age at stress occurrence and single episodes of occurrence per tooth is likely related to weaning stress although congenital syphilis could also contribute to LEH formation among the Pierhead/Fontabelle sample.

Carious lesions were present among 50% of the relatively complete individuals from the Pierhead/Fontabelle sample. When carious lesions were counted for the total sample, 27% of the teeth contained lesions. No males from the Pierhead/Fontabelle sample exhibited carious lesions. This is not surprising as all of the known males exhibited severe rates of attrition and high frequencies of antemortem tooth loss. The moderate frequency of carious lesions among the Pierhead/Fontabelle individuals differs from the low frequency of carious lesions expressed in the Newton Plantation. Corruccini et al. (1982) report occlusal carious lesions in 20% of the sample. However, they report much higher frequencies of interproximal decay at 57%. Interproximal decay increases with age and periodontal disease (Buikstra and Ubelaker 1994) and was minimal within the Pierhead/Fontabelle sample.

The differences in the types and locations of lesions within the Pierhead/Fontabelle sample compared with the Newton Plantation may have implications in diet and overall dental health of the population. The relatively low presence of occlusal carious lesions in the Newton Plantation sample may infer that while they consumed a diet high in sugars and carbohydrates they may have consumed enough raw vegetable matter to have kept their teeth relatively clean (Handler and Corruccini 1983). The higher
rates in the Pierhead/Fontabelle sample might be attributed to consumption of less raw vegetable matter and coarse foods that could keep the teeth clean. In addition, the low rates of interproximal carious lesions and root surface lesions found in the Pierhead/Fontabelle may suggest lower incidences of progressive periodontal disease compared with the Newton Plantation, although alveolar resorption and remodeling frequencies were high for the Pierhead/Fontabelle individuals.

Antemortem tooth loss was exhibited in 60% of the Pierhead/Fontabelle sample affecting both males and females. Between 12.5% to 81.2% of all teeth of relatively complete individuals were reported missing antemortem. Corruccini et al. (1982) also reported high frequencies of non-random antemortem tooth loss in the Newton Plantation and attributed it to human intervention as the enslaved on the Newton Plantation often reported tooth aches (Handler and Lange 1978). The majority of antemortem tooth loss in the Pierhead/Fontabelle sample occurred primarily in the mandibular molars, often occurring in the same molars of the opposite dental arcade. This would appear to be a non-random pattern, however, the under representation of maxillae among the Pierhead/Fontabelle sample should be noted. If antemortem tooth loss is to be attributed to carious lesions, the Pierhead/Fontabelle enslaved would have extracted their teeth intentionally to alleviate pain. However, few periapical abscesses were found in association with antemortem tooth loss among the Pierhead/Fontabelle sample suggesting antemortem tooth loss could be attributed to periodontal disease exhibited through horizontal alveolar bone loss.

Recording periodontal disease is difficult in prehistoric populations as only the more severe forms will leave traces in dry bone (Molnar and Molnar 1985). Only when
gingival inflammation involves the deeper layers and modifies the alveolar crest can periodontitis be recorded (Molnar and Molnar 1985). Clarke and Hirsch (1991) challenge the relationship of periodontitis and alveolar bone loss and attribute bone loss to other factors, including severe attrition, although Costa (1982) states that periodontal disease and carious lesions represent the majority of tooth loss in the United States.

Alveolar resorption was common among the Pierhead/Fontabelle sample. Sixty percent of the Pierhead/Fontabelle individuals exhibited alveolar bone loss of moderate to severe loss. Alveolar resorption affected both males and females equally and appears to be age progressive in the females. Since the age at death of males could not be aged to a more specific time, correlations between age and periodontal disease for the males cannot be made. However, there appears to be a correlation with severity of attrition and the percentage of horizontal alveolar bone loss in the males. This correlation between attrition and alveolar resorption among the Pierhead/Fontabelle males could suggest that attrition was leading to the eventual loss of teeth antemortem. Closer examination of alveolar resorption and antemortem tooth loss suggests that it might be closely related to periodontal disease. Antemortem tooth loss and alveolar resorption do not find a strong correlation as the individuals most affected by alveolar resorption did not exhibit high rates of antemortem tooth loss.

Hypercementosis was found in 30% of the individuals and was exhibited in 9% of the total sample. This may be an underestimation as hypercementosis is exhibited in exposed tooth roots through alveolar bone loss and in isolated dentition. The severity of hypercementosis was similar with the severity found on the Newton Plantation. Corruccini et al. (1982) note hypercementosis associated with clenching, physical trauma,
or bruxism. Corruccini et al. (1982) attribute hypercementosis to periodontal degeneration and malnutrition, as bruxism causes tooth movement which was not observed in the Newton Plantation, and is always observed in the molar region. According to Corrucinni et al. (1987) hypercementosis of enslaved Africans was most likely related to infectious gum diseases and deterioration (periodontal degeneration), or malnutrition. Although hypercementosis is often associated with advanced periodontal disease, malocclusion, and excessive wear (Corrucinni et al. 1987), Hillson (1996) states that the cause is unknown as it also occurs in unerupted teeth. It has also been thought to occur from Vitamin C deficiency (Corrucinni et al. 1987).

Finally, cultural modifications examined in this project in the form of pipe-stem facets were exhibited in the Pierhead/Fontabelle sample. These findings are similar to the findings at the Newton Plantation where both sexes were attributed for evidence of pipe-stem facets. Among the 5 burials associated with pipe-stem facets, 2 were males, 1 was a female, and 1 was not assigned a sex. Handler and Lange (1978) also found instances of pipe-stem wear in the dentition of the enslaved from the Newton Plantation. They report 20 cases of pipe-stem wear in the canines, incisors, and the premolars. In addition, Handler and Lange (1978) found whole pipes in 17 burials with 5 individuals showing indications of pipe wear.

The dentition recovered from the Pierhead and Fontabelle populations exhibited dental defects, dental disease, and cultural modifications exhibited in the Newton Plantation and typically seen other New World enslaved populations. However, the frequencies of the dental defects as evidenced by linear enamel hypoplasias differed between the comparison populations. The Pierhead/Fontabelle sample exhibited low
rates of LEH, moderate amounts of occlusal carious lesions, and high rates of periodontitis as demonstrated by antemortem tooth loss and alveolar bone loss. They exhibited low rates of hypercementosis but the severity was comparable to the Newton Plantation. In addition, the Pierhead and Fontabelle sample exhibited cultural modifications in the dentition similar to what Handler and Lange (1978) found in the Newton Plantation sample.

The dental analysis of the human skeletal remains from the Fontabelle and Pierhead burial grounds in Bridgetown corroborate many of the findings that Handler and Corruccini (1983) found at the Newton Plantation regarding overall health and lifestyle behaviors experienced by New World enslaved populations. The main difference lies in the low frequency of enamel hypoplasias exhibited in the Pierhead/Fontabelle sample suggesting they were not as highly stressed as other enslaved Africans in the New World.

The relationship between enamel and childhood morbidity is exhibited in the dentition from the Pierhead and Fontabelle sections of Bridgetown, Barbados. It is possible that the enslaved in the urban context of Bridgetown were better able to meet their caloric needs compared with those enslaved at the Newton Plantation. The variations in demographics in the urban context of slavery may have also contributed to the health of the enslaved peoples of Bridgetown. The enslaved at Bridgetown generally had better housing, clothing, and access to a wider variety of food (Welch 2003) which may contribute to better health of the urban enslaved from Bridgetown.

However, the high frequencies of periodontitis and moderate rates of carious lesions could be the result of poor oral hygiene and lack of dentistry among the enslaved. This project examined and analyzed the dentition of enslaved African’s from Bridgetown
and how their differences in lifestyles compared with those enslaved in the rural context may be reflected in their dentition. The analysis of this study will prove useful in reconstructing the hardships, coping capacities, and lifestyle behaviors of the urban enslaved as osteological evidence is lacking.
REFERENCES


Farmer, K., Smith, F., Watson, K. The Urban Context of Slavery: An Archaeological Perspective from Two Afro-Barbadian Slave Cemeteries in Bridgetown, Barbados. (forthcoming)


Appendix A

Form Used for Recording Dental Measurements and Morphology
DENTAL MEASUREMENTS AND MORPHOLOGY RECORDING FORM

Site Name/Number ________________________ / ________________________ Observer

Feature/Burial Number ________________________ / ________________________ Date

Burial/Skeleton Number ________________________ / ________________________

Present Location of Collection

Maxilla

<table>
<thead>
<tr>
<th>Tooth</th>
<th>$I^1$</th>
<th>$I^2$</th>
<th>C</th>
<th>$PM^1$</th>
<th>$PM^2$</th>
<th>$M^1$</th>
<th>$M^2$</th>
<th>$M^3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesiodistal Diameter</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buccolingual Diameter</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crown Height</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mandible

<table>
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<tr>
<th>Tooth</th>
<th>$I_1$</th>
<th>$I_2$</th>
<th>C</th>
<th>$PM_1$</th>
<th>$PM_2$</th>
<th>$M_1$</th>
<th>$M_2$</th>
<th>$M_3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesiodistal Diameter</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buccolingual Diameter</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crown Height</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(after Buikstra and Ubelaker, 1994:Chapter 6: Attachment 20)
Appendix B

Form Used for Visual Recording of Permanent Dentition
DENTAL INVENTORY
VISUAL RECORDING FORM: PERMANENT DENTITION

Site Name/Number / Observer

Feature/Burial Number / Date

Burial/Skeleton Number

Present Location of Collection

MAXILLARY

BUCCAL

BUCCAL

MANDIBULAR

(after Buikstra and Ubelaker, 1994:attachment 14a:CHAPTER 5)
Appendix C

Form Used for Dental Inventory
DENTAL INVENTORY RECORDING FORM
DEVELOPMENT, WEAR, AND PATHOLOGY: PERMANENT DENTITION

<table>
<thead>
<tr>
<th>Site Name/Number</th>
<th>Observer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feature/Burial Number</td>
<td>Date</td>
</tr>
<tr>
<td>Burial/Skeleton Number</td>
<td></td>
</tr>
<tr>
<td>Present Location of Collection</td>
<td></td>
</tr>
</tbody>
</table>

**Tooth presence and development:** code 1-8. For teeth entered as "I" (present but not in occlusion), record the stage of crown/root formation under "Development." **Occlusal surface wear:** use left dentition, for anterior teeth (code 1-8) and for molars (code 0-10). Record both sides for marked asymmetry. Record each molar quadrant separate in the spaces provided (+) and the total for all four quadrants under "Total." **Caries:** code each carious lesion separately (1-7); **Abscesses:** code location (1-2). **Calculus:** code 0-3, 9.

Note surface affected (buccal/labial or lingual).

<table>
<thead>
<tr>
<th>Tooth Presence</th>
<th>Development</th>
<th>Wear/Total</th>
<th>Caries</th>
<th>Abscesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 M₁ &lt;sup&gt;3&lt;/sup&gt;</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>2 M₂</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>3 M₁</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>4 P₁</td>
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<td>---</td>
</tr>
<tr>
<td>5 P₁</td>
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<td>---</td>
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<tr>
<td>6 C</td>
<td>---</td>
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<td>---</td>
</tr>
<tr>
<td>7 P₂</td>
<td>---</td>
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<td>---</td>
</tr>
<tr>
<td>8 P₂</td>
<td>---</td>
<td>/</td>
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<td>---</td>
</tr>
<tr>
<td>9 P₂</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>10 P₂</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>11 C</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
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<tr>
<td>12 C</td>
<td>---</td>
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<tr>
<td>13 P₄</td>
<td>---</td>
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<td>---</td>
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<tr>
<td>14 M₃</td>
<td>---</td>
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<td>---</td>
</tr>
<tr>
<td>15 M₇</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>16 M₇</td>
<td>---</td>
<td>/</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Tooth</td>
<td>Presence</td>
<td>Development</td>
<td>Wear/Total</td>
<td>Caries</td>
</tr>
<tr>
<td>-------</td>
<td>----------</td>
<td>-------------</td>
<td>------------</td>
<td>--------</td>
</tr>
<tr>
<td>17 M3</td>
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<td></td>
</tr>
<tr>
<td>18 M2</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>19 M1</td>
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</tr>
<tr>
<td>21 P2</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22 C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23 I2</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>24 I1</td>
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<td></td>
</tr>
<tr>
<td>25 I1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26 I2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27 C</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>28 P1</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>29 P2</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>30 M1</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>31 M2</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>32 M3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Estimated dental age (juveniles only)**

<table>
<thead>
<tr>
<th>Position between teeth</th>
<th>Location (1-4)</th>
<th>Position between teeth</th>
<th>Location (1-4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

**Comments:**

_________________________________________________________________
_________________________________________________________________
_________________________________________________________________
_________________________________________________________________

(after Buikstra and Ubelaker, 1994: attachment 16:CHAPTER 5)
Appendix D

Form Used for Recording Supernumerary Teeth and Abscesses for the Maxillary Dentition
SUPERNUMERARY TEETH AND ABSCESSES
VISUAL RECORDING FORM: MAXILLARY DENTITION

Site Name/Number / Observer

Feature/Burial Number / Date

Burial/Skeleton Number / 

Present Location of Collection

Right Buccal View

Left Buccal View

Palatal View

(after Buikstra and Ubelaker, 1994: attachment 15a:CHAPTER 5)
Appendix E

Form Used for Recording Supernumerary Teeth and Abscesses for the Mandibular Dentition
SUPERNUMERARY TEETH AND ABSCESSES
VISUAL RECORDING FORM: MANDIBULAR DENTITION

Site Name/Number / Observer

Feature/Burial Number / Date

Burial/Skeleton Number

Present Location of Collection

Frontal Labial/Buccal View

Right Lingual View

Left Lingual View

(after Buikstra and Ubelaker, 1994:attachment 15b:CHAPTER 5)
Appendix F

Form Used for Recording Cultural Modifications
PREMORTEM DENTAL MODIFICATIONS
RECORDING FORM

Site Name/Number / Observer

Feature/Burial Number / Date

Burial/Skeleton Number /

Present Location of Collection

Type of Modification: 
1. Aesthetic Modification: Filing
2. Aesthetic Modification: Drilling
3. Dental Restorations and Appliances
4. Dental Wear Associated with Artifact Production and/or Use
5. Tooth Ablation

Comments:

(after Buikstra and Ubelaker, 1994:attachment 19:CHAPTER 5)
Appendix G

Form Used for Rapid Recording of Initiation Sites of Carious Lesions
Divide dentition into permanent anterior, and permanent posterior teeth. For each division, record counts of sites present and sites carious as follows:

<table>
<thead>
<tr>
<th>Sites</th>
<th>Present</th>
<th>Carious</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fissure and pit sites</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Approximal Sites</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cervical Sites</td>
<td></td>
<td>enamel caries</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CEJ caries</td>
</tr>
<tr>
<td></td>
<td></td>
<td>root surface caries</td>
</tr>
<tr>
<td></td>
<td></td>
<td>gross cervical caries</td>
</tr>
<tr>
<td>Tooth Count</td>
<td></td>
<td>gross crown caries</td>
</tr>
<tr>
<td></td>
<td></td>
<td>gross gross caries</td>
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</table>

Lesions are expressed as percentages of sites present. Count 1 fissure & pit per cheek tooth, if any vestige of fissure system present. Count 2 approximal sites (mesial and distal) per tooth and 1 for most distal tooth in quadrant. Count 1 cervical site per tooth, if any vestige of CEJ is present.

Appendix H

Form Used to Record Enamel Defects
ENAMEL DEFECTS (HYPOPLASIAS AND OPACITIES)  
RECORDING FORM

Site Name/Number / Observer

Feature/Burial Number / Date

Burial/Skeleton Number

Present Location of Collection

Type: code 0-7; Location: Measure distance from CEJ to most occlusal portion of defect; Color: code 1-4 For hypocalcifications (type 6 or 7) only.

Maxilla, Right

<table>
<thead>
<tr>
<th>Tooth</th>
<th>M(^3)</th>
<th>M(^2)</th>
<th>M(^1)</th>
<th>PM(^2)</th>
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(after Buikstra and Ubelaker, 1994: CHAPTER 5: Attachment 18a)
Appendix I

Codes for Types of Enamel Defects and Opacities
Codes for Recording Enamel Defects and Hypocalcifications (opacities)

Enamel Hypoplasias are represented from numbers 1-5; 6-7 reflect characteristics of hypocalcified enamel:

0. Absence
1. Linear horizontal grooves
2. Linear vertical grooves
3. Nonlinear horizontal pits
4. Nonlinear arrays of pits
5. Single pits
6. Discrete boundary opacity
7. Diffuse boundary opacity

Hypocalcification Codes:

1. Yellow
2. Cream/white
3. Orange
4. Brown

(after Buikstra and Ubelaker, 1994: 56)
Appendix J

Chart Used to Delineate Chronology of LEH of Anterior Maxillary and Mandibular Dentition
Timing of anterior tooth growth. Representation of right anterior quadrant of mandibular and maxillary dentition is depicted. Each height of each tooth type is divided into deciles (10 equally spaced zones). The age of appearance of the enamel at the incisal edge is considered coincident with the completion of cuspal enamel. (after Reid and Dean, 2000:138).
Appendix K

Letters of Permission
June 12, 2006

Christopher Crain
1240 East 22 Road
Manton, MI 49663

Dear Mr. Crain,

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Picture # P 7190026

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[Signature]  [Name]
[Date]  [Date]

Please return this letter in the self-addressed, stamped envelope provided. Thank you for your time and attention to this matter.

Sincerely,

Jennifer Yamazaki
3801 Winchell Ave #112
Kalamazoo, MI 49008
June 12, 2006

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Manton, MI 49663

Dear Mr. Crain,

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[Signature]

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Date

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Sincerely,

Jennifer Yamazaki
3801 Winchell Ave #112
Kalamazoo, MI 49008
June 12, 2006

Christopher Crain
1240 East 22 Road
Manton, MI 49663

Dear Mr. Crain,

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June 12, 2006

Christopher Crain
1240 East 22 Road
Manton, MI 49663

Dear Mr. Crain,

I would like to request your permission to reproduce a picture to include in my thesis:

Picture # P 7130082

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