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Microscopic Enamel Defects in a Contemporary Population: Biological and Social Implications

Lise Marie Mifsud
University of Tennessee, Knoxville

To the Graduate Council:

I am submitting herewith a thesis written by Lise Marie Mifsud entitled "Microscopic Enamel Defects in a Contemporary Population: Biological and Social Implications." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Anthropology.

Murray K. Marks, Major Professor

We have read this thesis and recommend its acceptance:

Walter E. Klippel, Lyle Konigsberg, Mike Elam

Accepted for the Council:
Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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Microscopic Enamel Defects In A Contemporary Population: Biological and Social Implications

A Thesis

Presented for the

Master of Arts

Degree

The University of Tennessee, Knoxville

Lise Marie Mifsud

August 1998

Dedication

This thesis is dedicated to my parents

Mr. Jerry Mifsud

and

Ms. Jeanette Monterio

and my sister

Meshelle A. Mifsud

Without their loving support this thesis would not have been possible.

and my fiancé

Robert Brown

Who knew that the years we spent apart would only make us stronger. Now it is our time to be together.

Acknowledgments

There are so many individuals who gave their professional and personal time towards the completion of this thesis. Collecting my sample would not have been possible without the help of the staff from the following oral surgeons offices: Dr. Hollander, Dr. Stultz, Dr. Conroy, Dr. Wilson, Dr. Limchayseng, Discount Denture Service of Knoxville, Marin County Dental Services Clinic, Knox County Public Health Clinic, and Memphis Public Health Clinic. Assistance with acquiring the digital images was provided by Dr. David Cole and Dr. Larry Anovitz at Oak Ridge National Laboratories. The digital images were processed into a finished product by Rob Brown. The students in the Anthropology department at the University of Tennessee were always there to offer words of encouragement and valuable suggestions.

The members of my committee deserve special mention, for each of them contributed to this work. Dr. Mike Elam introduced me to the possibilities of technology and made the digital images a reality. Dr. Lyle Konigsberg provided assistance with the statistics. He was always available to answer questions, and did so in a way that was not belittling. Any mistakes the reader finds in said statistics are the sole responsibility of the author. Dr. Walter Klippel had the uncanny ability to ask the pertinent questions, which gave direction to my research focus. I appreciate all of the forehead rubbing he did on my behalf.

Finally, I would like to thank the chair of my committee Dr. Murray Marks whose previous research on this topic made this work possible. He provided laboratory space, supplies, training, advice, support, and encouragement. I thank him for his enthusiasm and for instilling in me an interest in things not readily visible on the surface.

Abstract

In this study, the frequency of microscopic defects in enamel, termed Wilson bands, are analyzed according to socioeconomic affiliation, the sex, and ethnicity of the individual. The sample consists of 193 anterior teeth collected from private practice and public health oral surgeon's offices. These defects have been studied in great detail by dental researchers, dental anthropologists, and bioarchaeologists to ascertain: etiology, morphology, inter- and intra- population differences, prehistoric diet and health, and dietary and morbidity conditions of underprivileged contemporary populations.

The results of this study are compared to results of previous researchers and similarities and differences in findings are discussed. Of particular interest is the applicability of Wilson bands to the stresses incurred during the weaning process, and the relationship of socioeconomic affiliation to the occurrence of Wilson bands. The social ramifications of these findings are discussed.

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Chapter 1

Introduction

Use of developmental defects in tooth enamel to infer nutritional imbalance and other metabolic conditions has been readily employed by anthropologists and dental researchers for several decades (see Cook and Buikstra, 1979; Goodman et al., 1980; Pindborg, 1970; Rose, 1973; Sarnat and Schour, 1941; Sarnat and Schour 1942). The high mineral content and resulting permanence of enamel make for excellent preservation of the teeth, as well as the insult recorded by the enamel defect. The dentition records interactions between an individual and the physical and biological environments. These interactions are recorded both microscopically (in the form of Wilson bands) (after Wilson and Schroff, 1970) and macroscopically (in the form of enamel hypoplasias) (after Kreshover, 1940; Massler et al., 1941; Sarnat and Schour, 1941; Sarnat and Schour, 1942) in the mineralized tissue, enamel.

These defects have been studied in great detail by dental researchers (Kreshover, 1940; Massler et al., 1941; Osborn, 1990; Pindborg, 1970), dental anthropologists (Goodman et al., 1984; Hillson and Bond, 1997; Lukacs and Joshi, 1992; Marks, 1993), and bioarchaeologists (Cook and Buikstra, 1979; Goodman and Rose, 1990; Rose, 1973; Skinner and Goodman, 1992) to ascertain: etiology, morphology, population differences, prehistoric diet and health conditions, and dietary and health conditions of underprivileged, living populations (Goodman et al., 1987; Goodman et al., 1992; Goodman et al., 1992; Infante and Gillespie 1974a, 1974b, 1976, 1977; Sweeney et al.,

1971). Despite the vast amount of research conducted, none have addressed the frequency of microscopic enamel defects as they appear in a random sample of modern adults. The subsequent research focus will attempt to address this dearth of information through the analysis of extracted teeth collected from oral surgeons located in Tennessee and California. Information recorded with each tooth includes the patient's: age, sex, ethnicity, and socioeconomic affiliation. These variables will be utilized to determine any correlation between the variables and the presence, absence, and numeration of microscopic enamel defects.

Chapter 2

Developmental Dental Anatomy

Enamel Formation (Amelogenesis)

In order to ascertain what pathophysiological pathways result in enamel defects, it is necessary to first understand normal dental histology and anatomy. Enamel is an ectodermal tissue, which constitutes the anatomical crown of the tooth or that portion of the tooth exposed in the oral cavity. It is highly mineralized, consisting of 96 percent inorganic material in the form of hydroxyapatite; and 4 percent water and the organic protein, enamelin (Avery 1992:85). Enamel must resist the masticatory forces that are placed on it. This is accomplished with the assistance of healthy, shock absorbing, underlying dentin (constituting the bulk of the crown and root) and the periodontal complex.

Several authors describe the formation of enamel and the subsequent discussion is drawn from the following sources: Avery 1992; Goodman and Rose 1991; Jordan 1992; Marks 1993; Rose et al. 1985; Scott 1974; Ten Cate 1995.

Enamel formation begins during the sixth month, *in utero,* when the inner enamel epithelium maps out the future form of the crown of the tooth. Enamel organs start to form after a few micrometers of dentin have been deposited by odontoblasts at the dentin-enamel junction (DEJ) (as the term implies, the border between dentin and enamel; see figure 1). This process begins at the cusp tips and proceeds towards the cervical neck.

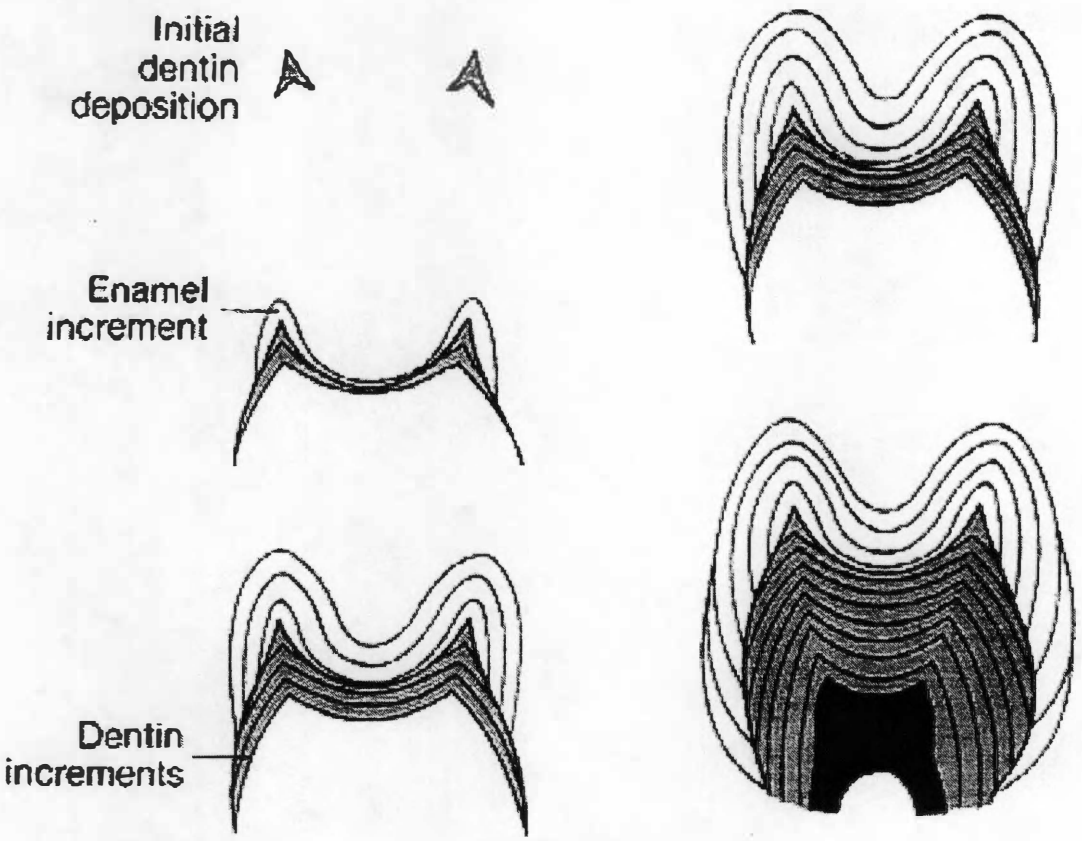

Figure 1. The Deposition of Enamel and Dentin

Source: Avery JK (1992) Essentials of Oral Histology and Embryology. Mosby. Saint Louis, MO.

Ameloblasts (enamel producing cells) differentiate and pass through the following functional stages: morphogenesis, organization and differentiation, secretion, maturation and protection. The morphogenetic stage begins during the bell and crown stages of tooth formation. Cells of the dental organ and dental papilla interact by differential growth to form the shape of the tooth crown. Cells of the inner enamel epithelium are cuboidal with large, centrally located nuclei. Mitochondria and other cytoplasmic components consistent with protein synthesis activities are scattered throughout the cells.

The differentiation stage occurs when the cells of the inner enamel epithelium differentiate into ameloblasts (figure 2). The cells elongate and their nuclei shift proximally towards the stratum intermedium. The Golgi complex increases in volume and migrates from its proximal position to occupy a major portion of the central core of the cell. The amount of rough endoplasmic reticulum increases and mitochondria cluster in the proximal region, although some still remain scattered throughout the cell. The ameloblast becomes a highly polarized cell. Adjacent ameloblasts are closely aligned to each other and this alignment is maintained by the development of attachment specialization at their proximal and distal ends, called desmosomes. The desmosomes hold the cells in a row as they move peripherally from the dentin-enamel junction, depositing enamel matrix.

At the instant enamel matrix is deposited, the secretory phase commences with the synthesis of organic matrix deposited in increments. When ameloblasts begin secretion, the overlying cells of the stratum intermedium enlarge and change shape from spindle to

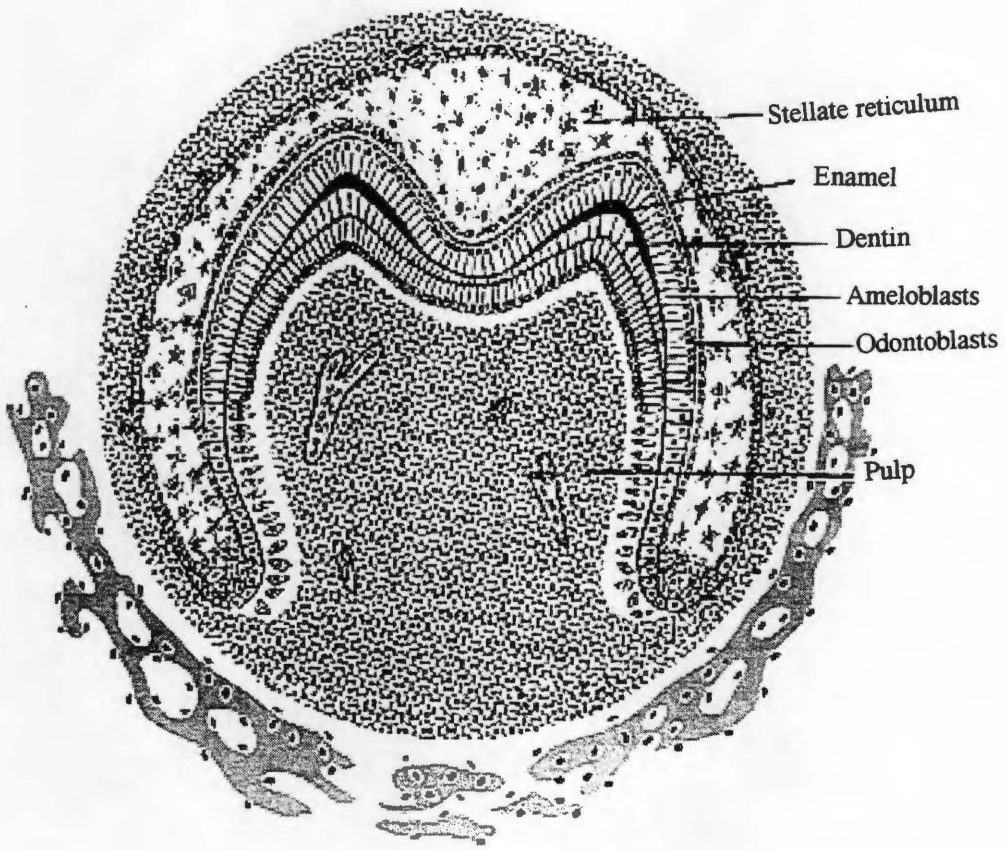

Figure 2. The Late Bell Stage of Enamel Formation in a Multicusped Tooth.

Source: Avery JK (1992) Essentials of Oral Histology and Embryology. Mosby. St. Louis, MO.

pyramidal. Short conical extensions, called Tome's processes, develop at the apical end of the ameloblast during this stage. The terminal bar apparatus appears at the junction of the cell bodies and the Tome's process. Their function is to maintain contact between adjacent cells.

Once the amelogin is deposited, the matrix begins to mature and mineralize, commencing the maturation stage. The protein of mature enamel is termed enamelin. Small crystals of mineral grow in length and diameter rapidly. The first matrix deposited along the dentin-enamel junction is the first to mineralize. This process continues first at the cusp tip, moves laterally onto the crown, and finally to the cervical region of the crown. Due to the high mineral content of enamel, almost all water and organic material are lost from it during maturation (a mere four percent remains when maturation is complete). As this phase nears completion, the terminal bar apparatus disappears, leaving a smooth enamel surface.

The protective stage commences when enamel maturation nears completion. The ameloblasts lose their striated borders and secrete an organic cuticle between the flattened distal end of the cell and the enamel surface, called a hemidesmosome. This serves to protect the ectodermal surface from surrounding mesoderm and provides an ectodermal sheath for guiding tooth eruption.

Morphology of Enamel

Enamel is composed of interlocking rods, which resist masticatory forces. These rods are deposited in a keyhole shape by the formative ameloblastic cells. The number of ameloblasts contributing to each enamel rod, or prism, varies depending on the researcher consulted. Avery (1992:85) states that four ameloblasts contribute to one enamel prism. Ten Cate (1989) states the number of ameloblasts can vary from one to six. The number of rods per tooth crown varies from 5 million in the mandibular lateral incisor, to 12 million in the maxillary first molar (Skobie and Stern, 1980).

Each rod is filled with apatite crystals and consists of a head and a tail, which interdigitate with the neighboring enamel rod to the right and left (figure 3). At the central axis, crystals in the head follow the long axis of the rod; and crystals in the tail, follow the direction of its long axis (Avery,1992). However, crystals which are further away from the central axis, veer laterally as they approach the rod boundary (Ten Cate, 1985). The different directions of the crystals (which are not always continuous with one another) result in enamel structure which is not perfectly divided into prisms with defined boundaries (Boyde, 1976).

The enamel rods, or prisms, take the form of three distinct prism patterns (Boyde, 1976; Hillson, 1996; Osborn, 1973). Pattern 1 enamel has a simple organization, which occurs when the plane of the developing surface is parallel to the DEJ and there is no artifact of movement of the ameloblasts across the surface they are creating. This enamel pattern is uncommon. The prisms in Pattern 2 are placed one above the other in vertical

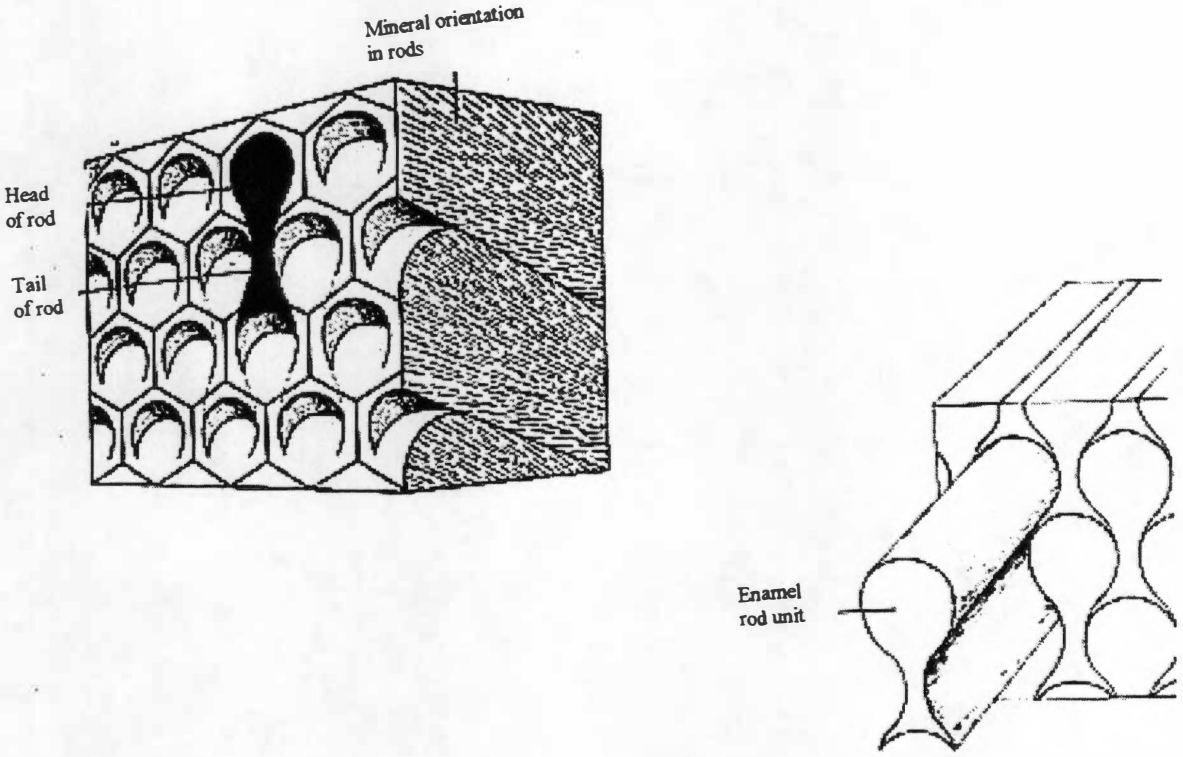

Figure 3. The Morphology of Enamel Rods.
The Figure on the Left is an Illustration of How Rods Interact with Each Other.
The Figure on the Right is an Illustration of One Rod Unit.
Source: Avery JK (1992) Essentials of Oral Histology and Embryology. Mosby. St. Louis, MO.

rows, with inter-prismatic sheets of enamel between them. This prism pattern can occur locally in human enamel, but are rarer than pattern 1. Pattern 3 prisms are the most common in human enamel. In this pattern, each prism has a projection of inter-prismatic enamel attached to its cervical border, which interlocks with the two prisms directly below. It is this prism pattern which has the characteristic keyhole shape. Because irregularities are often found, Gantt (1982) suggested that there are two variants for Pattern 3.

Groups of rods will bend to the left and to the right at a slightly different angle than the adjacent group of rods. It is possible that this feature provides enamel with strength for mastication and biting (Avery, 1992). If light is projected through a thin-section, the bands will appear alternating light and dark Hunter-Schreger bands, due to the differences in organization and courses taken by the neighboring group of rods (Osborn, 1973, 1990). These alternating bands extend through approximately one-half of the thickness of enamel (Avery, 1992). The dark regions, diazones, represent prisms cut cross-sectionally; while parazones, represent more longitudinally oriented prisms.

At the occlusal surface, where the tooth is exposed to masticatory and bacterial forces, several changes occur. The volume of enamel increases at the cusps, but the number of rods remains constant. One possible explanation for this phenomenon is the increase of diameter and the oblique approach of rods near the tooth surface (Skobe and Stern, 1980). Once the rods reach the occlusal surface of the tooth, they extend in several directions. This gnarled enamel dissipates force and offers protection from fracturing due

to masticatory forces (Marks, 1993). The portion of the occlusal surface (measuring 20 to 40 μ) contains no prisms. This phenomenon is most evident near the cervical region and in deciduous teeth. While viewing prismless enamel in four teeth (3 permanent incisors and 1 canine), Miyoshi and co-workers (1972) came to the unpopular conclusion that an enamel prism is not continuous from the DEJ to the surface.

Incremental Lines

Enamel also contains a system of incremental lines, or striae of Retzius. These normal, microscopic lines are the result of the rhythmic, recurrent deposition by ameloblast secretion and mineralization, in a predictable appositional pattern. The striae occur when the cell is in a resting phase due to a decrease in cell pressure. Osborn (1973) believes that a stria is formed every five days. However, other researchers have found variation in this chronology (see Aiello and Dean, 1990; Osborn and Ten Cate, 1983; Warshawsky et al., 1984).

In a cross-section of enamel, striae of Retzius look similar to the concentric lines that form rings of a tree. At these lines, it is theorized that fewer crystals exist and that the enamel rods change direction, bend slightly, or constrict (Avery, 1992; Osborn, 1971; Wilson and Shroff, 1970). The appearance of the striae of Retzius on the outer surface of the teeth is termed perikymata, or imbrication lines of Pickerill (figure 4).

A portion of the enamel in deciduous and some permanent teeth is formed *in utero*. The remaining portion is formed postpartum. The sudden change in environment

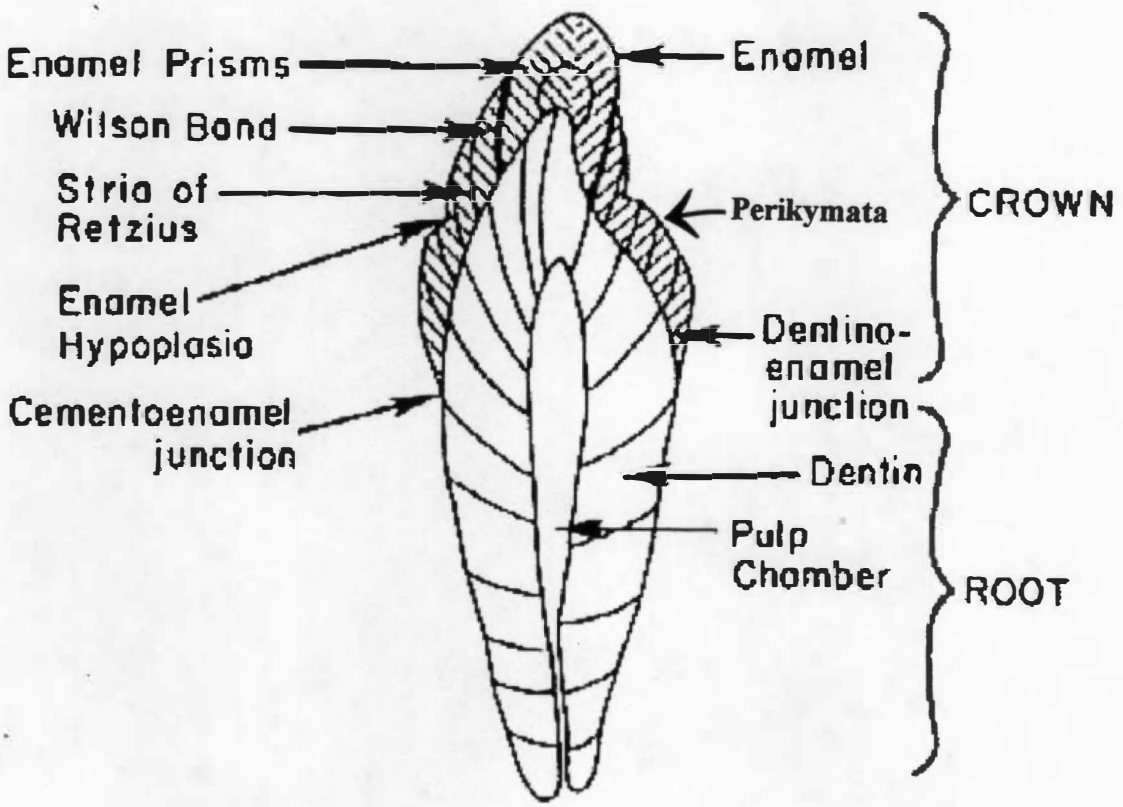

Figure 4. The Relationship Between Microscopic and Macroscopic Components.

Source: Rose et. al (1985) Diet and Dentition: Developmental Disturbances. In RI Gilbert Jr. And JH Meilke (eds.): The Analysis of Prehistoric Diets. Orlando: Academic Press.

and nutrition at birth, results in a more pronounced pathological incremental line called the neonatal line (figure 5). The enamel formed between the neonatal line and the dentin-enamel junction represents prenatal enamel formation. This enamel is whiter, of a better quality, and typically free of defects. The enamel formed from the neonatal line towards the outer surface is formed postnatally and contains a greater amount of defects.

The location and width of the neonatal line is affected by several factors. Skinner (1992) noted that the location of the neonatal line in deciduous teeth differed significantly between all tooth classes. The width of the neonatal line is dependent on the length and difficulty of the birth process (Eli et al., 1989; Skinner, 1992). The neonatal line is wider in infants that experienced a more traumatic birthing.

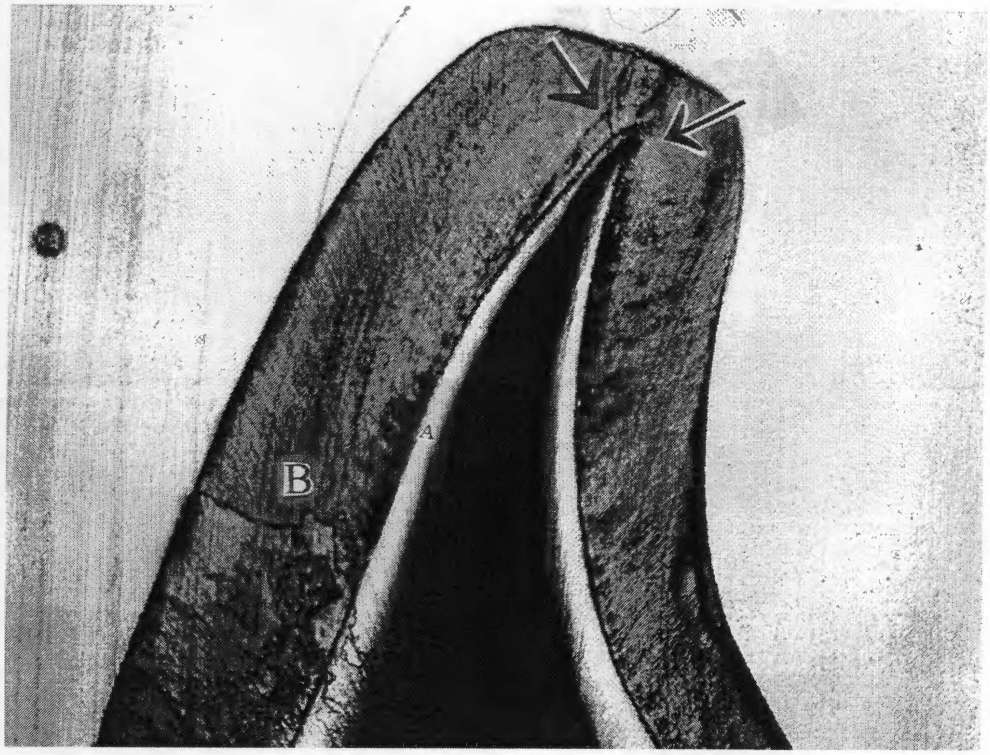

Figure 5. Neonatal Line in a Labio-Lingual Thin Section of a Left Maxillary Lateral Incisor (1.25x). Arrows Designate the Neonatal Line. "A" is the Dentine and "B" is the Enamel.

Chapter 3

Enamel Defects

Morphology of Microscopic Enamel Defects

Because enamel rods are derived from several ameloblasts, which simultaneously secrete and mineralize, mal-mineralized lines appear where any ameloblasts hesitate or are destroyed during enamel formation. However, Suckling (1986) determined that hypo-calcification can occur after a disruption in matrix formation. The resultant abnormal lines establish each successive contour of enamel matrix during its deposition. These hypo- or hyper-mineralized areas occur because the ameloblast is a cell that is particularly sensitive to changes in its environment. If the insult is a temporary, minor, physiological disruption, the direction and chemical composition of the ameloblast may be affected (Kreshover, 1940; Ten Cate, 1985; Rose et al., 1985). A defect of this sort is observable at the microscopic level as a line of enamel prisms, exhibiting abnormal shape and direction, defined as a Wilson band (Rose 1979; Rose et al., 1985) (see Figure 6). Rose (1977) defines Wilson bands as,

> "a specific class of enamel defects that are defined as striae of Retzius that exhibit a linear arrangement of abnormal or absent prism structure observed with the light microscope" (1977:441).

However, some researchers have contested this definition (see Condon, 1981; Cook, 1981; Jablonski, 1981; Rose et al., 1981; Rudney, 1981). First, it is difficult to determine at what point striae should be classified as abnormal. Second, Rose's definition

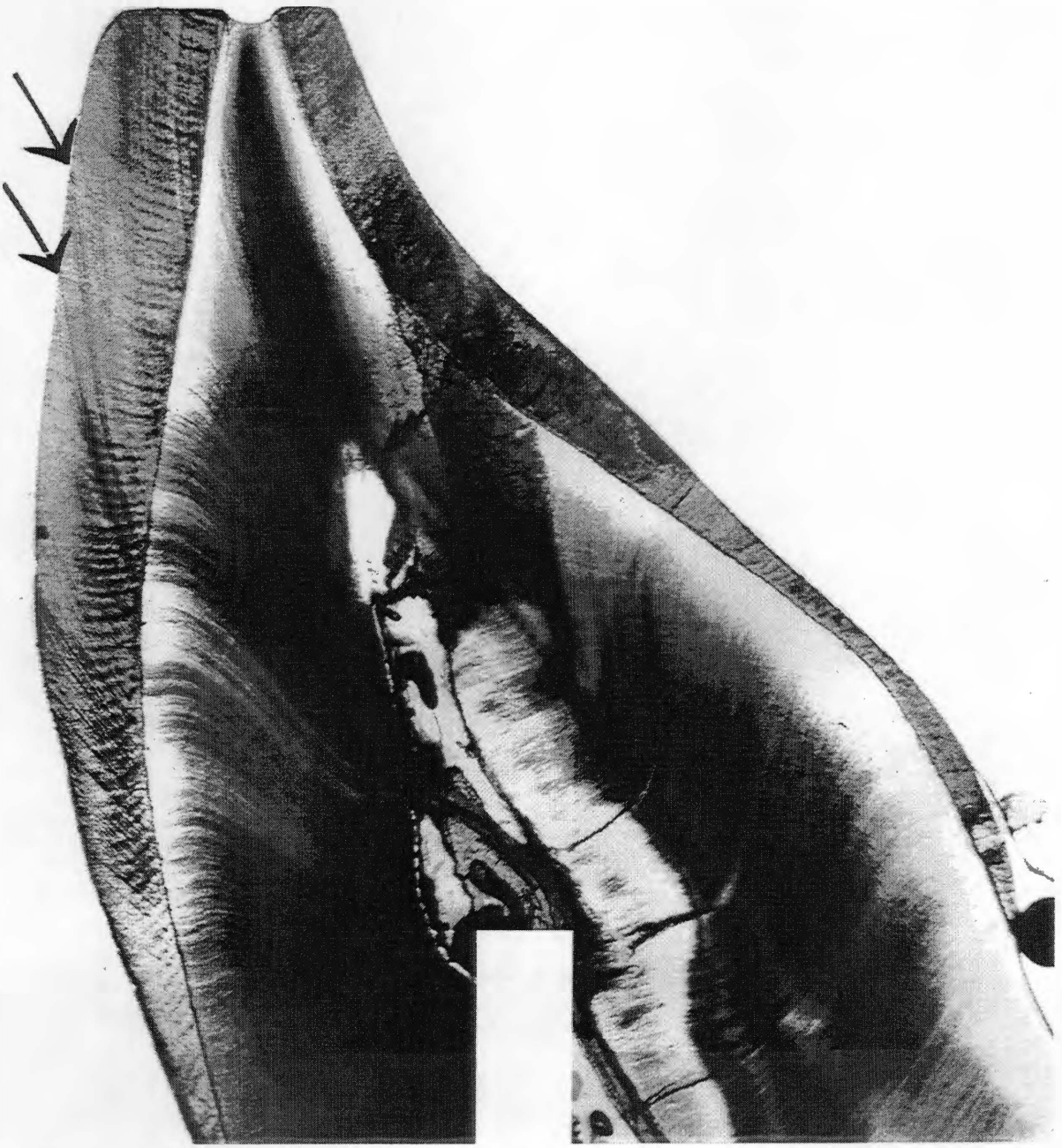

Fig 6. Wilson Bands (denoted by arrows) on the Labial Surface of a Labio-Lingual Thin Section of a Permanent Mandibular Canine at x10 Magnification.

is too constrictive, resulting in low occurrence rates. As a result, an expanded description of Wilson bands has been employed by researchers.

"At low magnifications with the optical microscope (100x) Wilson bands appear as ridges or troughs in the flat enamel surface of the dental thin section, whereas at high magnification (500-1000x) the prisms can be observed ending or bending into the enamel" (Rose et al., 1985:288; see Figure 6).

Morphology of Macroscopic Enamel Defects

More severe insults may result in the death of the ameloblast; as a result no further enamel is produced. This macroscopic deficiency in enamel thickness, due to premature death of the ameloblast, is defined as an enamel hypoplasia (Pindborg, 1970) or enamel surface defect (Condon, 1981). An enamel hypoplasia appears as a transverse, or linear region, of depressed enamel (Figure 4). The depressed enamel is either continuous forming a linear enamel hypoplasia, or discontinuous resulting in pitting of the enamel (Goodman et al., 1980; Sarnat and Schour, 1941).

Dental enamel hypoplasias are a class of quantitative enamel developmental defects. When used as a general term, enamel hypoplasias refer to all macroscopic defects in enamel thickness. The morphology of the defects may vary from minor single and multiple pits, small furrows, deep and wide vertical and horizontal troughs of decreased enamel thickness, entirely missing enamel, pit patches and cluster bands (Goodman and Rose, 1991; Eckhardt et al., 1992; Goodman et al., 1992).

By using the pattern of the defect within and among teeth, researchers can determine whether they resulted from one of three conditions: a hereditary anomaly,

localized trauma, or a systemic metabolic stress (Shawashy and Yaeger, 1986). Those defects resulting from hereditary factors generally affect the entire tooth crown and are the most severe. Defects resulting from trauma and other non-systemic factors can also be severe, but are usually found on one tooth or a few adjacent teeth. These defects cover only a thin portion of the crown. Defects resulting from systemic metabolic stress are likely to be found on a variety of teeth, which were developing at the time of the stress and mirror the relative completion of the tooth at the time of the upset (Shawashy and Yaeger, 1986).

The relationship between Wilson bands and enamel hypoplasias has not been fully established. Rose (1977) and Cook (1981) concluded that there is not a one to one correlation between the occurrence of enamel hypoplasias and Wilson bands in the mandibular canine although, in theory, they result from the same phenomena. This finding is supported by recent research on African-American skeletal populations (Marks, 1993).

Condon (1981) conducted a comparative histological study of the mandibular canine and first premolar pairs and offers clarification as to the correlation between these two enamel defects. Wilson bands appear to represent very short periods (1-5 days) of metabolic disturbance, resulting from acute stress, from which the ameloblasts quickly recover. In contrast, the histological structure of hypoplasias indicate that they represent chronic, stressful, disturbance of a large number of ameloblasts over a period of time lasting from several weeks to two months. Due to differences between Wilson bands and hypoplasias, one must utilize caution when selecting tooth type. Whereas Wilson bands

are found throughout the enamel, hypoplasias are primarily found in the midcoronal and cervical portions of canine and premolar crowns (Condon, 1981; Condon and Rose, 1992). Inter- and intra-tooth comparison indicates that the cuspal portion of each tooth is not as sensitive as the remaining enamel to disturbances causing hypoplasias. One possible explanation for this is that this portion of the tooth is formed *in utero*, offering some protection from the physiological stressors which cause enamel defects.

Pathological striae, or Wilson bands, are more difficult to observe, but are more sensitive and record precise episodes of stress over a longer interval than enamel hypoplasias (Skinner and Goodman, 1992). The limitations of hypoplasias lies in the fact that they are not capable of recording the early stages of crown formation, i.e., up to one-third of the tooth formation period. Relying only on enamel hypoplasias in the secondary dentition results in loss of information pertaining to the early stress period of an individual.

Goodman and coworkers (1980) report that the most sensitive indicator of hypoplasias between birth and three years is the incisors, while canines are most sensitive between three and six years of age.

Etiology of Enamel Defects

The etiology of developmental defects has been linked to nutritional imbalances and other metabolic conditions through experimental and animal research, as well as clinical and epidemiological studies on contemporary populations.

"The extant questions do not concern whether such factors can cause an enamel developmental defect, but rather, what factors are most likely to cause a defect, which

types of defect result, and how these studies improve our understanding of the sensitivity of enamel to disruption in humans" (Goodman and Rose, 1991:282).

Experimental and Animal Studies

Several experimental studies on enamel hypoplasias were conducted from the 1930's to 1950's. These studies demonstrated that a large number of nutritional deficiencies and excesses had the potential to lead to an enamel defect in laboratory animals (Schour and Massler, 1945). Researchers also learned that other stressors, or factors that disrupt normal physiology and growth (e.g. hormonal imbalances and diseases), could lead to an enamel defect (Kreshover, 1960).

"The inference to be drawn from this early experimental research is that enamel defects are nonspecific indicators of physiological perturbations" (Goodman and Rose, 1991:282).

Recent experimental animal research on the etiology of enamel defects has been conducted in New Zealand on sheep. Pathological changes have resulted in enamel hypoplasias and hypocalcifications (Suckling and Thurley, 1984a; Suckling, 1986). Animal experimentation which subjects sheep to trauma (Suckling, 1980; Suckling and Purdell-Lewis, 1982a; Suckling, 1989), fluoride supplementation (Suckling and Purdell-Lewis, 1982b; Suckling and Thurley, 1984b), and induced parasitism (Suckling et al., 1983, 1986) have resulted in producing enamel defects. Kreshover (1960) infected a rabbit with vaccinia virus and determined that enough evidence exists to suggest that developmental tooth defects are nonspecific and can be attributed to a wide array of systemic disturbances.

Socioeconomic and Ethnic Correlates

Studies involving social constructs have revealed a correlation between dental defects and general living conditions, nutritional, hereditary, neurological and neonatal pathological etiologies. Two phenomena occur with regards to the stages of socioeconomic development experienced by a society. First, there is a tendency for the rate of enamel hypoplasias to increase, as stressors increase, when people start to become technologically more advanced, i.e., agricultural and industrial groups (Skinner and Goodman, 1992). Second, in general, individuals in developed countries have lower rates of enamel defects than their counter-parts in underdeveloped countries (Goodman and Rose, 1991). For example, temporal differences were found with regards to the prevalence of enamel defects in African-American and European-American adult human skeletons (from the Hamann-Todd collection) and living African-American and European-American children from Cleveland (El-Najjar et al., 1978). Enamel hypoplasias were found to be more common in both skeletal samples, than in the living population. The prevalence of enamel defects for African-American and European-American groups in this particular area of the United States has declined over time and may be due to improved nutritional conditions and the elimination of common childhood diseases. The change over time disproves the notion that genetic differences account for susceptibility.

Socioeconomic conditions are a contributing factor to an increase in enamel hypoplasias in Nigerian children (Enwonwu, 1973). Those children in high socioeconomic

classes had no hypoplasias while their counter-parts in the low socioeconomic class exhibited a 21 percent incidence rate. Different ethnic groups from different regions of the world, including: South Africa (Hargreaves et al., 1989), Burnaby, British Columbia (Skinner and Tat Wai Hung, 1986), India (Lukacs and Joshi, 1992), Tamascalsingo region of Mexico (Goodman, 1988; 1991), and Solis, Mexico (Goodman et al., 1992) displayed an increase in the number of hypoplastic defects which mirrored their low socioeconomic status and ethnic affinity.

Nation et al. (1987) have contested the influence of socioeconomic affiliation in their study. The prevalence of developmental defects in different ethnic, socioeconomic, and sex categories were assessed utilizing a cross-section of the population in California children (Nation et al., 1987). The highest rate of defects were found in African-American children (60 percent), followed by Caucasians (38 percent), and Hispanics (18 percent). However, no correlation was found between the presence of enamel defects and socioeconomic level. To the contrary, the study revealed a decreased incidence of enamel defects in Hispanic children, despite the fact that the majority came from families with low education levels. This difference between Hispanic and Caucasian children seems to be due to factors directly or indirectly related to ethnic groups.

"This is further strengthened in the comparisons between Caucasians and Hispanics with similar socioeconomic backgrounds, where a lower prevalence of defects was noted in the Hispanic children in all groups of parental educational levels" (Nation et al., 1987: 333).

It was determined that there were no significant differences between males and females in the study group. The findings reported by Nation et al. (1987), regarding

socioeconomic, ethnic, and sex differences are factors which will be addressed in the present study.

Epidemiological and Clinical Studies of Contemporary Populations

To try to ascertain the etiology of enamel defects, researchers viewed contemporary populations that display enamel defects. The first researchers to undertake such a study were Sweeney and co-workers (1969) who found that children from Guatemala City suffering from enamel defects were twice as likely to have suffered from a previous infectious disease. A correlation between enamel hypoplasias and low cord-blood-serum-vitamin A level was observed in the study group.

In a follow-up study, Sweeney et al. (1971) found a correlation between the severity of malnutrition and the incidence of enamel hypoplasias. Those children suffering from third-degree malnutrition exhibited an increase in the percentage of enamel hypoplasias (73 percent) compared to those children suffering from second-degree malnutrition (42.9 percent).

In support of the established relationship between malnutrition and enamel defects, Goodman and colleagues (1989) conducted a longitudinal study of two groups of adolescents from the highland community of Texonteopan, Mexico. One group had nutritional supplements since birth and during permanent tooth development, while the other group consisted of matched controls. The results indicated that the frequency of enamel hypoplasias is nearly double in the control group versus the supplemented group.

These results were supported in a study on rural Guatemalan children (May et al., 1992). Those children who were given a nutritional supplement responded positively, indicating that enamel defects among these children are nutritional in origin.

Genetic factors appear to be the mitigating factors in a study conducted by Infante and Gillespie (1974, 1976, 1977). The study on children in rural Guatemala found,

> "that siblings of children with the lesion had a prevalence significantly greater than the total study population, suggesting that factors operating at the family level enhance the occurrence of the hypoplastic lesions in children" (Gillespie,1974: 1055).

They noted an increase in the prevalence of dental caries in those individuals suffering from enamel hypoplasias. The data suggest that the synergistic effect of under-nutrition and infection underlie the occurrence of anterior linear enamel hypoplasias and may predispose the deciduous molars to enamel defects.

A correlation between enamel defects and congenital allergies in children has been established (Rattner and Myers, 1962). It was determined that an interference with ameloblast metabolism occurs when there is congenital systemic involvement of ectodermal tissues. This is the precise tissue involved in congenital allergies.

An entourage of other disorders has been interpreted as possible etiologies for enamel defects (table 1). Some of these disorders include: malnutrition (Navia,1989 ; Rose and Boyd, 1978), mental retardation (McMillian and Kashgarian, 1961; Jaffe et al., 1985; Bhat and Nelson, 1989), vitamin D deficiency (Grahnen and Selander, 1954), low serum calcium (Nikiforuk and Fraser, 1981), intestinal lymphangiectasia (Dummer and Cardiff, 1977), gastrointestinal disturbance (Dummer, 1977), coeliac and Salmonella

Table 1. Some Conditions Which Cause Enamel Defects and Wilson Bands

Type of Research	Etiology	Source
Experimental/Animal Studies		
	Nutritional deficiencies and excesses	Schour and Massler (1945)
	Hormonal imbalances and diseases	Kreshover (1960)
	Trauma (sheep)	Suckling (1980); Suckling and Purdell-Lewis (1982a); Suckling (1989)
	Fluoride supplementation (sheep)	Suckling and Purdell-Lewis (1982b); Suckling and Thurley (1984b)
	Induced parasitism (sheep)	Suckling et al. (1983, 1986)
	Infection with vaccinia virus (rabbit)	Kreshover (1960)
Studies on contemporary populations		
	Low socioeconomic status in Nigerian Children	Enwonwu (1973)
	Low socioeconomic status in South African Children	Hargreaves et al. (1989)
	Low socioeconomic status in British Columbian Children	Lukacs and Joshi (1992)
	Low socioeconomic status in Mexican children	Goodman et al. (1992)
	Low cord-blood-serum-vitamin A levels and previous incidence of infectious disease	Sweeney et al. (1969)
	Severe malnutrition	Sweeney et al. (1971); Goodman et al. (1989); May et al. (1992) Navia (1989); Rose and Boyde (1978)
Clinical and epidemiological studies		
	Genetic factors	Infante and Gillespie (1974, 1976, 1977)
	Congenital allergies	Rattner and Myers (1962)
	Mental retadation	McMillian and Kashgarian (1961); Jaffe et al. (1985); Bhat and Nelson (1989)
	Vitamin D deficiency	Grahnen and Selander (1954)
	Low serum calcium	Nikiforuk and Fraser (1981)
	Intestinal lymphangiectasia	Dummer and Cardiff (1977)
	Gastrointestinal disturbance	Dummer (1977)
	Coeliac and Salmonella infection	Smith and Miller (1979)
	Hypoparathyroidism	Sarnat and Schour (1941)
	Down's syndrome	Johnson et al. (1965)
	Childhood nephrotic syndrome	Shusterman and Fellers (1969)
	Maternal diabetes	Noren et al. (1978); Noren (1984)
	Neonatal symptomatic hypocalcaemia	Stimmler et al. (1973)
	Cerebral palsy	Bhat and Nelson (1989)
	Hearing defects	Bhat and Nelson (1989)
	Premature or low birth weight	Noren (1984); Seow et at. (1989); Seow (1992)
	Neonatal asphyxia	Pindborg (1970)
	Neonatal toxemia	Pindborg (1970)
	Abnormal labor	Pindborg (1970)

infection (Smith and Miller, 1979), hypoparathyroidism (Sarnat and Schour, 1941), Down's syndrome (Johnson et al.,1965), childhood nephrotic syndrome (Shusterman and Fellers, 1969), maternal diabetes (Noren et al., 1978; Noren, 1984), neonatal symptomatic hypocalcaemia (Stimmler et al., 1973), cerebral palsy and hearing defects (Bhat and Nelson, 1989), premature or low birth weight (Noren, 1984; Seow et al., 1989; Seow, 1992), and neonatal asphyxia, toxemia and abnormal labor (Pindborg, 1970).

This brief survey of experimental animal studies, socioeconomic and ethnic correlates, and clinical and epidemiological studies conducted on contemporary human and non-human populations indicates that there is an increase in prevalence as living conditions become increasingly poor. Goodman and Rose (1990) present two epidemiological models which account for enamel defect formation. In the first model, environmentally and culturally derived stressors encountered by the individual combine with the individual's ability to resist the stressors to determine the type, duration, and intensity of systemic physiological perturbation. If the ameloblast is active during this time, it will be affected. The second model is termed a threshold model in which an individual is pushed beyond a threshold due to a combination of factors. These factors occur at different stages. In the first stage, unknown etiological/susceptibility factors affect ameloblastic activity. The next stage brings the individual closer to the threshold stage with nutritionally related factors. At the final stage, illness becomes severe enough to disrupt ameloblast function and cause an enamel defect. Despite the formulation of this model, it is still not clear which specific nutrients have the greatest effect on enamel

formation, or the effect of synergistic factors (i.e. the combination of disease and malnutrition) on the etiology of enamel defects.

Chapter 4

The Use of Enamel Defects in Anthropology

Early Research

Enamel defects have been incorporated into research conducted by paleopathologists, bioarchaeologists, and dental anthropologists in an effort to determine the health conditions, physiologic stresses, and cultural practices (i.e., age of weaning) of past and present populations. For several reasons, the dentition is an ideal specimen for the research constructs and constraints encountered by anthropologists. First, by virtue of its high mineral content, the dentition has an excellent recovery rate potential in archaeological and paleontological contexts. Second, due to the fact that no remodeling takes place once a tooth is formed, the resulting enamel defects are permanently recorded. However, this virtue is negated in the case of severe attrition, pre-mortem tooth loss, and carious lesions. Finally, because tooth formation and eruption sequences have been researched for several populations (see Massler et al., 1941; Nolla, 1960; Moorrees et al., 1963; Fanning and Brown, 1971; Gohdo, 1982; Shellis, 1984; Demirjian, 1986; Sunderland, 1987; Smith, 1991), it is possible to determine the age at which the resulting enamel defect occurred (however, see methodological concerns in Chapter 5).

Sognnaes (1955) was the first anthropologist to critically examine post-mortem changes on the morphology and histology of enamel, dentin, and cementum; in order to determine its integrity in an archaeological context. He observed that enamel exhibited few post-mortem histological changes, while dentin and cementum underwent remarkable

changes. The post-mortem changes in enamel were readily discernible from the pre-existing normal pathological processes (i.e., dental caries) and were classified as: canals, surface erosions, and diffuse disintegration.

Another significant early study, conducted by Schuman and Sognnaes (1956), evaluated gross and histological surface enamel defects, incremental growth lines and interglobular dentin from non-human primates. This was the first study to assay differences between Wilson bands and hypoplasias.

Falin (1961), citing Sognnaes' (1955) study, proceeded to view histologic and gross enamel, dentin and cementum from the Bronze, Mesolithic and Neolithic ages in Europe. He concluded that the ante-mortem state of the teeth (i.e., severe attrition) could have an effect on the post-mortem preservation.

Swardstedt (1966) was the first to study the patterns of hypoplasias within an archaeological population. He noted an inverse correlation between enamel hypoplasia frequencies and social status in a medieval Swedish population, as well as an increase in frequency over time. This proliferation over time could be traced to an increase in the frequency of low social status individuals. The most significant increase in the number of hypoplasias occurred between two and five years of age. He attributed increases in hypoplasia frequencies to nutritional inadequacies and a proliferation of disease. Here, Swardstedt initiated the first inter-tooth methodology. The objective was to classify the bands formed at a certain age, on specific teeth. This information was then utilized to determine their relative frequency within the study population.

The first paleoepidemiological approach, using tooth enamel, was initiated by Rose (1973) to determine the rate of stressful occurrences and the specific cultural practices, which produced the resulting enamel defect. Upon viewing histological samples from successive Native-American populations from Illinois, Rose hypothesized that childhood stress would increase with a shift from hunting and gathering to horticulture, and later to agriculture, resulting from an over-dependence on maize. The archaeological sample consisted of: Middle Woodland hunter-gatherers from Gibson Mound, Mississippian Late Woodland horticultural transition from Dickson Mounds, and Middle Mississippian maize dominated subsistence from a later period at Dickson Mounds.

It was determined, through enamel histology, that a gradual increase in susceptibility to illness occurred temporally. This was most likely due to the combined effects of an increase in population, leading to increased transmission of illness, which contributed to psychological and physiological stresses and an overall decrease in dietary quality. It was determined that there was a negative correlation between the prevalence of dietary quality. It was concluded that,

"...within each culture and sex group there is a higher percentage of individuals in the younger age groups with a defect than in the older age groups" (Rose, 1973:141).

Therefore, a person that was highly susceptible as a child is more likely to contract other incidences of disease throughout his or her life, thus being subjected to a greater number of diseases with little immunity. These individuals suffer from greater incidences of morbidity, eventually leading to an early age of death.

Recent Research

Early research on enamel defects substantiated the notion that teeth are a viable means of inferring the health status and cultural practices of past populations. The work that followed in the intervening decades expanded upon and refined the groundwork laid by previous researchers. This research covers the following topics: health status of past populations, change in health status over time, specific cultural practices (i.e. weaning practices and socioeconomic affiliation) of past societies, and their effect on the health of an individual.

Determining Health of Past Societies

In an effort to assess the health of past populations, several researchers have utilized morbidity and mortality patterns (in combination with enamel defects). Several diverse archaeological assemblages have been assessed using the severity and frequency of enamel defects. These populations range in time from the most ancient archaeological remains from Sterkfontein and Swartkrans hominids (White, 1978); to Neanderthals (Ogilvie et al., 1988); prehistoric populations from the Libben (Rose et al., 1978) and Dickson Mounds sites (Goodman and Armelagos, 1988), historic African-American populations (Marks, 1993), and modern-day, rural cultures (see above section on epidemiological and clinical studies of contemporary populations). An increase in the

prevalence of defects at the individual or population level is interpreted as an increase in stress load.

An increase in the number of defects per individual indicates an increase in the likelihood of childhood morbidity and eventual mortality. Those individuals with a greater percentage of Wilson bands (Rose, 1973; Rose et al., 1978) and enamel defects (White, 1978; Cook and Buikstra, 1979; Goodman and Armelagos, 1988; Goodman, 1991) died at an earlier age than those individuals who did not manifest defects in their enamel. The correlation of enamel defects and early-childhood health in prehistoric populations is well documented. Cook and Buikstra (1979) studied Middle Woodland and Late Woodland populations from Illinois and inferred that because the deciduous dentition is formed during the prenatal period, one could obtain information about the condition of the mother, as well as the condition of the child. It was determined that gross enamel defects occurred in the deciduous dentition of 83 of a possible 170 children under six years of age at death in Late Woodland populations from Illinois. The Late Woodland represented a shift to an increased reliance on agriculture. Circular caries, secondary to a hypoplasia, are significantly more common in the Late Woodland populations, suggesting an increase in the cariogenicity of their diet. It was also determined that,

> "...those individuals experiencing insult after birth severe enough to result in enamel defects die relatively early. Individuals whose enamel defects reflect prenatal events are somewhat more likely to survive the first two years of life and appear later in the mortality sample" (Cook and Buikstra, 1979: 656).

Goodman and Armelagos (1988) cite three processes, which may account for this phenomenon. First, the data may result from differential lifelong patterns of exposure and consequent susceptibility to illness. Second, individuals who are exposed to and survived a period of stress may suffer a loss in their ability to overcome other stresses. Third, differential lifelong patterns of behaviorally and culturally based exposure to stresses may expose certain individuals to illness more often than others.

Another means of inferring the health conditions of past populations is through assessment of skeletal indicators of stress and infection in combination with enamel defects. One such study viewed the association between Harris lines and enamel hypoplasias in a prehistoric Native-American population from California (McHenry and Schulz, 1976). No significant association was noted between the two. This is partially explained by the resorptive quality of bone. However, it is more likely that the two conditions are caused by entirely unrelated factors: hypoplasias are caused by trauma and Harris lines result from trauma, followed by quick recovery. Similarly, Kolaridou (1991) found no correlation between enamel defects and Harris line formation in a study on a French medieval population. These findings suggest that many factors may influence the development of stress indicators (Roberts and Manchester, 1995).

In a similar study, the relationship between microscopic enamel defects, macroscopic enamel defects and radiopaque transverse lines (i.e., Harris lines) in a prehistoric skeletal sample were evaluated (Clarke, 1978). Contrary to the results posited by McHenry and Schulz (1976) and Kolaridou (1991), a statistically significant association

between Harris lines and enamel hypoplasias was indicated by the results; as well as a statistically significant association between the ages of occurrence of Harris lines and enamel hypoplasias. The main differences in occurrence rates is due to subtle differences in the peak age of occurrence of each type. Clarke suggests that the subtle differences in the peak age of occurrence:

> "...may indicate that there is not a direct relationship between the occurrence of the two types in an individual, and rather, that an individual possesses both types because he or she is a highly susceptible individual" (Clarke, 1978:102).

Cribra orbitalia and Harris lines, in combination with two types of enamel hypoplasias (shallow horizontal grooves and transverse disturbances), are another type of skeletal lesion utilized to assess the health of a prehistoric population from Schleswig Holstein, North Germany (Kuhl, 1992). Cribra orbitalia and Harris lines are found in all of the cremations where pieces of the orbital roof and long bone were preserved. In young children, enamel hypoplasias and Harris lines occurred at the same time but a one-to-one correlation cannot be determined. The etiology of the skeletal defects indicates nutritional disturbances and childhood disease. Cribra orbitalia, in conjunction with enamel hypoplasias, was found to contribute to subadult mortality in ancient Nubia (Mittler et al., 1992).

Temporal Change in Health

In an effort to address the impact of secular change on specific culture groups, enamel defects have been utilized to assess the relative health of populations over time.

Specifically, a skeletal population is utilized which contains individuals interred over a long period of time (i.e. a mound burial or cemetery). Dickson Mounds, in Illinois, is ideal for this purpose because it contains burials which date from the Mississippian Acculturated Late Woodland (A.D. 1050-1200) and the Spoon River Focus of the Middle Mississippian (A.D. 1200-1300) (Lallo and Rose, 1979). Using this temporal period, Lallo and Rose (1979) viewed how a change in cultural and ecological parameters affects biological parameters. They concluded that a change in one parameter would have an effect on the others.

However, Wood et al. (1992) refute this hypothesis and offer their own explanation. It is based on the notion that an individual who manifests an enamel surface defect lived long enough after the stressor to resume normal enamel formation. Temporal trends are explained because the population consists of two groups of individuals. One that is relatively advantaged, the other disadvantaged. The advantaged group experiences childhood illness and are able to live long enough to develop enamel surface defects. This group enjoys a higher fertility rate than their disadvantaged counterparts. Wood et. al contends that,

> "individuals with observable lesions were principally from the less frail (i.e., advantaged) group, and they had a lower mean age of death than the disadvantaged group because of their high fertility, not because of poor survival. ...This hypothesis would therefore explain the temporal trends reported in both lesion frequency and mean age at death" (Wood et al., 1992: 355).

In a rebuttal to this proposed model, Goodman (1993) asserts that this is not a feasible explanation. There are several reasons which are beyond the scope of this study

and will not be addressed here (refer to Goodman, 1993). However, Goodman contends that the above mentioned scenario is not probable because there is no situation in which a clearly advantaged group, past or present, has more hypoplasias than a disadvantaged group.

> "On the contrary, enamel hypoplastic defects have repeatedly been shown to be more prevalent under conditions of lower socioeconomic status, increased exposure to disease, and decreased access to food and other basic resources" (Goodman, 1993: 284).

The secular trend in enamel hypoplasia frequency has also been interpreted for Japanese sites from the prehistoric to modern period (Yamamoto, 1992). The results showed a fluctuation in health over time.

Another question which bioarchaeologists-archaeologists attempt to address, when viewing temporal change, is the effect of European contact on indigenous populations. Several different populations have been studied and include the Maya (Wright, 1989) and Native-American sites from Spanish Florida (Larsen and Hutchinson, 1992). In the case of the Maya, there was an increase in developmental stress during the Historic period (Wright, 1989). The opposite was true for the Native-Americans from Florida. They were found to be in better health during the later Mission Period (Larsen and Hutchinson, 1992). However, this finding is not consistent with other data and may be due to sample bias related to the older age structure of the Mission Period skeletal sample.

Cultural Indicators of Stress

Another topic of anthropological research is the effect of weaning on the health of the individual and the resulting enamel defect. Several researchers have noted that there is an increase in the frequency of enamel defects between the ages of 1 and 4 years (see Rose and Boyd, 1978; Clarke, 1980; Corruccini et al., 1982; Goodman et al., 1984; Corruccini et al., 1985; Goodman et al., 1987; Lanphear, 1989; Ubelaker, 1992). The peak age of occurrence is culture specific and,

> "... is explained as a result of the direct nutritional, immunological and psychodynamic effects of weaning, and other developmental behaviors and physio/immunological patterns associated with the weaning age interval" (Clarke, 1980:79).

The weaning age is generally older in children from hunter-gatherer societies and younger in more industrialized societies. For example, in a skeletal sample which dates from: Mesa Verde area Pueblo I-III Anasazi, Illinois Late Woodland, Mississippian Acculturated Late Woodland and Middle Mississippian Dickson mounds (Clarke, 1980); the age distribution of lesions in the sample indicates a common peak occurring between 1 to 3 years of age. Utilizing the same skeletal assemblage, Goodman and coworkers (1984) found a peak age of frequency to be between 2 and 4 years of age. Likewise, an ancient Ecuador population, consisting of 1185 individuals from archaeological sites spanning 8,000 years, shows an increase in childhood stress with a shift in subsistence to agriculture (Ubelaker, 1992). The increased number of enamel defects in children was attributed to the physiological stress suffered during weaning.

The effects of weaning and the incidence of enamel defects were studied in a historic skeletal sample of 296 individuals from a late 19th century American poorhouse cemetery (Lanphear, 1989). Both frequency and chronological distribution were assessed in the mandibular canines and maxillary central incisors. The frequency of enamel hypoplasias per individual by tooth ranged from 70% to 73%, with a peak age at stress of 2.5 to 3.0 years for the maxillary central incisor and 3.5 to 4.9 years for the mandibular canine.

"...The peak age at stress between 2.4 and 4.0 years in this 19th century sample transects the ranges reported for prehistoric populations (2-6 years) and for modern groups (0-3 years). These results indicate that the stresses associated with weaning probably occurred earlier in incipient industrial societies than in prehistoric hunter/gatherers and agriculturists, yet not as early as in modern industrial groups" (1989:35).

Another historic population from the middle 19th century Florence, Italy was studied to determine the age of occurrence of enamel hypoplasias (Moggi-Cecchi et al., 1994). The age interval, which showed the greatest proportion of hypoplasias, was that between 1.5 and 3.5 years. This age range was correlated with historical sources on the weaning habits of 19th-century Italian populations, which indicate a weaning period between 12 and 18 months. Wide grooves in the enamel were noted between 2.0 and 2.5 years, while shallow grooves were noted between 2.5 and 3.0 years. The distribution of these grooves are believed to be correlated with the introduction of dietary supplements until weaning is complete.

Despite the interpretation by several researchers that peak episodes of stress at 36 months are due to weaning, Skinner and Goodman (1992) contest that this phenomenon is most likely due to flawed methodology. They cite both biological and anthropological reasons for the unlikelihood that weaning stress would occur at such a late age. For example, it is commonly observed that in non-industrialized societies weaning takes place at around 13 to 24 months. The reason cited by anthropologists for a difference in time is due to a lag between weaning and the onset of stress. This difference can be as great as one year (Corruccini et al., 1985). Supplementation in the diet takes place among the San at 6 months even though they breast-feed frequently and for a number of years (Gaulin and Konner, 1977). "For these reasons it is suggested that dental reconstruction of age at weaning is uncertain" (Skinner and Goodman, 1992:168).

Subsequently, Blakey and coworkers (1994) attribute the occurrence of hypoplastic defects among enslaved African-Americans to factors other than post-weaning stresses. Their analysis of this sample exhibited a peak age between 1.5 and 4.5 years of age. This age range is later than those reported for similar plantation populations. Blakey and coworkers caution that:

"High hypoplasia frequencies during the middle years of enamel development are more likely the result of a combination of 1) multiple environmental stresses, 2) differences in hypoplastic susceptibility in enamel, and 3) random factors" (1994:371).

Although weaning stress appears to have some effect on the formation of hypoplasias, a direct correlation could not be traced between the two. These findings raise doubts about the assignment of peak stress episodes to weaning events by previous researchers.

Another prevalent use of enamel defects in anthropology has been the determination of status based on the predominance of these defects. Storey (1988) compared two skeletal populations from Mesoamerica. The low-status sample from Teotihuacan was compared to a high-status sample from Copan. Those individuals from Teotihuacan showed an increase in childhood mortality and prenatal enamel defects, while those individuals from Copan had less infant mortality and markedly less prenatal defects.

Indication of Previous Medical Conditions

An avenue of recent research incorporated the use of Wilson bands in an effort to solve a forensic case (Skinner and Anderson, 1991). A histological analysis of enamel defects was used to determine the likelihood that the timing of specific episodes of stress apparent in the enamel corresponded to those recorded in the medical record. It was concluded that Wilson bands have the potential for making the identification of immature, and otherwise unremarkable, skeletons much more probable when used in conjunction with other methods.

Chapter 5

Materials and Methods

Permanent maxillary and mandibular incisors and canines (n=194) were collected from private practice and public health oral surgeon's offices located in Marin County, California, Memphis and Knoxville, Tennessee. The collection process took place from May 1996 until October 1997. The sample consists of 194 permanent anterior teeth from 78 individuals, of which 46 are males and 32 are females (see Table 2).

Documentation, embedding, and sectioning were conducted in Dr. Murray Marks' mineralized tissue histology laboratory at the University of Tennessee, Knoxville. Embedding and sectioning procedures followed those of Marks (1993) and Marks et. al (1996).

Documentation

Documentation for each tooth included recording the following information: the patient's age, sex, and ethnicity, the collection locale, and tooth type. Each tooth was assigned a random four digit number from a random number table (Leonard, 1976). Using helios digital calipers, measurements were recorded for each tooth, and included: crown length, root length, mesial distal diameter of the crown, mesial distal diameter at cervix, labio-lingual diameter of crown, labio-lingual diameter at cervix, and the curve of the CEJ on the mesial side of the tooth (after Wheeler, 1983). Measurements for each tooth are reported in the appendix (refer to Table A-1).

Table 2. Sample Specifics

	Total Teeth	Total Individuals	Teeth With Wilson Bands	Individuals With Wilson Bands	Practice Locale With Wilson Bands	Sex of Individual With Wilson Bands
Total Number	193	78	81	46	Private: 13 Public: 68	Male:39 Female:42
Percentage			42%	59%	Private: 16% Public: 84%	Male:48% Female:52%

Embedding Procedures

Each tooth was cleaned by soaking in a 50/50 solution of water and bleach for twelve hours. The tooth was removed, rinsed under water, dried and remaining debris was removed using a dental pick. Teeth were then placed in ethyl alcohol for 48 hours to facilitate dehydration. They were removed and air-dried in the lab.

To maintain a horizontal orientation during the embedding process, a wire stand was constructed for each tooth. The stand was held in place in the Peel-A-Way disposable molds (22 x 30 mm) using a few drops of Super Glue (Cyanoacrylate). Paper identification tabs were inscribed with each specimen's four digit identification number and placed in the holding facets of the mold.

Epoxide Resin and Hardener were mixed according to weight (a ratio of 5 to 1: resin to hardener). The mixture was stirred until it changed temperature and color, and then it was poured over the tooth, until the mixture covered the tooth. It is essential that enough of the epoxy mixture be poured to allow for shrinkage, which occurs during curing. The epoxy covered teeth were placed into a vacuum desiccator and air was extracted until bubbles ceased to escape from around the tooth. The molds were then placed into a pressure chamber under 50 p.s.i. of air and permitted to cure overnight. Embedded teeth were removed from the Peel-A-Way trays and sharp edges were removed using 150 grit sandpaper. Removal of sharp edges not only protects one's fingers from cuts, it also makes for easier affixing of the slide to the section.

Thin-Sectioning Procedures

The embedded teeth were mounted on a Buehler Isomet Slow Speed Saw and two longitudinal labio-lingual thin-sections, averaging 0.4 mm thick, were made for each tooth. A slide etched with the four digit identification number was affixed to each thin-section using Duro Superglue. After the saw finished a cut, the section was cleaned with alcohol to remove cutting oils and residue.

Wilson and Schroff (1970) and Rose (1977) proposed that enamel rod orientation could be differentiated using a mild acid solution (see Hillson, 1996 for a list of solutions). Slides were etched using a one normal solution of hydrochloric acid for 15 seconds with moderate agitation. The slides were then rinsed in tap water, placed in 95% alcohol, and dried. Following the etching of the slides, there was no need to polish the sections, as the etching eliminated most of the scratches.

Light Microscopy

Sections were viewed using a Nikon light microscope in Dr. Simek's Analytical Archaeology Laboratory at the University of Tennessee, Knoxville. Each slide was viewed under high magnification (x100) with the substage condenser fully opened. A Wilson band was recorded if it was traceable at least half-way from the DEJ to the enamel surface and conformed to the definition proposed by Rose and co-workers (1985) (refer to Chapter 3).

The neonatal line is a typical Wilson band, resulting from the abrupt change in nutrition and environment during the birthing process. When discernible in the permanent central incisors, the neonatal line was not recorded as a defect. This decision was based on the fact that although this is technically a defect, it is the result of a natural, albeit stressful, perinatal event.

Digital images of the enamel (for age estimation) were taken with the assistance of Dr. Larry Anovitz and Dr. David Cole at the lab of Dr. David Cole, Oak Ridge National Laboratory, Oak Ridge, Tennessee. Each image was captured at 1.25x using a Sony Digital Photo Camera (DKC-5000) and an Olympus light microscope. Images were manipulated using Photoshop software and saved to zip disk. Due to the size of the enamel and the low magnification (1.25x) two to four images were required for each tooth. The piecemeal images for each tooth were composed by Mt. Robert Brown at Oddworld Inhabitants, San Luis Obispo, California.

Interpretation of Defect Timing

Methodological Concerns

Perhaps the most difficult exercise in enamel histology is the assignment of the chronological age of occurrence to a specific enamel defect (see Marks, 1993; Goodman and Rose, 1991; and Smith, 1991 for a summary). The basis of this difficulty lies in the fact that crown maturation standards (both within and between populations) are poorly understood and give different results according to whether the study was conducted by

measuring tooth formation/calcification/eruption sequences in radiographs (Nolla, 1960; Moorrees, et al., 1963; Fanning and Brown, 1971), or formation/calcification/eruption sequences using histology (Massler, et al., 1941; Gohdo, 1982; Shellis, 1984; Sunderland, et al., 1987). Another inconsistency lies in the fact that researchers must assume that tooth formation rates from modern populations (consisting of different ethnic groups) are representative of, not only all ethnic groups being studied, but also adequately represent rates for archaeological populations. Other assumptions involve 1) that tooth size variation has little impact on age determination; and 2) tooth development rates are continuous, despite the fact that human beings mature at different rates, depending on sex and ethnicity (see Demirjian, 1986).

The first researchers to study the chronometric potential of enamel hypoplasias were Sarnat and Schour (1941). Their sample consisted of a contemporary clinical series in which the locations of enamel defects were compared with the standards for enamel crown development refined by Massler and coworkers (1941).

Swardstedt (1966) revised the Sarnat and Schour (1941)/Massler et al., (1941) crown development chronology by accounting for differing growth rates within and between teeth. Although the chronology for enamel crown development initiated by Massler et al., (1941) is used by Swardstedt, this method is more accurate than the previous Sarnat and Schour (1941) method because he is more precise in his description of how to translate the position of an enamel hypoplasia on a tooth crown to the specific age of the individual at the development of the defect. Swardstedt calculated the mean

tooth crown heights for each tooth type and obtained the time each crown begins and ends calcification. The tooth is then divided into half-year intervals. The intervals vary in width and are related to the assumption of differences in the velocity of growth. Although there are some fundamental questionable assumptions made in Swardstedt's method, it is a chronology which laid the basis for subsequent studies.

Goodman and coworkers (1980) modified Swardstedt's interpretation of the Massler et al. (1941) standards by incorporating an assumption of constant velocity of tooth growth. Although they admit that there is a likelihood that crown formation does not occur at a constant velocity, they suggest that the notion of constant velocity in crown formation continues to be used as a null assumption until researchers are able to address this question.

Rudney (1979; 1981) calculated the age of Wilson band occurrence by counting the number of bands which intersect the dentin-enamel junction through standardizing their location into percentages of total dentin-enamel junction length and then appropriating those segments into developmental chronologies.

Cook (1981) contends that using a methodology which uses a fixed number of intervals disobeys current knowledge about enamel formation. Being that the crown grows at a constant rate, a bigger tooth will take longer to form than a smaller one. Dividing the tooth into one-half units introduces inaccuracies. The most efficient method to employ is to measure the distance of the defect from the cuspal tip of the dentin-enamel junction and then use the known rate of dental development to calculate the age of

occurrence. The rate of dental development is determined through zones (1-6) which correspond to chronological ages. The result is a count of the frequency per millimeter, rather than the frequency per developmental interval. However, one disadvantage of this method is that it cannot be use on extremely worn dentition, thus limiting the sample size. In order to correct for this problem, Rose and co-workers (1981) counted backward from the age of crown completion to the dental defect. Another limitation is that the results are population specific.

Blakey and Armelagos (1985), in their study of enamel defects on deciduous dentition of prehistoric populations from Dickson Mounds, Illinois tested the effect of crown size on determining chronological age of enamel defects. They chose to use tooth-specific, or individual crown heights, as the basis for timing the age of occurrence of enamel defects. They cite three reasons in support of this decision: 1) to curtail the degree of speculation required in timing defects in larger than average teeth; 2) to maintain consistency with previous research in which timings do not exceed the standard developmental periods; and 3) the timing differences between the two methods appear to be slight.

In an effort to rectify the inherent assumptions and possible fallacies associated with age estimation efforts, Hodges and Wilkinson (1990) test the effect of tooth size and make suggestions based on sample quality. Their results support the conclusion that tooth size variation is responsible for differences in chronological distributions within a sample. They suggest that individual tooth specific crown heights be used whenever possible for

aging defects. This number is obtained by measuring the distance of the defect from the cementum-enamel junction (CEJ) and subtracting it from the crown height. This value (the distance of the defect from the incisal surface) was divided by the crown height to obtain the percentage of crown formed at the time the defect was formed. The percentage of crown formed is then compared to a table to determine the corresponding chronological age-at-stress.

However, if severe attrition exists and crown height cannot be measured using the tooth-specific method, a method should be utilized which incorporates that sample's mean crown heights. This method is achieved by taking the percentage of crown formed at each age (calculated by dividing the CEJ distances in the Goodman et al., (1980) chart by the crown height given in the chart) and multiplying the values by the sample's mean crown height. The resulting values are the total amount of enamel formed by the end of each age interval, or the distance from the incisal surface. To calculate the distance from the CEJ, the distance form the incisal surface is subtracted from the mean crown height. This results in an adjustment of the Goodman chart CEJ distance values according to the mean heights of the population under study. Once again, a table is consulted by locating the measured distance of the defect from the CEJ. Next to this value, the corresponding age-at-stress is given.

Berti and Mahaney (1992) subject tooth-specific and sample-specific chronology methods to simulations of enamelization to predict their confidence intervals. The results

indicate that a true confidence interval for their estimated ages at the linear enamel hypoplasia formation is not possible.

To alleviate the aforementioned inconsistencies, researchers need to make a concerted effort to define crown formation sequences and timing rates for different populations (Hillson and Bond, 1997). Despite the method chosen for constructing chronological age at occurrence of enamel defects, it is important to retain and report raw data (Goodman and Rose, 1991; Skinner and Goodman, 1992). In the event that our understanding of chronological age determination improves, and new standards are developed, recalibration of raw data can be quickly facilitated.

Evaluating Age of Wilson Band Occurrence

By virtue of the fact that this sample consists of modern individuals, utilization of crown maturation standards, derived from a modern population (i.e., Swardstedt, 1966; Goodman et al., 1980), is applicable without the introduction of many of the concerns discussed previously. However, there is still the persisting problem of the lack of knowledge about crown maturation standards for different ethnic groups.

The maxillary central incisors and mandibular canines were selected for analysis (see the discussion in the tooth sensitivity section) and a digital image was made for each (see Figure 7). To minimize distortion, throughout the composing process, a print resolution of 300 pixels per inch was maintained. The finished image has several advantages over standard micrographs. First, it provides the researcher with the ability to

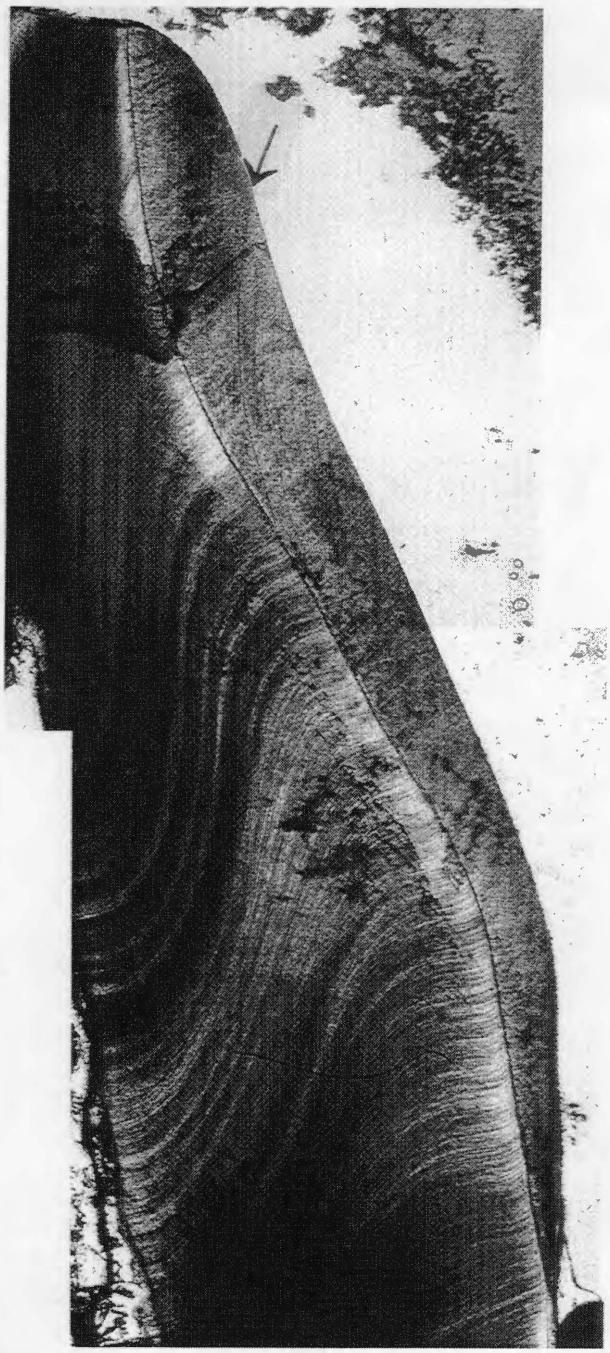

Figure 7. An Example of Composed Digital Image Used in This Study Showing the Lingual Surface of a Mandibular Right Canine. Arrow Designates Position of the Wilson Band (at x 10 magnification).

take images of a tooth, using enough magnification for the Wilson band to be seen. This is accomplished by composing several images into the final image. Second, the brightness and contrast of the image can be manipulated by the investigator to accentuate the Wilson band. This feature affords the utilization of thicker slides, which would otherwise be indiscernible.

The chronological method chosen is a combination of the Schour and Massler (1941)/Massler et al., (1941), subsequently redrawn and calibrated by Swardstedt (1966) and Goodman et al., (1980). Despite the aforementioned problems with the Goodman and coworkers (1980) methodology (see methodological concerns section, this Chapter), it has several advantages. It is the most readily employed by previous researchers (i.e., Rose, 1973; Boyde, 1978; Marks, 1993) and allows for comparability across studies. Crown maturation standards were calculated from a modern sample of European and European-Americans. This is similar (though not exact) to the ethnic sample in the present study. Since there is no correct method (Hillson and Bond, 1997), the raw data will be retained for recalculation (Skinner and Goodman, 1992), in the event that a new method is formulated, which rectifies the inherent erroneous assumptions. Age was determined by following the enamel defect from its emergence on the enamel surface to its intersection at the DEJ. At this location, the tooth was divided into six-month growth increments representing initial crown formation to crown completion (see Figure 8). For the maxillary central incisors, the tooth was divided into nine units. In the case of the mandibular canines, the tooth was divided into eight units. The length of the tooth was

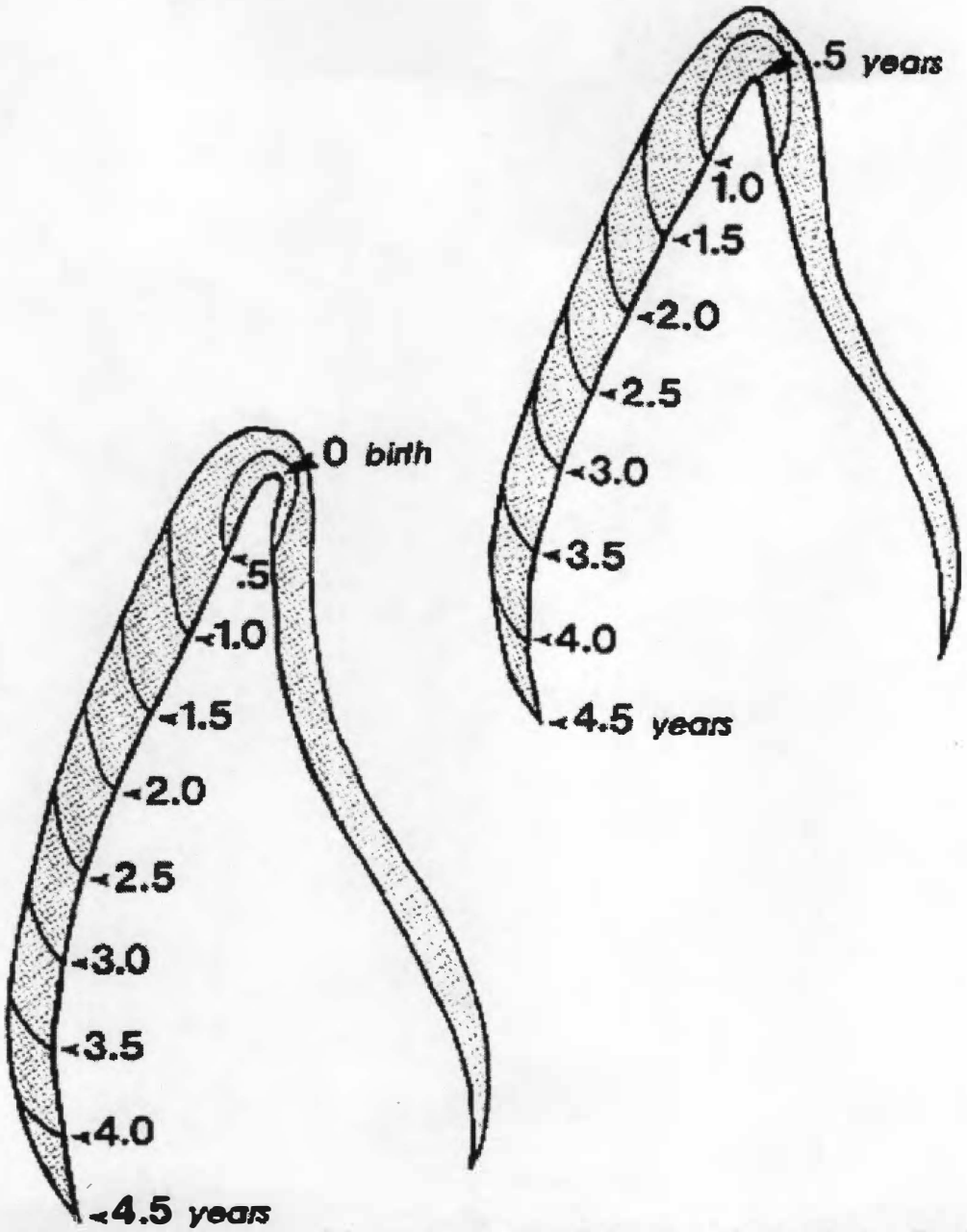

Figure 8. An Example of the Chronometric Method Used in this Study. The Figure on the Left is an Incisor. The figure on the Right is a Canine.

Source: Swardstedt T (1966) Odontological Aspect of a Medieval Population in the Providence of Jamtland/Mid-Sweden. Stockholm: Tiden-Barnangen AB.
As Presented in: Marks MK (1993) Dental Enamel Microdefects As Indicators of Morbidity Among Historic African Americans. Ph.D. Dissertation, University of Tennessee, Knoxville.

determined by measuring the digital image from the CEJ to the occlusal surface. Attrition was minimal for this sample and did not affect most of the teeth. A more persistent problem was destruction by carious lesions and destruction during tooth extraction. When these intrusions destroyed the CEJ, the opposite side was used. In the rare case that both sides were destroyed, the CEJ was estimated. In general, the side of the tooth that most clearly demonstrated the enamel defect was the side captured by the computer.

Tooth Sensitivity

There is considerable but little understood differences in inter-tooth and intra-tooth sensitivity to the stressful events, which result in enamel defects (see King and Wei, 1994; Skinner and Goodman, 1992). This stems from the fact that ameloblasts responsible for forming enamel on different teeth are not equally sensitive to developmental disruption (Goodman and Rose, 1990). Rose et al., (1978) determined that the teeth most sensitive to systemic growth disruptions and stress are the mandibular canine and maxillary central incisors. Subsequent research (see Lanphear, 1990; Mack and Coppa,1992; Lukacs and Joshi,1992; Moggi-Cecchi et al., 1994) found an increase in sensitivity in the maxillary central incisor and mandibular canine to hold true for hypoplastic defects. In fact, Goodman and Armelagos (1985) found that between these two teeth, the maxillary central incisors were 1.36 times more susceptible than the mandibular canines to hypoplasias.

The maxillary central incisors and mandibular canines were selected from the sample of teeth collected and used to estimate age of Wilson band occurrence. They were

chosen because of their reported sensitivity, their documented crown formation periods, and their applicability to previous studies.

With regard to intra-tooth sensitivity, Goodman and Armelagos (1985) found the highest density of hypoplastic events located just cervical to the midpoint and Suga (1989) asserts that lingual enamel is less vulnerable to the stresses recorded by enamel defects. Condon and Rose (1992) state that when viewing both Wilson bands and hypoplastic defects, the mandibular canine exhibits a greater frequency of both defects. Upon viewing Wilson bands exclusively, they noted that Wilson bands exhibit the greatest sensitivity, occurring throughout the tooth crown, while enamel surface defects were absent in the occlusal region, infrequent in the midcoronal region and were most common in the cervical region. Skinner and Goodman (1992) support these findings. They assert that Wilson bands are more sensitive and record precise periods of stress over a longer interval than enamel surface defects. The limitations of surface defects lies in the fact that they are not capable of recording the earliest stages of crown formation, up to one-third of the tooth formation period. Relying only on surface defects in the permanent dentition results in loss of information pertaining to the early stress period of an individual.

Statistical Procedures

The sample consisted of teeth that were the by-product of extractions. As a result, several teeth were collected for each individual (including rights and lefts for the same tooth) . Since several teeth are forming in the adult dentition simultaneously, a systemic

stressor which affects the individual will affect multiple teeth. Mere reporting of Wilson band frequencies by individual would result in inflated frequencies. In order to address the duplication of right and left teeth, McNemar's test was performed by tooth type to determine whether asymmetry exists between the presence of a Wilson band and the side on which it occurs. However, the sample size was too small (N= 88 duplicate teeth) for the test to work properly. As a result, those teeth which had both right and left antimeres were isolated, and one side was chosen at random for each tooth (see Konigsberg, 1987).

This revised sample size (N=149) was analyzed by tooth type (i.e., maxillary canines, maxillary lateral incisors, maxillary central incisors, mandibular canines, mandibular lateral incisors, and mandibular central incisors) using Statmost for Windows. The presence or absence of a Wilson band was recorded in a 2x2 contingency table, and a Chi-square test was performed for each of the three variables (i.e., sex, collection locale, and ethnicity).

Chapter 6

Results

Wilson Band Frequency Paired with Sex, Collection Locale, and Ethnicity

The results of the Chi-square test for independence of variables yielded some interesting and unexpected results. The purpose of this test is to determine if the observed Wilson band frequencies depart significantly from the frequencies proposed by a null hypothesis (Madrigal, 1998). The null hypothesis in this case is that the observed Wilson band frequencies are not significantly different from those expected if the null hypothesis were true. The Chi-square results and their associated probabilities are reported in Table 3, Table 4, and Table 5. The raw Chi-square tests are located in the Appendix (Table A-3). A low probability (less than .05) indicates that the frequency of Wilson bands and the variable being tested are related. A high probability (above .05) indicates that the frequency of Wilson bands and the variable being tested are not related. Those teeth which demonstrate a low probability are demarcated with an asterisk. These teeth show a relationship between Wilson band frequency and the variable for which they are being tested.

Table 3 demonstrates such a relationship: for all the teeth tested, the probability exceeded the 0.05 level of significance. The results indicate that there is no relationship between Wilson band frequency and the sex of the individual.

Table 3. Chi-square results for the relationship between Wilson band frequency and the individual's sex.

Tooth Type	Chi-Square Value	Probability
Maxillary Canine	0.3688	0.5436
Maxillary Lateral Incisor	0.7521	0.3858
Maxillary Central Incisor	0.042	0.8377
Mandibular Canine	0.0002	0.9882
Mandibular Lateral Incisor	0.2292	0.6321
Mandibular Central Incisor	0.4733	0.4915

* Significant at .05

Table 4. Chi-square results for the relationship between Wilson band frequency and collection locale.

Tooth Type	Chi-Square Value	Probability
Maxillary Canine	0.0099	0.9209
Maxillary Lateral Incisor	0.351	0.5535
Maxillary Central Incisor	0.1421	0.7062
Mandibular Canine	1.5512	0.213
Mandibular Lateral Incisor	2.4212	0.1197
Mandibular Central Incisor	10.3678	0.0013*

*Significant at .05

Table 5. Chi-square results for the relationship between Wilson band frequency and the individual's ethnicity.

Tooth Type	Chi-Square Value	Probability
Maxillary Canine	0.2625	0.6084
Maxillary Lateral Incisor	0.2778	0.5982
Maxillary Central Incisor	0.0007	0.9783
Mandibular Canine	4.2525	0.0392*
Mandibular Lateral Incisor	2.3457	0.0126*
Mandibular Central Incisor	2.75	0.0972

* Significant at .05

Table 4 is a test of Wilson band frequency and collection locale. The locales consist of private practice oral surgeons and public health oral surgery clinics. The majority of the teeth indicate that there is no relationship between Wilson band frequency and where the teeth came from (for implications, see discussion section). However, this was not the case for the mandibular central incisor. It indicates that there is an association between Wilson band frequency and collection locale. The public health department exhibited the majority of the Wilson bands (N=12), while mandibular central incisors collected from private practice locales had no teeth with Wilson bands (refer to Appendix Table A-3, entitled "Mandibular central incisors by collection locale").

Table 5 tests the independence between the individual's ethnicity and Wilson band frequency. Although the original sample consisted of teeth from African-American, European-American, Asian, and Hispanic, this table only gives results for the African-American and European-American sample. The Asian (N=3) and Hispanic (N=3) specimens have a sample size that is too small to test for independence. The high probabilities for the maxillary teeth indicate there is no relationship between Wilson band frequency and ethnicity. However, the mandibular teeth give different results. The canines and lateral incisors denote low probabilities (0.0392 and 0.0126, respectively) indicative of a relationship between an individual's ethnicity and Wilson band frequency. The majority of the teeth (for mandibular canines N= 16; for mandibular lateral incisors N=16) did not exhibit Wilson bands and were from European-Americans (see Appendix

Table A-3, entitled "Mandibular canine by collection locale" and "Mandibular lateral incisor by collection locale"), indicating that individuals from European-American ancestry were less likely to manifest a Wilson band. The central incisors exhibit a low probability (0.0972) in comparison to the maxillary teeth. However, this is significant at the 0.05 level, and indicates that there is no relationship between Wilson band frequency and ethnicity.

Chronology of Wilson Band Occurrence

Age of Wilson band occurrence was facilitated by measuring and dividing the digital images into the appropriate six-month age intervals. Mandibular canines (N=11) and maxillary central incisors (N=7) with Wilson bands were selected from the sample, and the proper chronometric determination made for each of the two tooth types. Figure 9 is a graphic representation of the number of Wilson bands that are manifested in each of the half-year age intervals. Wilson bands are absent from the teeth until 1.5 years, where they remain until 4.0 years of age. The canines are the most sensitive for every age unit in which Wilson bands appear, except during 2.5 and 3.0 years, when the incisors exhibit a greater number of Wilson bands.

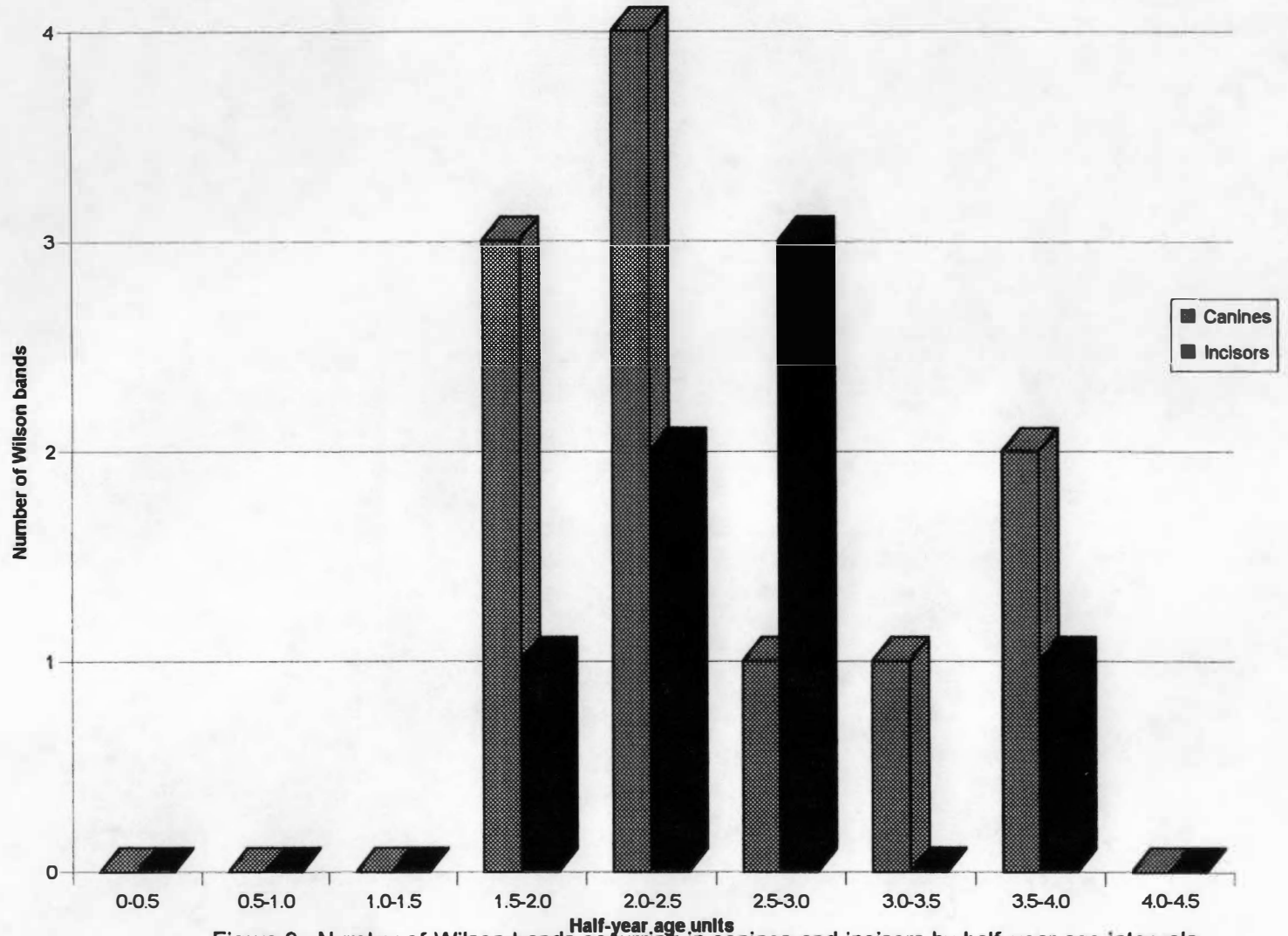

Figure 9. Number of Wilson bands occurring in canines and incisors by half-year age intervals.

Chapter 7

Discussion

Wilson Band Frequencies by Sex, Collection Locale, and Ethnicity

Although enamel defects have been utilized by anthropologists and dental researchers for decades (see Chapter 4), none, to date, have studied microscopic enamel defects as they occur in a sample of the adult living population. By virtue of the fact that this sample consists of a modern population, in which specific information (i.e., sex, ethnicity, age, and socioeconomic affiliation) could be collected, many of the constraints (i.e., unknown age, sex, and the biasing effects of mortality selection) that bioarchaeologists encounter were avoided. However, dealing with a modern population introduced some new problems to contend with. The extraction process resulted in damage to the CEJ on several specimens. Fortunately, not every specimen which had this damage exhibited it bilaterally and the CEJ was discernible on one side. In situations where this was not the case, an average height for that tooth type was used. The nature of the sample dictates that many of the teeth contained restorations, fillings, and carious lesions, which destroy the enamel (and the possible citing Wilson bands). For this reason, it is entirely possible that Wilson band frequency is underrepresented.

The results for the frequency of Wilson bands and the individual's sex indicate that there is no relationship between the two. This result is supported by previous researchers: Nation et al. (1987), Skinner and Hung (1986), King and Wei (1992), Lukacs and Joshi (1992), and Marks (1993), and is to be expected for this sample.

However, the results for the frequency of Wilson bands and collection locale deviate somewhat from what was expected. For the purpose of this study, socioeconomic standing is based on collection locale. As a rule, individuals frequenting the public health facilities are assigned to a lower socioeconomic standing than those who visit private practice oral surgeons. There are several problems with making a division based on collection locale. It is necessary to note that because enamel defects form when an individual is young, the defect is actually representative of the parent's socioeconomic affiliation. It is not unheard of for individuals with adequate income to visit dental school facilitates to have dental work done, in an effort to save money. However, this does not occur at the public health department. To receive treatment at one of these facilities, an individual must provide proof of income and residency (Knox County Public Health Department, personal communication). The specific income amount is determined by the County Commission and is not made public knowledge. However, these individuals have an income which is deemed below average for the particular county in which they reside. Based on time constraints and resources available in this study, this division is feasible if one realizes that it is not a definitive division, and is open to future refinements.

Previous studies involving bio-social constructs have revealed an inverse correlation between dental defects and general living conditions. Socioeconomic conditions are a contributing factor to an increase in enamel hypoplasias in different regions of the world, including: Nigeria (Ewonwu, 1973), South Africa (Hargreaves et al., 1989), British Columbia (Skinner and Hung, 1986), India (Lukacs and Joshi, 1992), and

Mexico (Goodman, 1991; Goodman et al., 1992). Nevertheless, this is only true for the mandibular central incisors. The remaining teeth do not show a relationship between Wilson band frequencies and socioeconomic affiliation. Although this finding is supported by Nation and co-workers (1987), whose study contests the influence of socioeconomic affiliation, these studies agree for different reasons. Their study revealed a decreased incidence of enamel defects in Hispanic children, despite the fact that the majority came from families with little education; while the present study is confounded by the fact that ethnicity is influenced by collection locale (see below).

When the raw data are considered, teeth collected from the public health department numbered 107 (table 6). Of this number, 52 teeth, or 49% manifested Wilson bands. There were 42 teeth collected from private practice oral surgeons. Of this number, 10 teeth, a mere 24% manifested Wilson bands. These percentages demonstrate that a tooth from an individual who frequents the public health department is likely to exhibit a Wilson band. One obvious implication is that individuals in a lower socioeconomic class are experiencing a greater amount of physiological stress, which is recorded by the enamel defect. The inconsistency in the Chi-square test may stem from the division of teeth into tooth types, resulting in smaller numbers in each category, which are not discernible by the test as significant.

Table 6. Number of teeth from each collection locale and percentage of Wilson bands reported by tooth class.

Tooth Type	Teeth in Sample from Private Practice	Teeth in Sample from Public Health
Maxillary Canine Count	5	17
% with Wilson Bands	2%	35%
Maxillary Lateral Incisor Count	7	15
% with Wilson Bands	57%	33%
Maxillary Central Incisor Count	8	14
% with Wilson Bands	38%	36%
Mandibular Canine Count	4	23
% with Wilson Bands	0%	48%
Mandibular Lateral Incisor Count	10	23
% with Wilson Bands	20%	57%
Mandibular Central Incisor Count	8	15
% with Wilson Bands	0%	80%

With respect to the results of the Chi-square test on Wilson band frequencies and the individual's ethnicity, the maxillary teeth indicated that there is no relationship between the two. However, as a group, the mandibular teeth had much lower probabilities and the mandibular canines and lateral incisors verify that a relationship exists between an individual's ethnicity and Wilson band frequency. Examination of the raw data counts show that the difference is attributed to the fact that a majority of the teeth from European-Americans do not manifest Wilson bands. A total of 114 teeth were collected from European-Americans. From this sample, 73 teeth, or 64%, of the teeth had no Wilson bands. This result is confounded by the fact that the European-American sample constituted 38 of the 42 teeth collected from private practice locations, which have a low percentage (24%) of Wilson band occurrence. In the case of the mandibular canines and lateral incisors, European-Americans are not under as much physiological stress as their African-American counterparts.

Implications for Age of Wilson Band Occurrence

Previous researchers have attributed a peak age of occurrence, between one and four years, to the stresses suffered during weaning (see Chapter 4). This age is culture specific and becomes progressively younger as a society increases in industrialization. The age is dependent on the tooth utilized. For example, Lanphear (1989) found the age to be between 2.5 and 3.0 years for the maxillary central incisor, and between 3.5 to 4.9 years for the mandibular canine. For this study, the age of occurrence for the maxillary central

incisors is between 1.5 and 4.0 years, with the greatest frequency between 2.0 and 3.0 years. The age of occurrence for the mandibular canines is between 1.5 and 4.0 years, with the greatest frequency between 1.5 to 2.5 years.

Because weaning times are culture specific, it is necessary to ascertain the standard weaning age for the population under study. In general, weaning is a gradual process, whereby the infant is breastfed and solid foods are progressively introduced into the diet. For this reason, one would not expect to find an exact age, but rather an age range, during which time, the infant is gradually introduced to solid food. In the United States, this range varies according to personal preference and the constraints faced by working mothers. Among specialists, there is little consensus about the proper weaning age (Dettwyler, 1994). The American Academy of Pediatrics recommends one year, while the World Health Organization and UNICEF recommend at least two years. To add to this confusion, ideas about weaning have changed during the last few decades. As few as twenty years ago, the prevailing opinion was that nursing is potentially harmful to the infant and could result in suffocation. Recently, health professionals and parents have come to the realization that there are both psychological and physical benefits for the infant and mother. Thus, teeth in this sample from older individuals could have entirely different weaning periods than those from younger individuals.

Linking peak age of occurrence to weaning stress is fraught with difficulties. Not only is it a culturally and temporally specific practice, it is also constrained by individual opinion. The event is continuous and assigning a finite age is problematic. Difficulties

also arise in choosing the correct weaning age. For example, if one implements the one year of age weaning suggestion from the American Academy of Pediatrics, then none of the teeth in this sample would be found to exhibit weaning stresses. However, if one utilizes the WHO and UNICEF weaning age of two years, half of the teeth in this sample exhibit a peak age of occurrence assignable to weaning stress.

The unpredictable nature of weaning in this sample is supported by Skinner and Goodman (1992) and Blakey and co-workers (1994). Blakey and co-workers advise that finding a direct relationship between weaning stress and the formation of hypoplasias is not readily discernible. In fact, the high frequencies of hypoplasias are most likely the result of a combination of environmental stresses, differences in susceptibility of enamel, and random factors.

Difficulties in assigning the peak age of occurrence to the effects of weaning stress in a recent population raises serious questions about its applicability to past populations. This dilemma raises doubts about the assignment of peak age of stress to weaning events by previous researchers.

Sensitivity of Wilson Bands

Although Wilson bands are more difficult to observe than macroscopic enamel surface defects, they are more sensitive and record precise episodes of stress over a longer period (see Chapter 3). The results of this study support this notion. From the 193 total teeth that were collected, Wilson bands were present in 78 teeth, or 40% of the teeth in

this study. Of these 78 teeth, only one had an identifiable surface defect. This enamel surface defect had a corresponding Wilson band present. The question that needs to be addressed by future research is the importance of obtaining the unique information provided by the analysis of microscopic defects. For many anthropologists, this is a goal of utmost importance, in which case, sacrificing some of the teeth is a worthwhile endeavor. This should only be done by someone that is experienced at making thin-sections, as the process is destructive.

With respect to Wilson band sensitivity between different tooth types and within a particular tooth, some interesting trends present themselves. The mandibular teeth demonstrated a greater inter-tooth sensitivity than the maxillary teeth. The few instances where the results of the Chi-square test deviated from the expected, it was the mandibular teeth which manifested these deviations. In the case of Wilson band manifestation and socioeconomic standing, the mandibular central incisors indicate a relationship between an individual's socioeconomic affiliation and an increase in Wilson band frequency. These findings are different than those reported by King and Wei (1992) for macroscopic white enamel opacities. They attest that as a group, the mandibular incisors mineralize early in childhood, and as a result are less affected than those that form later.

The fact that a majority of the mandibular canines and lateral incisors from European-Americans do not exhibit a high Wilson band frequency may be explained by the suggestion made by Smith and Peretz (1986). They hypothesize that a better blood supply

in the lower jaw might explain the low occurrence of hypoplastic defects among those teeth. This is an interesting speculation which requires further study.

Upon viewing intra-tooth occurrence of Wilson bands, some interesting outcomes were noted. Suga (1989) found that the lingual enamel is less vulnerable to the stresses recorded by enamel defects. However, in the present study, 25% of all teeth with Wilson bands, displayed a Wilson band on the lingual enamel surface, while the defect was absent on the labial surface of the same tooth. This is curious considering that the same physiological factors impact the enamel organ, which is responsible for enamel formation and mineralization. As noted by Suga, the enamel on the lingual surface of the anterior teeth is thinner. This being the case, one would assume that within a single tooth, which exhibits more enamel in one area than another, there would be a greater chance for the stress recorded by the defect to manifest itself on the region with the greatest amount of enamel.

The other trend has to do with the position of the Wilson band in the crown. The defects in this sample occur in greater frequencies in the upper portion of the crown. Only 20% of the Wilson bands were seen in the cervical half of the crown. This factor could be due to the age of the ameloblast. Goodman and Rose (1990) state,

> "when enamel secretion is disrupted the more recently activated ameloblasts, those that are most cervical, produce the least disrupted prisms. Physiologically, it is plausible to suggest that older ameloblasts might somehow become fatigued" (1990: 101).

Chapter 8

Conclusion

The purpose of this study was to investigate Wilson band frequencies in a contemporary adult population. Teeth were collected from public health and private practice oral surgeons. The two locales were divided into different socioeconomic classes. Those individuals who frequent private practice oral surgeons were assigned a higher socioeconomic standing than their public health counterparts. The collection process involved recording each individual's age, sex, and ethnicity. A Chi-square test was done for Wilson band frequency versus: sex, collection locale, and an individual's ethnicity.

While some results were expected, others were not. The Chi-square test on Wilson band frequency and sex revealed an expected result of no relationship between the two. However, unexpected results occurred in the Chi-square results for Wilson band frequency and collection locale. One would expect that those teeth from individuals with a low socioeconomic standing would exhibit higher frequencies of Wilson bands. This was true only in the case of the mandibular central incisors. However, when the raw data are considered, teeth collected from the public health department displayed Wilson bands in 49% of the teeth, while 24% of the teeth from the private practice oral surgeons manifested Wilson bands. These percentages indicate that a tooth from an individual who frequents the public health department is likely to have a Wilson band. One obvious implication is that individuals in a lower socioeconomic class are experiencing a greater

amount of physiological stress, which is recorded by the enamel defect. The inconsistency in the Chi-square test may stem from the division of teeth into tooth types, resulting in smaller numbers in each category, which are not discernible by the test as significant.

With respect to the results of the Chi-square test on Wilson band frequencies and the individual's ethnicity, the maxillary teeth indicate that there is no relationship between the two. However, as a group, the mandibular teeth had much lower probabilities and the mandibular canine and lateral incisor verify that a relationship exists between an individual's ethnicity and Wilson band frequency. This difference is attributed to the fact that a majority of the teeth from European-Americans do not manifest Wilson bands. This is confounded by the fact that the European-American sample consisted of teeth that were collected from private practice locations, which have a low overall occurrence of Wilson bands.

The mandibular canines and maxillary central incisors were selected for chronometric assessment. The age of occurrence for the incisors is between 1.5 and 4.0 years, and the age for the canines is between 1.5 and 4.0 years. The peak age of incidence is between 2.0 and 3.0 years for the incisors and between 1.5 to 2.5 years for the canines. Linking the peak age of incidence to the stresses suffered during weaning is problematic. Weaning times are not only culturally and temporally specific practices, they are also constrained by individual opinion. The event itself is continuous and assigning a finite age to such an occurrence is problematic. Difficulties in assigning the peak age of occurrence to the effects of weaning stress in a modern population raises serious questions about its

applicability to past populations. This dilemma raises doubts about the use of the weaning hypothesis by previous researchers.

Sensitivity within and between teeth indicate some interesting trends. The mandibular teeth demonstrated greater inter-tooth sensitivity than the maxillary teeth. When viewing intra-tooth sensitivity, the lingual enamel surface displayed Wilson bands in 25% of the teeth, without a corresponding defect on the labial surface of the same tooth. Most of the defect in this sample occur in greater frequencies in the upper portion of the crown. Only 20% of the Wilson bands were seen in the cervical half of the crown.

Although Wilson bands are more difficult to observe than macroscopic enamel surface defects, Wilson bands are more sensitive and record episodes of stress for a longer period of time. If it is the goal of the researcher to get at specific episodes of stress, then Wilson bands are more precise than surface defects.

The findings clearly demonstrate that microscopic enamel defects occur in contemporary populations in conjunction with increasing hardships. The unique information afforded anthropologists through the study of contemporary societies has the potential to enlighten our understanding of enamel defects, as well as our understanding of historic and prehistoric individuals.

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Appendices

Appendix Table A-1

Table A-1 Tooth Measurements

Number	Tooth Type	Crown Length (mm)	Root Length (mm)	M-D Crown Diameter (mm)	M-D Crown Cervix (mm)	L-L Diameter of Crown (mm)	L-L Crown at Cervix (mm)	CEJ Mesial (mm)
6148-1	8	11.7	9.6	9.15	7	7.31	6.53	2.31
7115-1	24	9.78	11.05	5.57	4.02	5.97	6.01	3.61
1568-1	23	7.66	11.97	5.29	3.76	6.01	5.98	3.59
3174-2	10	10.11	14.65	7.35	5.8	6.63	6	3.18
7817-2	9	11.61	12.51	8.81	6.74	6.98	6.24	2.5
2076-2	8	11.67	14.82	8.92	6.56	6.94	6.25	3.01
1528-2	7	10.55	14.27	7.3	5.2	6.89	6.24	3.17
4432-3	8	11.29	13.31	8.61	6.06	7.05	6.36	2.16
9352-3	9	11.58	12.9	8.66	6.43	6.98	6.27	2.89
8613-3	7	10.74	10.31	6.94	5.03	6.08	5.15	2.65
0948-3	11	10.39	17.71	7.49	6.39	7.62	7.18	2.24
4079-3	6	11.15	16.08	7.21	5.98	7.73	7.25	2.3
5125-4	24	7.76	10.75	5.18	2.98	5.39	4.68	N.A.
9008-4	23	7.04	14.22	5.57	3.39	6.02	5.37	2.22
6522-4	26	7.08	NA- FX Tooth	5.78	3.32	5.93	5.14	N.A. FX Tooth
0273-5	27	9.48	14.51	6.81	5.21	7.68	6.64	1.9
8528-6	9	10.85	11.76	7.84	6.85	7.71	7.02	2.7
7550-7	26	9.56	15.21	6.26	3.89	6.04	6	1.91
1009-8	23	9.57	15.88	6.39	4.83	5.57	7.05	2.71
3614-9	22	7.9	17.32	6.72	5.29	6.66	7.21	3.77
1687-10	27	9.93	14.94	6.5	5.43	6.63	6.8	4.87
6814-10	22	9.63	12.82	6.57	4.82	6.57	6.69	2.1
8142-11	22	10.9	17.61	7.19	6.55	7.09	8.56	5.48
7289-12	8	11.91	11.21	8.7	6.9	7.39	6.72	3.43
6534-12	9	12.62	12.22	8.44	7.24	7.42	6.83	3.57
3185-12	11	11.16	17.8	8.08	6.14	8.4	8.01	2.15
0800-12	6	10.34	16.9	8.17	6	8.65	8.07	2.89
4386-12	7	11.23	12.6	6.96	5.12	6.92	6.64	4
9317-12	10	10.54	13.21	6.87	5.4	7.05	6.57	3.51
8160-13	7	8.77	13.19	6.93	5.38	6.39	6.24	3.31
1550-14	8	10.74	11.84	8.04	7.08	6.42	5.93	3.29
0385-14	10	10.71	13.54	6.3	5.61	5.98	5.8	3.36
4111-15	6	9.87	19.61	8.3	6.59	9.27	8.71	2.9
0959-15	10	9.51	13.63	7.21	5.81	6.78	6.61	3.15
9071-16	22	12.1	17.2	7.58	6.76	8.26	8.19	3.22
9642-16	24	9.57	12.25	5.79	4.62	6.02	5.78	1.94
2726-16	23	10.08	13.41	6.69	5.08	6.5	6.41	1.81
9620-17	8	7.76	14.72	8.74	6.59	7.15	6.74	2.62
8618-17	7	7.3	15.34	6.61	4.94	5.86	6.51	3.48
7129-17	9	7.57	14.77	8.44	6.76	7.17	6.83	3.58
9089-17	10	8.1	15.21	6.61	4.98	6.24	6.15	3.51
7803-18	9	10.83	13.76	8.41	7.34	6.74	6.26	2.72
5986-18	6	9.23	15.72	7.84	6.53	7.62	7.7	3.14
7539-18	10	8.8	12.59	6.01	4.75	5.31	5.34	2.02
1964-19	26	9.9	16.19	5.49	4.34	5.84	6.22	2.36
0315-19	27	12.1	15.13	6.18	5.24	7.39	7.38	3.58
9420-20	22	11.17	17.86	6.62	5.75	8.08	8.2	3.01
8203-20	27	11.75	16.73	7.43	5.96	8.35	8.19	3.05
8748-20	26	9.92	13.18	6.13	4.72	6.74	6.4	2.78
3722-20	24	8.6	9.85	5.62	5.83	5.63	5.48	2.96
4149-21	23	8.45	12.6	5.82	4.11	6.19	5.79	1.31
4635-22	22	9.89	18.95	6.69	5.97	8.19	8.26	2.72
1108-22	23	7.39	15.28	6.06	4.76	6.21	6.12	2.38
6234-23	24	8.56	11.58	4.73	4.07	5.32	5.27	2.69
9502-24	7	9.81	15.21	6.36	5.2	6.02	5.89	3.79
1818-25	23	6.8	15.27	5.81	3.9	6.41	6.21	3.12
3713-26	27	12.37	18.64	7.42	7.52	8.93	8.61	3.95
8748-26	22	12.04	17.75	7.56	6.96	8.64	8.34	3.49
7513-27	24	9.37	14.81	6.35	5.11	6.25	6.24	2.58
1849-27	23	10.13	15.47	6.54	5.51	6.18	6.62	2.71
9559-28	22	11.09	17.62	6.49	5.94	7.7	7.58	3.45
8454-28	6	9.7	13.65	7.34	6.33	7.94	7.28	2.93
6893-29	25	9.36	14.19	5.87	4.62	6.38	6.2	2.17
6294-29	26	10.35	16.97	6.62	5.22	6.92	6.85	2.39
1474-30	22	8.85	19.98	6.68	5.92	7.9	7.8	3.56
5270-30	23	7.49	15.02	5.51	4.13	5.96	5.77	2.38
0833-30	24	6.2	13.91	4.46	4.11	5.42	5.16	2.3
7275-30	25	6.28	13.24	4.54	3.78	5.48	5.18	3.55
3689-30	26	7.26	15.43	5.49	5.07	5.9	5.82	3.5
8001-31	11	11.05	19.87	7.57	5.94	8.94	8.88	3.18
7402-32	11	11.54	14.17	6.65	5.54	6.2	6.55	2.82
3706-32	25	9.09	12.62	5.14	4.08	5.48	5.34	3.01
0348-33	11	11.45	19.16	8.13	6.16	8.01	7.95	2
7633-34	23	6.65	13.23	5.29	4.33	6.04	5.69	2.12
0931-34	22	6.94	18.4	6.15	5.25	7.05	7.83	3.04
1826-35	10	9.46	11.3	5.97	5.23	5.3	5.57	2.74
5659-36	26	7.93	14.77	5.05	4.32	5.87	5.4	2.46
1916-36	25	6.89	12.43	4.75	3.88	5.76	5.51	1.89
2742-37	26	6.76	10.76	4.76	4.42	5.54	5.23	2.03
3441-38	11	10.72	16.63	7.49	6.48	7.96	7.9	2.34

Table A-1 Tooth Measurements

5655-39	8	10.18	11.11	8.08	6.03	6.41	6.23	2.26
4654-39	7	9	13.55	6.22	4.91	5.69	5.54	3.34
4938-40	25	9.81	14.4	5.26	3.83	5.96	5.87	2.09
0631-40	26	10.62	15.01	5.42	3.77	6.01	5.97	1.64
6094-41	22	9.39	16.01	6.59	6.17	6.49	7.15	2.38
9232-41	25	9.85	11.67	5.45	4.4	5.35	5.49	3.63
7793-41	24	10.01	10.25	5.4	4.36	5.51	5.82	2.25
3810-41	23	10.34	10.5	5.92	4.53	5.25	5.81	3.04
3964-42	11	9.97	23.95	7.38	6.83	9.72	9.37	2.47
8405-43	11	10.36	17.14	6.99	6.45	8.07	7.7	2.21
4746-44	22	10.43	14.51	6.08	5.53	7.15	7.42	3.58
4332-44	27	9.32	13.44	6.27	5.94	7.41	7.47	2.92
6428-45	8	8.83	12.26	8.19	5.65	6.89	6.82	2.49
6684-45	9	10.01	11.4	8.31	6.67	7.14	6.69	2.14
7246-45	10	9.05	13.23	5.88	4.83	6.12	5.58	3.08
2103-45	7	NA FX Tooth	10.81	6.26	NA FX Tooth	5.06	NA FX Tooth	NA FX Tooth
9536-46	22	10.79	16.03	6.78	6.11	7.83	7.9	3.05
4971-46	26	7.72	10.42	5.52	4.84	6.23	5.81	2.14
8951-47	22	9.26	14.11	6.43	NA FX Tooth	6.28	NA FX Tooth	NA FX Tooth
1547-48	23	9.93	12.72	5.78	3.85	5.88	5.93	2.77
1212-48	24	7.87	10.95	5.37	3.89	5.34	5.13	NA FX Tooth
9386-48	25	9.13	10.82	5.27	4.47	5.96	5.42	3.38
6312-48	26	9.82	12.68	6.53	4.16	6.13	5.7	NA FX Tooth
0942-48	27	11.26	13.73	6.44	5.27	7.03	7.26	4.2
2329-49	22	11.52	16.12	7.08	5.98	7.83	7.67	2.32
7936-49	24	9.69	11.68	5.46	4.09	5.82	5.58	1.72
8101-49	25	9.84	12.47	5.32	4.45	6.13	5.6	2.31
9641-49	26	10.12	13.65	5.98	4.84	6.74	6.53	2.31
4054-49	27	11.29	16.21	6.93	6.27	7.66	7.41	2.92
7468-50	27	12.92	20.57	6.1	NA FX Tooth	7.51	8.83	3.05
3321-51	23	10.13	15.16	6.85	4.85	6.24	6.26	2.46
4281-51	24	6.96	11.61	4.6	3.77	5.66	5.84	3.2
6847-51	25	8.76	11.37	5.41	4.01	5.62	5.58	2.11
2461-51	26	8.58	15.9	6.67	4.76	6.36	6.28	1.78
1032-52	11	10.38	17.32	7.4	6.58	8.89	8.4	3.05
5362-52	10	10.45	15.22	6.13	5.14	6.44	5.95	2.34
9712-52	9	11.72	15.1	8.98	7.82	7.27	6.95	2.61
8275-52	8	11.73	15.98	9.07	7.6	7.62	6.93	3.66
9514-52	7	10.76	15.1	6.37	5.04	6.45	5.97	2.51
5472-53	22	11.41	17.94	7.52	5.8	8.83	9.03	2.96
2120-53	23	9.92	14.46	6.61	5.02	7.14	6.97	2.59
5448-53	24	9.08	13.07	6.33	4.3	6.75	6.51	2.5
8894-53	26	9.04	14.07	6.61	5.05	7.16	7.13	2.42
0030-53	27	10.89	16.99	7.62	6.8	9.15	9.04	2.95
9893-54	10	9.73	13.39	6.55	4.88	6.02	5.76	3.18
6357-54	9	11.82	14.34	9.36	6.72	6.68	6.18	3.19
7775-54	8	11.61	13.75	9.12	6.77	6.52	6.05	3.1
6945-54	7	9.81	13.68	6.76	5.39	6.04	5.67	1.77
4045-54	6	9.94	16.42	8.34	6.02	8.15	7.14	2.06
0357-55	7	10.59	13.36	7.06	NA DAMAGE	6.35	6.44	NA DAMAGE
3137-55	11	9.99	16.08	7.88	5.85	7.83	8.38	3.58
6182-56	22	11.04	18.49	7.01	5.89	8.74	8.89	NA DAMAGE
7049-57	26	9.2	13.9	5.36	4.5	6.51	6.5	3.53
3323-58	27	13.43	15.09	8	6.43	8.02	8.41	2.61
9105-58	25	10.49	11.22	5.57	3.73	6.12	5.78	1.9
6701-58	26	10.55	12.98	6.28	3.95	6.58	6.61	1.45
9738-59	26							
6176-60	22	11.41	16.35	6.82	5.89	7.73	7.75	3.99
1178-60	27	10.94	16.3	7.09	6.49	7.76	7.67	4.21
5066-61	8	8.46	13.46	6.89	5.18	6.29	6.02	2.92
1599-62	9	9.04	12.71	8.43	6.22	6.54	5.99	2.04
1189-63	11	9.84	18.8	6.93	6.32	6.98	7.57	3.65
7828-63	9	12.44	13.14	8.48	7.7	6.7	7.36	4.13
3138-63	8	10.62	12.49	8.92	8.05	6.51	7.47	4.32
1165-63	6	10.52	18.23	7.43	6.26	7.83	9.06	2.26
9133-64	25	9.86	13.06	5.28	3.81	5.48	5.62	2.65
9939-64	26	9.64	11.78	5.28	3.64	5.57	5.88	1.9
5764-64	27	10.19	14.89	5.89	4.31	6	6.28	2.44
2897-65	11	11.39	15.87	8.24	6.86	8.86	8.78	2.99
1855-66	22	10.3	15.12	6.52	5.46	6.85	6.97	2.84
3235-66	23	10.1	13.4	6.01	4.23	5.87	5.97	1.88
2152-66	24	9.46	11.81	5.57	4.31	5.39	5.38	1.37
1305-67	25	9.71	11.8	5.46	4.29	5.81	5.49	2.49
0665-67	26	9.91	14.05	6.19	5.03	5.64	5.78	1.82
1040-67	27	10.94	16.28	6.38	5.37	6.73	6.9	1.96
9758-68	10	9.8	13.43	6.21	4.75	6.14	6.12	2.86
6822-68	8	10.3	12.97	8.71	6.5	6.45	6.22	2.92
4813-68	6	9.63	17.04	7.19	5.58	7.58	7.06	2.15
5124-69	11	10.71	17.02	7.19	6.2	7.7	7.51	3.7
2355-69	10	10.28	14.05	6.53	5.25	5.32	5.43	3.04
6932-69	8	10.55	14.1	8.5	6.65	4.76	6.17	3.17
9267-69	7	9.15	14.23	6.3	4.9	5.44	5.31	2.88

Table A-1 Tooth Measurements

4634-70	22	10.16	17.26	6.53	5.5	7.08	7.39	1.8
5192-70	23	9.35	13.87	5.57	4.06	6.27	6.12	2.46
1389-70	24	5.9	11.18	4.48	4.44	5.68	5.43	1.61
2250-70	25	6.1	12.08	4.45	4.05	5.08	5.55	1.98
6368-70	26	9.1	12.38	5.51	4.38	5.7	6.01	2.6
6326-70	27	11.28	16.53	6.56	5.46	7.52	7.64	2.31
2389-71	26	8.62	6.99 (FX)	4.86	3.79	4.33	4.27	0.62
6493-71	25	8.32	5.46 (FX)	4.2	3.53	4.35	3.56	0.8
4184-72	9	11.01	12.09	8.69	7	7.08	6.45	3.94
4632-72	11	10.27	18.16	7.78	6.83	8.49	8.67	2.34
1108-73	23	14.62	11.64	5.7	4.38	6.83	6.86	3.7
5207-73	24	10.68	12.35	5.31	3.78	6.53	6.39	3.29
5727-73	25	11.25	13.12	5.42	3.96	5.88	6.14	2.28
2085-73	26	11.97	12.94	5.67	4.48	6.8	6.85	2.31
1563-73	10	11.44	10.53	6.38	5.56	6.82	6.25	2
9269-73	9	12.16	14.78	8.31	6.66	7.39	7.28	3.1
7761-73	8	12.59	13.75	8.49	6.81	7.03	7.18	2.91
3868-73	7	11.06	11.09	6.53	5.16	6.61	6.22	1.49
6525-74	9	10.8	12.05	8.34	7.56	7.11	6.96	3.27
3681-74	8	10.62	12.51	8.56	7.42	7.67	6.87	4.08
6439-74	7	9.18	12.26	6.32	4.28	5.52	5.81	2.64
8376-75	10	10.77	14.3	7.7	5.46	6.59	6.86	2.21
1438-75	9	10.11	14.67	8.54	6.15	6.97	7.55	2.16
5132-75	8	10.24	15.66	8.37	5.94	7.3	7.46	2.71
7247-75	6	13.02	16.35	8.15	5.91	8.48	8.22	2.21
5486-76	7	10.29	14.51	7.08	5.57	7.69	7.37	3.06
3952-76	6	9.81	18.63	7.03	6.94	9.04	9.15	1.94
8161-77	8	11.31	14.95	9.15	6.85	7.05	6.45	3.17
7586-77	7	10.34	13.77	6.99	4.96	5.79	5.74	2.81
9076-77	6	11.8	19.46	7.81	5.95	8.55	7.98	2.2
3441-78	23	9.56	14	5.84	4.53	6.62	6.66	2.43

Appendix Table A-2

Table A-2 Wilson Band Frequencies

Number	Individual	Sex	Age	Race	Tooth Type	Collection Locale	Practice Type	Wilson Band Count
6148-1	1	M	58	Euro-Amer	8	Hollander	Private	0
7115-1	1	M		Euro-Amer	24		Private	0
1568-1	1	M		Euro-Amer	23		Private	0
3174-2	2	F	49	Euro-Amer	10	Hollander	Private	0
7817-2	2	F		Euro-Amer	9		Private	0
2076-2	2	F		Euro-Amer	8		Private	0
1528-2	2	F		Euro-Amer	7		Private	0
4432-3	3	F	52	Euro-Amer	8	Hollander	Private	1
9352-3	3	F		Euro-Amer	9		Private	1
8613-3	3	F		Euro-Amer	7		Private	3
0948-3	3	F		Euro-Amer	11		Private	0
4079-3	3	F		Euro-Amer	6		Private	2
5125-4	4	M	56	Euro-Amer	24	Hollander	Private	0
9008-4	4	M		Euro-Amer	23		Private	0
6522-4	4	M		Euro-Amer	26		Private	0
0273-5	5	F	46	Euro-Amer	27	Stultz	Private	0
8528-6	6	M	48	Euro-Amer	9	Hollander	Private	0
7550-7	7	M	47	Euro-Amer	26	Hollander	Private	1
1009-8	8	F	36	Euro-Amer	23	Conroy	Public Health	2
3614-9	9	F	66	Euro-Amer	22	Conroy	Public Health	0
1687-10	10	M	56	Euro-Amer	27	Conroy	Public Health	0
6814-10	10	M		Euro-Amer	22		Public Health	0
8142-11	11	M	54	Euro-Amer	22		Public Health	4
7289-12	12	F	40	Euro-Amer	8	Wilson	Public Health	0
6534-12	12	F		Euro-Amer	9		Public Health	0
3185-12	12	F		Euro-Amer	11		Public Health	1
0800-12	12	F		Euro-Amer	6		Public Health	1
4386-12	12	F		Euro-Amer	7		Public Health	0
9317-12	12	F		Euro-Amer	10		Public Health	0
8160-13	13	M	50	Euro-Amer	7	Wilson	Public Health	1
1550-14	14	F	31	Euro-Amer	8	KCPHD	Public Health	1
0385-14	14	F		Euro-Amer	10		Public Health	2
4111-15	15	M	56	Af-Amer	6	KCPHD	Public Health	0
0959-15	15	M		Af-Amer	10		Public Health	3
9071-16	16	M	35	Af-Amer	22	KCPHD	Public Health	1
9642-16	16	M		Af-Amer	24		Public Health	1
2726-16	16	M		Af-Amer	23		Public Health	2
9620-17	17	M	55	Af-Amer	8	KCPHD	Public Health	2
8618-17	17	M		Af-Amer	7		Public Health	2
7129-17	17	M		Af-Amer	9		Public Health	1
9089-17	17	M		Af-Amer	10		Public Health	0
7803-18	18	F	40	Euro-Amer	9	KCPHD	Public Health	0
5986-18	18	F		Euro-Amer	6		Public Health	0
7539-18	18	F		Euro-Amer	10		Public Health	0
1964-19	19	F	24	Euro-Amer	26	KCPHD	Public Health	0
0315-19	19	F		Euro-Amer	27		Public Health	0
9420-20	20	M	43	Af-Amer	22	KCPHD	Public Health	3
8203-20	20	M		Af-Amer	27		Public Health	1
8748-20	20	M		Af-Amer	26		Public Health	4
3722-20	20	M		Af-Amer	24		Public Health	2
4149-21	21	F	41	Euro-Amer	23	KCPHD	Public Health	3
4635-22	22	M	67	Euro-Amer	22	KCPHD	Public Health	0
1108-22	22	M		Euro-Amer	23		Public Health	0
6234-23	23	M	25	Euro-Amer	24	Limchayseng	Private	0
9502-24	24	M	42	Euro-Amer	7	Limchayseng	Private	0
1818-25	25	M	68	Euro-Amer	23	Limchayseng	Private	0
3713-26	26	M	30	Euro-Amer	27	Limchayseng	Private	0
8748-26	26	M		Euro-Amer	22		Private	0
7513-27	27	M	30	Euro-Amer	24	Limchayseng	Private	0
1849-27	27	M		Euro-Amer	23		Private	1
9559-28	28	M	30	Euro-Amer	22	Limchayseng	Private	0
8454-28	28	M		Euro-Amer	6		Private	0
6893-29	29	M	30	Euro-Amer	25	Limchayseng	Private	0
6294-29	29	M		Euro-Amer	26		Private	0
1474-30	30	M	71	Euro-Amer	22	Limchayseng	Private	0
5270-30	30	M		Euro-Amer	23		Private	0
0833-30	30	M		Euro-Amer	24		Private	0
7275-30	30	M		Euro-Amer	25		Private	0
3689-30	30	M		Euro-Amer	26		Private	0

Table A-2 Wilson Band Frequencies

8001-31	31	F	48	Euro-Amer	11	MCDC	Public Health	0
7402-32	32	M	49	Hispanic	11	MCDC	Public Health	0
3706-32	32	M		Hispanic	25		Public Health	1
0348-33	33	M	48	Af-Amer	11	MCDC	Public Health	1
7633-34	34	F	71	Euro-Amer	23	MCDC	Public Health	2
0931-34	34	F		Euro-Amer	22		Public Health	1
1826-35	35	M	49	Hispanic	10	MCDC	Public Health	2
5659-36	36	F	85	Euro-Amer	26	MCDC	Public Health	1
1916-36	36	F		Euro-Amer	25		Public Health	4
2742-37	37	F	49	Asian	26	MCDC	Public Health	0
3441-38	38	M	46	Euro-Amer	11	MCDC	Public Health	0
5655-39	39	F	47	Euro-Amer	8	MCDC	Public Health	2
4654-39	39	F		Euro-Amer	7		Public Health	0
4938-40	40	M	23	Af-Amer	25	KCPHD	Public Health	3
0631-40	40	M		Af-Amer	26		Public Health	1
6094-41	41	F	45	Euro-Amer	22	KCPHD	Public Health	0
9232-41	41	F		Euro-Amer	25		Public Health	2
7793-41	41	F		Euro-Amer	24		Public Health	0
3810-41	41	F		Euro-Amer	23		Public Health	0
3964-42	42	M	50	Af-Amer	11	KCPHD	Public Health	2
8405-43	43	F	44	Euro-Amer	11	KCPHD	Public Health	1
4746-44	44	M	32	Euro-Amer	22	KCPHD	Public Health	1
4332-44	44	M		Euro-Amer	27		Public Health	0
6428-45	45	F	63	Euro-Amer	8	KCPHD	Public Health	1
6684-45	45	F		Euro-Amer	9		Public Health	2
7246-45	45	F		Euro-Amer	10		Public Health	0
2103-45	45	F		Euro-Amer	7		Public Health	0
9536-46	46	M	40	Af-Amer	22	KCPHD	Public Health	1
4971-46	46	M		Af-Amer	26		Public Health	2
8951-47	47	F	40	Euro-Amer	22	KCPHD	Public Health	1
1547-48	48	F	43	Euro-Amer	23	KCPHD	Public Health	2
1212-48	48	F		Euro-Amer	24		Public Health	1
9386-48	48	F		Euro-Amer	25		Public Health	1
6312-48	48	F		Euro-Amer	26		Public Health	1
0942-48	48	F		Euro-Amer	27		Public Health	1
2329-49	49	F	42	Af-Amer	22	KCPHD	Public Health	1
7936-49	49	F		Af-Amer	24		Public Health	4
8101-49	49	F		Af-Amer	25		Public Health	4
9641-49	49	F		Af-Amer	26		Public Health	3
4054-49	49	F		Af-Amer	27		Public Health	0
7468-50	50	M	40	Euro-Amer	27	KCPHD	Public Health	3
3321-51	51	M	49	Euro-Amer	23	KCPHD	Public Health	0
4281-51	51	M		Euro-Amer	24		Public Health	0
6847-51	51	M		Euro-Amer	25		Public Health	2
2461-51	51	M		Euro-Amer	26		Public Health	0
1032-52	52	M	45	Euro-Amer	11	KCPHD	Public Health	1
5362-52	52	M		Euro-Amer	10		Public Health	0
9712-52	52	M		Euro-Amer	9		Public Health	0
8275-52	52	M		Euro-Amer	8		Public Health	1
9514-52	52	M		Euro-Amer	7		Public Health	0
5472-53	53	M	44	Euro-Amer	22	KCPHD	Public Health	0
2120-53	53	M		Euro-Amer	23		Public Health	0
5448-53	53	M		Euro-Amer	24		Public Health	1
8894-53	53	M		Euro-Amer	26		Public Health	1
0030-53	53	M		Euro-Amer	27		Public Health	0
9893-54	54	F	61	Af-Amer	10	KCPHD	Public Health	0
6357-54	54	F		Af-Amer	9		Public Health	0
7775-54	54	F		Af-Amer	8		Public Health	0
6945-54	54	F		Af-Amer	7		Public Health	0
4045-54	54	F		Af-Amer	6		Public Health	0
0357-55	55	M	32	Euro-Amer	7	KCPHD	Public Health	1
3137-55	55	M		Euro-Amer	11		Public Health	0
6182-56	56	M	61	Euro-Amer	22	KCPHD	Public Health	0
7049-57	57	M	30	Euro-Amer	26	KCPHD	Public Health	2
3323-58	58	M	23	Euro-Amer	27	KCPHD	Public Health	0
9105-58	58	M		Euro-Amer	25		Public Health	0
6701-58	58	M		Euro-Amer	26		Public Health	0
9738-59	59	F	57	Af-Amer	26	KCPHD	Public Health	0
6176-60	60	M	28	Euro-Amer	22	KCPHD	Public Health	1
1178-60	60	M		Euro-Amer	27		Public Health	1

Table A-2 Wilson Band Frequencies

5066-61	61	F	38	Af-Amer	8	KCPHD	Public Health	1
1599-62	62	F	49	Euro-Amer	9	KCPHD	Public Health	0
1189-63	63	M	20	Euro-Amer	11	KCPHD	Public Health	0
7828-63	63	M		Euro-Amer	9		Public Health	0
3138-63	63	M		Euro-Amer	8		Public Health	0
1165-63	63	M		Euro-Amer	6		Public Health	0
9133-64	64	M	39	Euro-Amer	25	KCPHD	Public Health	0
9939-64	64	M		Euro-Amer	26		Public Health	0
5764-64	64	M		Euro-Amer	27		Public Health	0
2897-65	65	M	48	Euro-Amer	11	KCPHD	Public Health	1
1855-66	66	F	29	Euro-Amer	22	KCPHD	Public Health	0
3235-66	66	F		Euro-Amer	23		Public Health	0
2152-66	66	F		Euro-Amer	24		Public Health	2
1305-67	67	F	29	Euro-Amer	25	KCPHD	Public Health	3
0665-67	67	F		Euro-Amer	26		Public Health	2
1040-67	67	F		Euro-Amer	27		Public Health	0
9758-68	68	M	34	Euro-Amer	10	KCPHD	Public Health	0
6822-68	68	M		Euro-Amer	8		Public Health	0
4813-68	68	M		Euro-Amer	6		Public Health	0
5124-69	69	M	55	Euro-Amer	11	KCPHD	Public Health	0
2355-69	69	M		Euro-Amer	10		Public Health	0
6932-69	69	M		Euro-Amer	8		Public Health	0
9267-69	69	M		Euro-Amer	7		Public Health	0
4634-70	70	F	33	Euro-Amer	22	KCPHD	Public Health	0
5192-70	70	F		Euro-Amer	23		Public Health	1
1389-70	70	F		Euro-Amer	24		Public Health	2
2250-70	70	F		Euro-Amer	25		Public Health	2
6368-70	70	F		Euro-Amer	26		Public Health	1
6326-70	70	F		Euro-Amer	27		Public Health	1
2389-71	71	F	39	Euro-Amer	26	Hollander	Private	0
6493-71	71	F		Euro-Amer	25		Private	0
4184-72	72	M	43	Euro-Amer	9	Hollander	Private	2
4632-72	72	M		Euro-Amer	11		Private	0
1108-73	73	F	40	Euro-Amer	23	Hollander	Private	1
5207-73	73	F		Euro-Amer	24		Private	0
5727-73	73	F		Euro-Amer	25		Private	0
2085-73	73	F		Euro-Amer	26		Private	0
1563-73	73	F		Euro-Amer	10		Private	3
9269-73	73	F		Euro-Amer	9		Private	0
7761-73	73	F		Euro-Amer	8		Private	0
3868-73	73	F		Euro-Amer	7		Private	1
6525-74	74	F	47	Asian	9	Hollander	Private	0
3681-74	74	F		Asian	8		Private	0
6439-74	74	F		Asian	7		Private	0
8376-75	75	F	43	Af-Amer	10	Memphis	Public Health	0
1438-75	75	F		Af-Amer	9		Public Health	2
5132-75	75	F		Af-Amer	8		Public Health	0
7247-75	75	F		Af-Amer	6		Public Health	0
5466-76	76	M	61	Af-Amer	7	Knoxville	Private	1
3952-76	76	M		Af-Amer	6		Private	0
8161-77	77	M	45	Euro-Amer	8	Knoxville	Private	4
7586-77	77	M		Euro-Amer	7		Private	1
9076-77	77	M		Euro-Amer	6		Private	0
3441-78	78	M	45	Euro-Amer	23	Knoxville	Private	0

Appendix Table A-3

Maxillary Canine By Collection Locale

StatMost for Windows Wednesday, May 20, 1998 12:54:01 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	4.0000	1.0000	5.0000	
	3.4091	1.5909	22.73%	Private
	0.5909	-0.5909		Practice
	0.0024	0.0052	0.0076	
2	11.0000	6.0000	17.0000	
	11.5909	5.4091	77.27%	Public
	-0.5909	0.5909		Health
	0.0007	0.0015	0.0022	
TOTAL	15.0000	7.0000	22.0000	
	68.18%	31.82%	100.00%	

Chi-Square = 0.009860
Degree of Freedom = 1
Probability = 0.920902

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Maxillary Lateral Incisor By Collection Locale

StatMost for Windows Wednesday, May 20, 1998 12:56:05 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	3.0000	4.0000	7.0000	
	4.1364	2.8636	31.82%	Private
	-1.1364	1.1364		Practice
	0.0979	0.1414	0.2393	
2	10.0000	5.0000	15.0000	
	8.8636	6.1364	68.18%	Public
	1.1364	-1.1364		Health
	0.0457	0.0660	0.1117	
TOTAL	13.0000	9.0000	22.0000	
	59.09%	40.91%	100.00%	

Chi-Square = 0.350997
Degree of Freedom = 1
Probability = 0.553549

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Maxillary Central Incisor By Collection Locale

StatMost for Windows Wednesday, May 20, 1998 12:59:15 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	5.0000	3.0000	8.0000	
	5.0909	2.9091	36.36%	Private
	-0.0909	0.0909		Practice
	0.0329	0.0575	0.0904	
2	9.0000	5.0000	14.0000	
	8.9091	5.0909	63.64%	Public
	0.0909	-0.0909		Health
	0.0188	0.0329	0.0517	
TOTAL	14.0000	8.0000	22.0000	
	63.64%	36.36%	100.00%	

Chi-Square = 0.142060
Degree of Freedom = 1
Probability = 0.706242

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Mandibular Canine By Collection Locale

StatMost for Windows Wednesday, May 20, 1998 1:02:40 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	4.0000	0.0000	4.0000	
	2.3704	1.6296	14.81%	Private
	1.6296	-1.6296		Practice
	0.5383	0.7830	1.3214	
2	12.0000	11.0000	23.0000	
	13.6296	9.3704	85.19%	Public
	-1.6296	1.6296		Health
	0.0936	0.1362	0.2298	
TOTAL	16.0000	11.0000	27.0000	
	59.26%	40.74%	100.00%	

Chi-Square = 1.551183
Degree of Freedom = 1
Probability = 0.212961

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Mandibular Lateral Incisor By Collection Locale

StatMost for Windows Wednesday, May 20, 1998 1:06:13 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	8.0000	2.0000	10.0000	
	5.4545	4.5455	30.30%	Private
	2.5455	-2.5455		Practice
	0.7670	0.9205	1.6875	
2	10.0000	13.0000	23.0000	
	12.5455	10.4545	69.70%	Public
	-2.5455	2.5455		Health
	0.3335	0.4002	0.7337	
TOTAL	18.0000	15.0000	33.0000	
	54.55%	45.45%	100.00%	

Chi-Square = 2.421196
Degree of Freedom = 1
Probability = 0.119704

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Mandibular Central Incisor By Collection Locale

StatMost for Windows Wednesday, May 20, 1998 1:11:26 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	8.0000	0.0000	8.0000	
	3.8261	4.1739	34.78%	Private
	4.1739	-4.1739		Practice
	3.5278	3.2338	6.7616	
2	3.0000	12.0000	15.0000	
	7.1739	7.8261	65.22%	Public
	-4.1739	4.1739		Health
	1.8815	1.7247	3.6062	
TOTAL	11.0000	12.0000	23.0000	
	47.83%	52.17%	100.00%	

Chi-Square = 10.367787
Degree of Freedom = 1
Probability = 0.001282

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Maxillary Canine by Sex

StatMost for Windows Tuesday, May 19, 1998 11:32:28 PM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	12.0000	4.0000	16.0000	
	10.9091	5.0909	72.73%	
	1.0909	-1.0909		Males
	0.0320	0.0686	0.1006	
2	3.0000	3.0000	6.0000	
	4.0909	1.9091	27.27%	
	-1.0909	1.0909		Females
	0.0854	0.1829	0.2683	
TOTAL	15.0000	7.0000	22.0000	
	68.18%	31.82%	100.00%	

Chi-Square = 0.368849
Degree of Freedom = 1
Probability = 0.543632

StatMost Report Created by Anthropology, University of Tennessee

Maxillary Lateral Incisors By Sex

StatMost for Windows Tuesday, May 19, 1998 11:53:17 PM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr	Without_Wi	With_Wilso	TOTAL	
1	5.0000	6.0000	11.0000	
	6.5000	4.5000	50.00%	
	-1.5000	1.5000		Males
	0.1538	0.2222	0.3761	
2	8.0000	3.0000	11.0000	
	6.5000	4.5000	50.00%	
	1.5000	-1.5000		Females
	0.1538	0.2222	0.3761	
TOTAL	13.0000	9.0000	22.0000	
	59.09%	40.91%	100.00%	

Chi-Square = 0.752137
Degree of Freedom = 1
Probability = 0.385801

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Maxillary Central Incisors By Sex

StatMost for Windows Wednesday, May 20, 1998 12:38:37 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	6.0000	3.0000	9.0000	
	5.7273	3.2727	40.91%	
	0.2727	-0.2727		Males
	0.0090	0.0158	0.0248	
2	8.0000	5.0000	13.0000	
	8.2727	4.7273	59.09%	
	-0.2727	0.2727		Females
	0.0062	0.0109	0.0172	
TOTAL	14.0000	8.0000	22.0000	
	63.64%	36.36%	100.00%	

Chi-Square = 0.041972
Degree of Freedom = 1
Probability = 0.837673

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Mandibular Canines By Sex

StatMost for Windows Wednesday, May 20, 1998 12:44:20 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	10.0000	6.0000	16.0000	
	9.4815	6.5185	59.26%	
	0.5185	-0.5185		Males
	3.617E-005	5.261E-005	8.878E-005	
2	6.0000	5.0000	11.0000	
	6.5185	4.4815	40.74%	
	-0.5185	0.5185		Females
	5.261E-005	7.652E-005	0.0001	
TOTAL	16.0000	11.0000	27.0000	
	59.26%	40.74%	100.00%	

Chi-Square = 0.000218
Degree of Freedom = 1
Probability = 0.988222

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Mandibular Lateral Incisors By Sex

StatMost for Windows Wednesday, May 20, 1998 12:48:31 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr	Without_Wi	With_Wilso	TOTAL	
1	11.0000	7.0000	18.0000	
	9.8182	8.1818	54.55%	Males
	1.1818	-1.1818		
	0.0473	0.0568	0.1042	
2	7.0000	8.0000	15.0000	
	8.1818	6.8182	45.45%	Females
	-1.1818	1.1818		
	0.0568	0.0682	0.1250	
TOTAL	18.0000	15.0000	33.0000	
	54.55%	45.45%	100.00%	

Chi-Square = 0.229167
Degree of Freedom = 1
Probability = 0.632142

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Mandibular Central IncisorsBy Sex

StatMost for Windows Wednesday, May 20, 1998 12:51:29 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	8.0000	6.0000	14.0000	
	6.6957	7.3043	60.87%	
	1.3043	-1.3043		Males
	0.0966	0.0886	0.1852	
2	3.0000	6.0000	9.0000	
	4.3043	4.6957	39.13%	Females
	-1.3043	1.3043		
	0.1503	0.1378	0.2881	
TOTAL	11.0000	12.0000	23.0000	
	47.83%	52.17%	100.00%	

Chi-Square = 0.473289
Degree of Freedom = 1
Probability = 0.491478

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Maxillary Canine By Ethnicity

StatMost for Windows Wednesday, May 20, 1998 1:19:18 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	4.0000	2.0000	6.0000	
	4.0000	2.0000	28.57%	African
	0.0000	0.0000		American
	0.0625	0.1250	0.1875	
2	10.0000	5.0000	15.0000	
	10.0000	5.0000	71.43%	European
	0.0000	0.0000		American
	0.0250	0.0500	0.0750	
TOTAL	14.0000	7.0000	21.0000	
	66.67%	33.33%	100.00%	

Chi-Square = 0.262500
Degree of Freedom = 1
Probability = 0.608408

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Maxillary Lateral Incisor By Ethnicity

StatMost for Windows Wednesday, May 20, 1998 1:22:07 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr	ROW	Without_Wi	With_Wilso	TOTAL	
	1	3.0000	2.0000	5.0000	
		3.0000	2.0000	25.00%	African
		-4.44E-016	4.44E-016		American
		0.0833	0.1250	0.2083	
	2	9.0000	6.0000	15.0000	
		9.0000	6.0000	75.00%	European
		0.0000	0.0000		American
		0.0278	0.0417	0.0694	
	TOTAL	12.0000	8.0000	20.0000	
		60.00%	40.00%	100.00%	

Chi-Square = 0.277778
Degree of Freedom = 1
Probability = 0.598161

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Maxillary Central Incisors By Ethnicity

StatMost for Windows Wednesday, May 20, 1998 1:25:39 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	2.0000	2.0000	4.0000	
	2.4762	1.5238	19.05%	African
	-0.4762	0.4762		American
	0.0002	0.0004	0.0006	
2	11.0000	6.0000	17.0000	
	10.5238	6.4762	80.95%	European
	0.4762	-0.4762		American
	5.387E-005	8.754E-005	0.0001	
TOTAL	13.0000	8.0000	21.0000	
	61.90%	38.10%	100.00%	

Chi-Square = 0.000742
Degree of Freedom = 1
Probability = 0.978263

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Mandibular Canine By Ethnicity

StatMost for Windows Wednesday, May 20, 1998 1:28:13 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	0.0000	4.0000	4.0000	
	2.3704	1.6296	14.81%	African
	-2.3704	2.3704		American
	1.4758	2.1467	3.6225	
2	16.0000	7.0000	23.0000	
	13.6296	9.3704	85.19%	European
	2.3704	-2.3704		American
	0.2567	0.3733	0.6300	
TOTAL	16.0000	11.0000	27.0000	
	59.26%	40.74%	100.00%	

Chi-Square = 4.252517
Degree of Freedom = 1
Probability = 0.039192

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Mandibular Lateral Incisor By Ethnicity

StatMost for Windows Wednesday, May 20, 1998 1:33:53 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSq / ROW	Without_Wi	With_Wilso	TOTAL	
1	1.0000	5.0000	6.0000	
	3.1875	2.8125	18.75%	African
	-2.1875	2.1875		American
	0.8934	1.0125	1.9059	
2	16.0000	10.0000	26.0000	
	13.8125	12.1875	81.25%	European
	2.1875	-2.1875		American
	0.2062	0.2337	0.4398	
TOTAL	17.0000	15.0000	32.0000	
	53.13%	46.87%	100.00%	

Chi-Square = 2.345701
Degree of Freedom = 1
Probability = 0.125629

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Mandibular Central Incisor By Ethnicity

StatMost for Windows Wednesday, May 20, 1998 1:36:11 AM

Contingency Table (Chi-Square) Results

Count / Expect / Res / ChiSqr / ROW	Without_Wi	With_Wilso	TOTAL	
1	0.0000	4.0000	4.0000	
	2.0000	2.0000	18.18%	African
	-2.0000	2.0000		American
	1.1250	1.1250	2.2500	
2	11.0000	7.0000	18.0000	
	9.0000	9.0000	81.82%	European
	2.0000	-2.0000		American
	0.2500	0.2500	0.5000	
TOTAL	11.0000	11.0000	22.0000	
	50.00%	50.00%	100.00%	

Chi-Square = 2.750000
Degree of Freedom = 1
Probability = 0.097254

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VITA

Lise Mifsud graduated from San Marin High School in Novato, California. In 1993 she graduated Phi Beta Kappa with a Bachelor of Arts degree in Anthropology from the University of California at Berkeley. In 1998 she received her Master of Arts degree in Anthropology from the University of Tennessee at Knoxville.