PROFILING METABOLIC STRESS IN MEDIEVAL DENMARK: AN ANALYSIS OF INTERNAL AND EXTERNAL ENAMEL DEFECTS

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ABSTRACT

MARIANNE E. REEVES: Profiling Metabolic Stress in Medieval Denmark:
An Analysis of Internal and External Enamel Defects
(Under the direction of Dale Hutchinson)

The purpose of this study was to assess the prevalence of both types of enamel defects and to determining the timing of each, based on a population-specific model of permanent mandibular canine crown growth (after Simpson, 1999). Analysis of n=410 canine teeth from two Catholic friary cemeteries in medieval Denmark, the Black and Gray Friars, revealed that 96% of individuals had 1 or more surface defects. In a subsample of n=63 thin-sectioned canines, only 25% showed evidence of 1 or more pathological striae. The population model revealed a non-linear pattern of canine crown growth, with growth slowing as the cervix was reached. Duration of crown growth was found to be 50.6 months, shorter than in some previous estimates for modern humans. The peak prevalence of microdefects occurred between 20 and 40 months, overlapping with surface defects, but also occurring in infancy prior to one year of age. Overall, it was found that: (1) PS did occur in infancy, expanding the stress profile window to include the earliest period of canine growth, (2) PS prevalence was unexpectedly lower and surface defects, higher, than in other archaeological populations, and (3) comparison of the distance functions (representing enamel growth geometry) were found to be significantly different from Simpson's (1999) equation. While the primary contribution of the study was methodological in assessing hidden cuspal enamel for defects and using a population-specific model for timing those defects, the methods allow greater understanding about the stressors that affected childhood

growth in an extremely tumultuous time in Danish history. Likely to have been critical in causing acute and chronic stress in Denmark are not only waves of infectious disease, but also the periodic famines throughout the medieval period. Weaning stress (including weanling diarrhea) is also a likely candidate for growth disruption in the samples analyzed here, as weaning represents a significant dietary transition in infants and toddlers.

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CHAPTER I

INTRODUCTION

DEFINING THE RESEARCH PROBLEM

The purpose of this dissertation is to determine how metabolic stress impacted early childhood growth in a sample of skeletal individuals from medieval Denmark (ca. A.D. 1250-1539). Studies of growth disruption provide a window onto the variation in the human response to metabolic stress and better insight into its etiologies. The consequence of the morbidity and mortality of medieval children went beyond loss to their immediate families; economic loss was felt as well, as children in towns left their households as early as age 10 years for full-time labor as servants or apprentices (Orme, 2001; Hanawalt, 1993). Moreover, evidence from clinical and bioarcheological studies suggests that stress impacts not only the health and well-being during childhood, but it continues to negatively affect physiological resiliency into adulthood (Swärdstedt, 1966; Cook and Buikstra, 1979; Rudney, 1983; Goodman and Armelagos, 1988; Goodman, 1991; Simpson et al., 1990; Duray, 1996). Disease and nutritional stress are often implicated in the etiology of non-specific stress markers in human skeletal material (see Larsen, 1999). The medieval children in the town of Odense, DK lived in conditions that included a high residential population density, a rudimentary sewage disposal system that ran along town streets, shared water sources and limited access to hot water for cleaning and bathing, and a pre-antibiotic system of medical care – all of which contributed to the likelihood of infectious disease transmission. Nutritional stress was also a threat, in the form of the Great Famine and numerous smaller

famines in the 14th century, which eventually led to a shift in diet to a greater dependence on meat and dairy products (Skaarup, 1978).

The primary contribution of this research is methodological. Specifically, "hidden" cuspal enamel is assessed for microdefects of enamel, along with the macrodefects visible from the enamel surface. This is crucial because it illuminates a period in early childhood that is often left out of bioarchaeolgoical studies of dental stress. This study also makes use of a population-specific model for enamel growth after Simpson (1999) to overcome issues error in enamel growth and disruption timing. But it does so in a way that relies on a small sample size for the model and minimizes destruction to dental samples.

While these methodological issues are important in themselves, they are more significant for allowing accurate assessment of growth disruption in the Odense friary samples. This biological data gives us valuable information about childhood health in Denmark in an extremely tumultuous time during Danish history with numerous infectious diseases, including the Black Death, multiple famines, farm abandonments, and the social and economic upheavals that resulted from all of these disasters.

Teeth are particularly useful for profiling stress in skeletal material because, unlike bone, teeth do not remodel after growth is complete. Dental enamel permanently records crown growth in discrete increments and, by extension, serves as a biomarker for stressful events that interrupt growth. Among the most common indicators of growth disruption in enamel are external defects called hypoplasias and internal defects known as pathological striae of Retzius (PS), also known as accentuated striae (AS) or brown striae of Retzius (BSR). Hypoplasias area areas of thinner enamel, visible on the surface of the tooth as a pit or furrow; pathological striae are disruptions of enamel prism structure visible only in the

enamel internally. Quantifying the prevalence and timing of these two types of defects provides a gross measure of the level of non-specific metabolic stress experienced by children and, in turn, gives a proxy for the overall stress level of the population (Rose et al., 1985; Goodman and Rose, 1990; Wright, 1997; Simpson, 1999). The research presented here tests suppositions that young medieval children: (1) experienced comparatively high levels of both acute and chronic metabolic stress resulting in the disruption of normal enamel growth, (2) that disruption occurred commonly in infancy, expanding the age range of susceptibility to stress to include the earliest years of childhood, and (3) that the pattern of enamel growth and pathological disruption is unique to the study sample and not indicative of a pan-human pattern of normal growth and stress response. If these hypotheses are supported, they will provide clearer insights into not only the etiology of the stressors affecting medieval children, but also into the range of variation in human crown growth and disruption. A more complete exposition of these hypotheses and presuppositions that support them follows.

Hypothesis 1

The first hypothesis was that children experienced comparatively high levels of enamel growth disruptions as a result of the metabolic stressors commensurate with an aggregate urban lifestyle.

Presupposition 1

The first presupposition was that the prevalence of enamel growth defects would be positively correlated with environmental stress factors, including nutritional and disease stress. The vast majority of disruptions to enamel growth that result in hypoplastic defects and pathological striae are non-specific stress indicators, meaning that the exact etiology of the stressor cannot be determined. However, both types of defects have been positively correlated with disease and nutritional stress in clinical and bioarchaeological studies.

Disease and undernutrition are often products of poor socioeconomic status in living groups, and a similar connection has been demonstrated in archaeological populations.

Growth disturbance as measured by prevalence of pathological striae of Retzius has also been associated in skeletal samples with infectious disease. In a study of a skeletal series representing the Meroitic (100 B.C. – A.D. 300) and X-Group (A.D. 300-600) cultural horizons in Lower Nubia, Rudney (1983) found a negative correlation between age at death and PS prevalence, indicating that those who died young were more susceptible to early growth disturbance than those surviving to adulthood. Additionally, childhood health appears to have improved over time from the Meoritic to X-Group periods, perhaps due to changes in political structure, consumption of tetracyclines, or changes in irrigations techonology that affected the prevalence of helminthic infections. Wright (1990) hypothesized that the increase in prevalence of pathological striae and shallow enamel hypoplasias from the Postclassic to Historic period Mayan skeletal groups is related to an increase in parasitic pathogens in the Historic period.

Like disease stress, hypoplasias and pathological striae have been positively correlated with malnutrition in clinical and skeletal studies. Goodman et al. (1991) argued that undernutrition is causally linked to enamel hypoplasia formation, based on their study defects in Aztec adolescents living in Mezonteopan, Mexico. The authors found linear enamel hypoplasia (LEH) prevalence in the range of 65% to 84% in children without nutritional supplementation. Infante (1974) found that between 19% and 39% of a sample of manlnourished Apache Indian children showed hypoplastic defects. An increase in the prevalence of pathological striae has been linked to changes in diet that include the the shift from hunting and gathering diet to primarily agriculture foods (Molnar and Ward 1975; Rose

et al. 1979; Simpson 1999). Rose et al. (1978) predicted that stress, as indicated by pathological striae, infectious disease lesions, and lower-age-at death, would increase in prehistoric skeletal samples over time in proportion to increasing dependence on maize agriculture, higher residential population densities, increased trade, and greater complexity of social organization. The three skeletal samples from the prehistoric Dickson and Gibson Mounds, Illinois, representing the Middle Woodland, Late Woodland, and Mississippian time periods, showed pathological stria prevalences of 10.3%, 21.4%, and 40.0% respectively, in support of the authors hypotheses. Simpson (1999) found that stress as measured by microstructural defects increased from the prehistoric to the mission period in northern Florida, in prevalences of 48% (summed prehistoric), 54% (early contact), and 83% (mission samples).

Presupposition 2

The second presupposition was that the distribution of hypoplasias and pathological striae would often be different, indicating that the defects have different etiologies and represent chronic and acute stress, respectively. Studies by Condon (1981), Rose et al. (1985), Wright (1990), and Simpson (1999) found that the distribution of enamel hypoplasias and pathological striae differed significantly in skeletal samples and, therefore, likely represent different stress etiologies. Rose (1977) examined the relationship between hypoplasias and pathological striae in permanent canines from the prehistoric Dickson Mounds site in Illinois. He found that only one hypoplasia in 26 defects was associated with a pathological stria, an extremely low co-occurrence. Conversely, in a study of a prehistoric Native American sample from the Libben Site, Ohio, Condon (1981) found that 74% of pathological striae in mandibular canines co-occurred with hypoplasias. Because co-occurrence was not absolute, Condon concluded that hypoplasias represent chronic, long-

term stress while pathological striae were indicative of shorter, more acute periods of stress. Wright (1990) found similar results, with hypoplasias and Wilson bands co-occurring at a rate of 23% (and Wilson bands co-occuring with hypoplasias at a rate of 51%) in Mayan samples from Lamanai, Belize. Wright notes that morphological differences in the defects suggest different etiologies, although Rose et al. (1985) concluded that the relationship between the defect types is not always clear. In terms of structure, pathological striae are marked by a brief disruption in prism structure lasting several days; while hypoplasias are produced by a slowing of ameloblast secretion, resulting in thinner enamel with normal imbricational striae converging in the defect (although see Hillson and Bond, 1997). Condon and Rose (1992), Goodman and Rose (1992) and Goodman and Armelagos (1985a,b) argue that that differences in susceptibility to dental defects occurs both within and between tooth types. Goodman and Armelagos (1985a) suggest that biological gradients in susceptibility to disruptions of ameloblasts exist between tooth types. Specifically the expression of hypoplastic defects may be affected by enamel prism length and direction, which differs within and between teeth. Similarly, Witzel and authors (2008) argue for a threshold model of increasing impairment to secretory ameloblasts in response to stress in a medieval German skeletal assemblage, and they espouse the acute versus chronic model of microdefect stress response. And Thomas (2003) found that accentuated striae nearly always preceded enamel hypoplasia in dental sample from medieval Tirup. She related this sequence to an initial stress event that compromised (or killed) ameloblasts, leading to a longer-term stress event as reflected by enamel hypoplasia. Most recently, Guatelli-Steinberg and authors (2012) have analyzed variation in enamel hypoplasia expression in great apes in terms of lateral enamel formation time and the angles of striae with the enamel surface. They concluded that striae

angles do influence hypoplasia expression in great apes and humans (acute angles resulting in fewer identifiable surface defects).

Presupposition 3

The third presupposition was that the conditions of aggregation concomitant with urbanization would be associated with an increase in stress prevalence; these conditions were present in medieval Odense. Larsen and colleagues have clearly demonstrated that an increase in aggregation associated with the adoption of farming and increased consumption of processed foods and refined carbohydrates, along with decreased physical activity, results in a decrease in health status. This evidence is intimately connected to evidence for increase in skeletal pathologies resulting from infectious diseases (Hutchinson and Larsen, 1990; Larsen, 1997).

Aggregate town life is an extreme on the continuum of population aggregation.[1] The relationship between increased population size and distribution and a decrease in overall health has been demonstrated in archaeological populations from Europe, and North America. Urban environments exacerbate certain types of disease proliferation and transmission (Betsinger, 2007; Roberts, 2000). Archaeological remnants of Odense's walls and structures have helped to establish that the town was walled, separating it from the surrounding countryside. Residents lived in houses in close proximity to one another, often also in close proximity to with animals and with an omnipresent risk of water contamination**.** Perhaps even more informative is the historical data on periodic famine, and infectious disease like the Black Plague, which arrived in Denmark in A.D. 1348 and was succeeded by

[1] Towns in medieval Scandinavia are estimated to have residential populations equal to or greater than 1000, and possibly between 5000 and 10,000 individuals (Helle, 1993).

numerous smaller outbreaks. Other infectious disease such as tuberculosis, smallpox (Hays, 1998), and leprosy (Anderson, 2000) were also prevalent during the time period.

Making a dietary argument for a medieval town is more difficult because towns acted as markets for local farmers as well as for the importation of foreign goods (*e.g.*, spices and fruits). While the Danish burghers' diet was grain-based, a variety of foods was available to those who had access to them. The diets of infants and young children were certainly milk and grain-based and are detailed in Chapter 5.

Hypothesis 2

The second hypothesis was that acute stress episodes resulting in growth disruption occurred in early infancy. This hypothesis is based on identifying pathological striae in "hidden" cuspal enamel. Evidence supporting this hypothesis would expand the age range of susceptibility to stress to include the earliest years of childhood.

Presupposition 1

The first presupposition of Hypothesis 2 was that, when hidden enamel is included in the sample and scored for defects, the timing of defects can include the first year. Several bioarchaeological studies have taken into account the enamel hidden by successive layers of enamel during cusp development, but not within the context of an incremental, non-linear crown growth model (see Thomas, 2003). Goodman and Song (1999), Wright (1997), and Suga (1997) account for hidden layers of cuspal enamel by adding several months to their hypoplasia chronologies to account for the initial period of hidden enamel development. Wright (1997) estimated that hidden (and unscorable) enamel accounted for a period of 1 year in Mayan dental samples, while Goodman and Song (1999) noted a period of 10 months for hidden enamel in mandibular canines. Goodman and Song (1999) note that the effect of the hidden enamel correction was to increase the mean age at LEH formation from 3 months

to approximately 4.5 months. It is important to note that the growth model used in these studies is based on equal divisions of crown height corresponding to chronological ages, inferring that crown growth is a linear process.

A tooth crown develops from the cuspal end first, and growth proceeds towards the cervical end. The first-forming, cuspal enamel is buried in the deepest enamel layers; thus, defects that occur in cuspal enamel are representative of metabolic stress that occurred in that early period of development (Hillson, 1996). Many studies of dental defects disregard cuspal enamel because seeing the hidden increments requires thin-sectioning (a destructive process). Hypoplasias, as surface defects, will only represent stress that occurred from the time that striae of Retzius (SOR) reach the surface (as perikymata) during crown extension. The earliest stress during growth will not be recorded on the enamel surface, necessitating analysis of the internal structure of enamel surrounding the dentin horn. Using hypoplasias and pathological striae together covers the entire time of growth subject to disruption – hypoplasias alone leave out the period of "hidden" cuspal enamel. The nature of tooth geometry dictates that the first layers of enamel laid down in the cusp are covered over completely by successive layers of enamel. Further down on the crown, the layers are not hidden; they reach the surface as countable growth markers (Hillson, 1996; Hillson and Bond, 1997).

Cook (1981) found that accentuated striae and prism disruption occur most commonly in infants between the ages of 6 and 24 months, a time period during which children may be undergoing a major dietary transition in the form of weaning. Rose and authors (1978) correlated accentuated striae with weaning stress in prehistoric human populations. And weaning stress has also been linked to accentuated striae in juvenile

baboons (Dirks et al., 2010; Dirks et al., 2002). Humphrey and authors (2008) analyzed strontium/calcium ratios in tooth crowns of wild-caught baboons and predicted that the ratios would change as juveniles experienced dietary transitions both at birth and at weaning. Changes in strontium/calcium ratios during enamel development were found to coincide with observational data on weaning timing (Humphrey et al., 2008). Moreover, Dirks and authors (2010) found that accentuated striae occurred in juvenile baboons at 6 months in one case, related to a reduction in sucking frequency, and 11 months in another, related to a cessation of nursing. Accentuated striae in this sample were determined to be indicators of weaning stress. Microdefects were found to occur most commonly in children aged 2-3.5 years in Wright's (1990) Mayan skeletal samples; however, this study does not take hidden cuspal enamel into account. In contrast, Simpson (1999) found that around 50% of microdefects occurred before the age of 18 months, in the cuspal portion of the crown.

While the number of studies documenting pathological striae in permanent cuspal enamel is relatively few, those documenting the presence of the neonatal line, a heavily accentuated stria in deciduous enamel, are numerous (Scour, 1936; Weber and Eisenmann, 1971; Whittaker and Richards, 1978). This line is often visible in the cuspal enamel of deciduous premolars and molars and permanent first molars, supporting the idea that even in the areas of highest decussation (*i.e.*, cuspal enamel), it is possible to detect accentuated striae.

Presupposition 2

The second presupposition is that infancy is a metabolically vulnerable period of childhood. This argument hinges on the immaturity of the immune system at birth. The immaturity of the immune system and lack of prior exposure to pathogens leave infants vulnerable to infectious disease (including viral, bacterial, and fungal infections). Natural, or

innate, immunity is (provided to) infants via two mechanisms. First, the body's natural barriers and lymphoid organs (which are still developing/ physiologically immature) provide immunity at birth, and second, colostrum, the first, anti-body rich milk secreted after pregnancy provides nursing infants and toddlers a boost in immunity during a critical immunoincompetent period. Acquired, or adaptive, immunity develops as a child becomes exposed to foreign microorganisms. It is arguable from a metabolic standpoint that infants are more vulnerable to infectious disease processes than slightly older children because, as a child ages, he/she gains experiential, acquired immunity (Roitt and Delves, 2001). This argument challenges the idea that the weaning period is the most significant time of stress for young children.

Hypothesis 3

The third hypothesis was that the pattern of enamel growth and disruption would be unique to the study groups/population. This hypothesis relies on the creation of an endogenous model for canine crown growth and comparison of that model to growth in other human populations. The most useful comparisons are between studies using the same histological and analytical methods, in this case with Simpson's (1999) analysis of the prehistoric and historic contact period samples from northern Florida, USA, which relied on counts of striae of Retzius along the dentino-enamel junction (DEJ) to establish overall crown formation duration, measurements of the external and internal locations along the lengths of respresentative striae, and the generation of regression equations to convert hypoplasia locations into internal locations along the DEJ (for comparison in distribution and timing).

Many bioarchaeological studies rely on the standards of Massler et al. (1941) for age-at-formation and crown completion and / or divide crown development into equal time zones

(Swardstedt 1966, Goodman and Song 1999). The division of crown height into equal developmental zones assumes that crown growth is linear, despite that Massler and colleagues demonstrated the opposite (Massler et al., 1941) The problem among studies with more reliable crown development chronologies is that there is still a significant amount of variation in crown completion times (Simpson 1999; Liversidge, 2000; Reid and Dean, 2006). Because the degree of variation in the duration of enamel growth among different human populations is largely unknown, imposing one population's growth schedule on another is potentially erroneous.

Presupposition 1

The first presupposition of Hypothesis 3 was that a comparison of published schedules of permanent crown growth would show a significant amount of variation - evidence for variation between populations. Permanent tooth crown development schedules for modern humans vary significantly (Reid and Dean, 2006; Dean 2000). The variation in development schedules translates into variation among defect chronologies because these are all based on some kind of developmental schedule. Goodman and Song (1999) identified sources for variation in linear enamel hypoplasia chronologies, including hypoplasia measurement error, variation in developmental timing, crown height variation, use of corrections for buried cuspal enamel and changes in enamel growth rates, and choice and interpretation of developmental schedule (see also Rose et al. 1995; Goodman and Rose 1990; Skinner and Goodman 1992; Simpson and Kunos 1998). Simpson (1999) and Ritzman and authors (2008) have pointed out the problems of more traditional, non-destructive anthropological studies that divide the labial surface of crowns into segments of equal breadth and then rely on the Massler et al. (1941) schedule to calculate age-at-formation of the defects. These studies fundamentally misinterpret crown growth as a linear process,

resulting in schedules that are incongruent with the growth process they are modeling (Hillson and Bond 1998; Simpson 1999). Simpson (1999) demonstrated the non-linear nature of crown growth – the density of striae of Retzius along the DEJ increases towards the cervix, indicating a slowing of growth as the crown nears completion.

Radiological studies of crown development such as Nolla (1960) are non-destructive but have other problems such as the accuracy of the radiographic image. Additionally studies using these standards have reported very different canine development chronologies, despite being based on similar populations (Simpson 1999, Simpson and Kunos, Beynon et al., 1998). Population-based variation in tooth calcification stages has been documented by in research relying on radiographic methods. Owsley and Jantz (1983) applied the developmental standards of Moorees et al. (1963) to juveniles in the Native American Arikara collection and concluded that the Arikara showed advanced calcification as compared to modern American Caucasians. Harris and McKee (1990) found that southern African-Americans attained specific calcification stages earlier than both southern European-Americans and northern European-Americans (Canadians). The authors suggested that these variations in rate of calcification were due to environmental and genetic differences between the groups. Watt and Lunt (1999) also concluded that the medieval Scots in their study showed faster dental development of the first permanent molar than in modern Caucasians. All of these studies clearly establish variation in enamel calcification at specific stages of development, but they do not address crown growth during the initial deposition of enamel matrix. This is largely due to the use of radiographs to determine developmental stage – and the inability of radiographs to detect the original immature, organic matrix deposited as the crown grows and takes shape. Huda and Bowman (1995) also noted that changes in the rate

of enamel formation in different parts of the crown are not accounted for in radiographic studies, which lead to flawed development chronologies.

Histological studies of crown development based on incremental growth structures have a number of advantages. They account for the non-linear nature of crown growth and the timing of hidden cuspal enamel, and they produce ages-at-formation of both hypoplasia and pathological striae in terms of days. Although this form of analysis is destructive, a method for molding crowns before thin-sectioning preserves their surface dimensions and features for future analysis. The amount of variation in modern humans even among histological studies employing similar methods is unknown, despite that enamel growth appears to be under greater genetic control than other bodily tissues. The use of small or pathological clinical samples in older histological studies (*e.g.* Massler et al. 1941 and Schour 1936), which have been hugely influential in biological estimates of defect timing in the past is questionable, and these standards may not be applicable to many populations.

Presupposition 2

The second presupposition was that the two friary cemetery samples chosen for the study would not show significant differences in enamel growth, defect prevalence, or defect timing. The choice of two samples from the same urban setting allows comparison of growth and disruption in samples related in terms of space, time, and likely, population genetics. The subtlety of the differences in the friaries themselves, even in extension to the individuals buried in their churchyards, does not warrant the assumption that one group was healthier than the other. Those interred at both cemeteries include a small number of royalty, nobility, and wealthy patrons, but it is unclear why certain individuals and groups chose one Odense friary over the other for burial. What is certain is that each cemetery contained a combination of men, women, and children representing social statuses that ranged from indigence to

royalty (the majority were probably in-between). Distinct differences between cemeteries in the patterns of normal growth and growth disruption produced by stress are not likely to be detected, given both the gross measure of the environmental factors that impact growth, the mix of individuals in each cemetery, and the complexity of the factors that impacted decisions for burial place.

It is important to note that burials in both cemeteries have been attributed to the post-A.D. 1250 medieval period and have not been dated any more specifically. Time within the cemeteries is difficult to determine, due to lack of grave goods, continual use of the cemetery throughout the medieval period, and the fact that only half of the sites, and only parts of the cemeteries, have been excavated due to existing buildings. No time-correlated patterns in burial position have been found (Becher, pers comm; see also Moller-Christensen, 1958). While the spatial patterning of burials may be somewhat useful in that older burials tend to be closer to church, it should be noted that the churchyard has moved in the past and that it extends past excavated areas.

SIGNIFICANCE OF THE RESEARCH PROBLEM

Many previous studies that have profiled childhood health based on dental defect prevalence and timing have relied solely on enamel hypoplasias, without reference to microdefects. Several bioarchaeological studies have utilized both hypoplasias and pathological striae to truly integrate data on chronic and acute events into descriptions of human health in the past (for example, Rose et al. 1985, Wright 1997, Simpson 1999; Thomas 2003, Antoine 2000). The value of this kind of research is that it produces hypoplasia chronologies with greater accuracy – important in archaeology because it provides a wider picture of stress in the past and, from a practical standpoint, it can potentially reduce the number of teeth needed for sectioning. Inclusion of pathological striae

in the stress profile provides a window onto stress in infancy that is often not available through traditional osteological analysis. The remains of infants and young juveniles frequently succumb to destructive taphonomic processes, essentially disappearing in a skeletal assemblage (Cook and Buikstra, 1979, Saunders et al. 1999). Teeth remain most frequently, providing access to the entire period of crown growth. This dissertation uses a model for timing both types of defects to not only gain further information about childhood stress levels in urban settings, but specifically stress in medieval Scandinavia (for which little is known in comparison to medieval Britain and continental Europe). Furthermore, the development of an endogenous canine crown development schedule and its comparison to schedules for other populations provides insight into the range of variation of growth in modern humans. Moreover, the methods allow more accurate insight into the types and timing of stressors in children in medieval Odense. Historical documents point to the types of stressors to which children were exposed, but relatively little empirical data exists on childhood health in medieval Denmark. The methods in this study allow important contributions to be made to a biological picture of growth disruption in Danish children.

SOLVING THE RESEARCH PROBLEM

Determining the prevalence and timing of metabolic disturbances involved a variety of methods, including identifying, counting, and measuring the locations of enamel defects microscopically. To most accurately determine the timing of these episodes, an endogenous enamel growth model for the mandibular canine was created, obviating the need to apply exogenous standards (with specific assumptions, see Chapter 3). The model generates an estimate of the time to total crown formation, which is converted into a chronological age of the individual at crown completion. The relationship between the distributions through time of hypoplasias and pathological striae was analyzed to assess the roles of chronic and acute

stress in childhood growth. Finally, total time to crown development in the study sample was compared to schedules produced from modern clinical and pathological samples, as well as archaeological samples from northern Florida, USA to determine the amount of variation between crown growth in the Danish sample and that of other samples.

Testing the research hypotheses requires comparison of data from the study sample to data from other populations. Because the methods used in this study parallel those employed by Simpson (1999) in his analysis of dental defects and crown growth in prehistoric and historic northern Florida, those data were chosen for comparison. Temporal comparisons within the friary cemetery samples are not possible because discrete stratigraphies of the sites were not identified in archaeologically – continued use of the sites over hundreds of years precludes any stratigraphic identification more specific than general period (*e.g.,* "medieval" versus "renaissance").

CHAPTER II

REVIEW OF THE LITERATURE

NORMAL ENAMEL GROWTH

Amelogenesis: Matrix deposition and maturation

In order to understand the etiology of enamel defects, a brief review of amelogenesis, the process of enamel formation, is provided. Amelogenesis is often described as a two-step process occuring within a tooth germ. It involves: (1) secretion of a primary organic matrix by cells called ameloblasts and (2) maturation of that matrix, during which ameloblasts remove the organic components and deposit secondary mineral mainly comprised of hydroxyapatite (Boyde, 1976; Hillson, 1996; Eisenmann, 1994). (Because the maturation phase of amelogenesis is itself a two-step process, some authors describe amelogenesis as occuring in three stages, secretion of the primary matrix, matrix removal, and secondary mineral formation (Schroeder, 1991). The process of enamel secretion begins at the occlusal tip of a tooth germ and proceeds toward the cervix of the tooth (Boyde, 1976; Hillson, 1996), following the path of general crown growth. Enamel growth occurs as both thickness and apposition, from the DEJ to the enamel surface, and elongation, the occluso-cervical spread of enamel. Clinical research has shown that tooth crowns elongate in a *non-linear* fashion, so that enamel is deposited at a variable rate in different parts of the crown (Sciulli, 1992; Shellis, 1984).

Ameloblasts are cells that possess organelles that enable them to synthesize and secrete enamel proteins (ca. 90% amelogenin and 10% non-amelogenin proteins) that form

an organic matrix capable of accepting hydroxyapatite crystals during maturation. Each ameloblast is rich with mitochondria, rough endoplasmic reticulum (ER), and golgi "profiles." The assembly and secretory pathways involve the rough ER, transport vesicles, the Golgi "apparatus," and secretory vesicles (Ten Cate 1994).

As internal epithelial cells begin to differentiate into ameloblasts, the cells elongate and become polarized. The nucleus shifts proximally away from the DEJ, and the majority of organelles cluster in the cell opposite the nucleus. Ameloblasts are closely aligned to one another, attached by junctional complexes at their proximal and distal ends of the cells. Filaments project from the complexes into the cytoplasm of adjacent ameloblasts, allowing junctional complexes to regulate what passes between ameloblasts to enter or exit forming enamel. Enamel protein is synthesized in the rough endoplasmic reticulum and transferred to the Golgi complex, where it is packaged into secretory granules. The granules move to the cell's distal end and are released against the dentin mantle/predentin. This initial enamel is mineralized almost immediately with inorganic ions from the dental follicle. The enamel is described as "structureless" because its hydroxyapatite crystals are deposited randomly, interdigitating with crystals from the dentin.

Following the deposition of this intial thin layer of structureless enamel, the ameloblasts move away from the dentin, and Tomes processes containing secretory granules form on each ameloblast's secretory end. Enamel protein is secreted from two distinct sites of the process, one at the cell body's periphery and the other on the surface of the process. Both secrete enamel matrix; however the orientation of the crystallites from each site is different. This differential crystallite organization gives the enamel structure. The majority of enamel

in a tooth crown is structured – only the first-formed and last-formed layers (when Tome's process pits are resorbed) exhibit crystals deposited randomly.

Once enamel has formed its full thickness in the secretory stage, the role of ameloblasts changes to maturing the enamel. This cyclical process involves removal of water and organic content, when the ameloblasts take on a "smooth-ended" appearance, and deposition of additional inorganic material, when the ameloblasts take on a "ruffle-ended" appearance.

Microstructure of normal enamel

Mature enamel is 96% inorganic by weight (Simmelink, 1994; Moss-Salentijn and Hendricks-Klyvert, 1990), rendering it the hardest structure in the body. Histologically, the structure of mature enamel is that of a mineralized epithelium and not a connective tissue (no fibrous structural elements are present in mature enamel) (Simmelink, 1994; Schroeder, 1991). The structures of mature enamel include: enamel rods, striae of Retzius, perikymata, and enamel cross-striations.

Rods, or *prisms*, are the fundamental structure of enamel. They are elongated cylindrical aggregates of hydroxyapatite crystals. The so-called "keyhole appearance" of a rod in cross-section results from the orientation of crystals parallel to the rod's long axis at its center and from those flaring laterally towards its periphery. The structural integrity of enamel results from the undulation of the rods as they extend from the dento-enamel junction to the outer one-third of the enamel's thickness (Hillson 1996).

Striae of Retzius (brown striae, growth lines, or Retzius lines) are a series of incremental lines (with optical and physical properties) which follow the pattern of crown growth layering during amelogenesis (Hillson, 1996; Risnes, 1990) . In coronal sections, striae appear similar to the concentric growth rings of a tree (Simmelink, 1994). In

longitudinal sections they appear as dark lines running from the dentinoenamel junction to the occlusal surface, maintaining the shape of the developing enamel front (Schroeder, 1991; Goodman and Rose, 1990). They also form concentric arcs at the cuspal and incisal edges in longitudinal sections (Swancar, 1986), revealing that some enamel is forever hidden by subsequent developing layers. Exactly what constitutes a stria of Retzius is still not well understood. Investigation into the structure and morphology of striae has shown that they cross-cut enamel rods (Hillson, 1996) and likely result from either: (1) a change in direction of the rods (Swancar 1986; Wilson and Schroff, 1970) or (2) a deficiency in mineralization of the rods (Moss-Salentijn and Hendricks-Klyvert, 1990; Swancar, 1986). One stria of Retzius is thought to represent a period of rest between two active phases of enamel secretion (Schroeder, 1991). The resting period in rod formation appears to occur every 7 days on average so that the area of enamel between each stria represents one week's formation time (Swancar, 1986). A more detailed model of the incremental growth of enamel is provided by Dean (1987). Dean describes the circaseptan (weekly) secretion of enamel as a product of the interaction of a 24 hour and 27 hour rhythm, resulting in a seven to eight day interference beat. Other important homologous rhythms in the body may provide clues to the etiology of striae of Retzius, including the frequency with which kidney transplants are rejected on a circaseptan rhythm and the almost weekly periosteal rhythms in human osteosarcomas (Dean, 1987).

Perikymata are the surface manifestations on unworn tooth crowns of the striae of Retzius. As such, they are used to calculate crown formation duration, which is used, in turn, to document growth and maturation in humans (Mann et al., 1991). Perhaps the most significant problem with using perikymata counts to predict tooth crown formation times is

that *cross striations*, the segments of prisms in the interval between Retzius lines, can vary in number (periodicity) from six to twelve (Reid and Ferrell, 2006; Thomas, 2003; FitzGerald 1998). An average of seven cross striations between striae has been assumed by many researchers to indicate that each cross striation represents a 24 hour period, or one day (Hillson, 1996; Risnes, 1986; Mann et al., 1991). By extension, striae of Retzius occur, on average, at weekly intervals. It is important to note that these intervals for formation are averages. Mann et al. (1991) point out that perikymata do not regularly record seven days of growth and that calculations of crown formation time relying on this regular periodicity may be erroneous (see Fitzgerald, 1998). Despite these problems of periodicity, counts of striae of Retzius have one very clear advantage over perikymata counts: striae at the cuspal/incisal tips of tooth crowns are visible (and countable) histologically, whereas cuspal perikymata formed at the onset of calcification remain hidden, buried under successive layers of enamel (Mann et al., 1991).

Counts of enamel cross-striations have been used by a number of researchers to estimate crown formation times (Dean and Beynon, 1991; Dean et al., 1993). There is ample evidence to support a prism periodicity rate of 24 hours for cross striations (Risnes, 1986; Smith 2006; Antoine et al., 2009), Bromage et al. (1997) measured cross-striations in human molar enamel and confirmed a weekly periodicity between adjacent striae of Retzius. Dean and Beynon (1991) have shown that cross-striation counts between adjacent striae are consistent within a tooth and dentition but vary between individuals. Lacruz and authors (2008) found that periodicities in the enamel of various hominin taxa varied by location in the enamel (eg. cuspal versus lateral), and other researchers have found the modern human

periodicties range from 6 to 12 days, with a mode of 8 or 9 days (Guatelli-Steinberg et al., 2005; Reid and Ferrell, 2006)

EVIDENCE OF GROWTH DISRUPTION: ENAMEL DEFECTS

Hypoplasias

The surface of a normal tooth crown is smooth and white (Hillson, 1996). Defects of enamel alter this appearance. Non-genetic pit, groove, and transverse linear defects on the enamel surface are classified as *enamel hypoplasia*, a deficiency of enamel thickness, indicating that the defect occurred during the secretion phase of amelogenesis (Hillson, 1996; Goodman and Rose, 1990). Hypoplastic lesions form in the secretory (first) phase of amelogenesis, as ameloblasts secrete calcium salts and proteins called amelogenins and enamelins (Suckling, 1989). The rate of secretion may be directly affected by the duration and severity of the stressor. Condon and Rose (1982) and Wright (1990) suggest that chronic metabolic stress slows the *rate* of secretion, producing hypoplastic defects. Acute (shorter-term) metabolic stress affects *total* ameloblast secretion, resulting in accentuated or pathological striae. It is important to note that extreme systemic disturbances can result in the death of ameloblasts, which would result in an absence of enamel in all teeth developing at the time of the disturbance. Disturbances that occur in the maturation, or second, phase of amelogenesis may result in hypocalcified enamel, an issue of enamel quality as opposed to quantity (Suckling, 1989).

The factors altering ameloblast secretion is unknown. Simpson (1999) suggested that cellular dehydration (occurring in individuals with acute conditions such as diarrheal diseases and high fevers) could alter secretory patterns. If dehydration negatively impacts protein biosynthesis or transport, or crystal formation in the cell, then it may be a part of the mechanism in the formation of enamel defects.

Enamel defects found on permanent teeth reflect *generalized childhood metabolic stress* because the crowns of the permanent dentition form after birth, during infancy and childhood. Childhood health is linked directly to population demography; and, because juvenile skeletal remains are often underrepresented in archaeological populations, enamel hypoplasia in adult remains provides a unique source of information on childhood well-being (Wright, 1997).

Hypoplasia is described as a general stress indicator because its causes are multiple and varied (Larsen, 1999). Anthropological studies on the prevalence of defects in modern and archaeological populations all over the world assume that most defects result from general (non-genetic) systemic stress (Boyde,1970) and have identified the major stressors as: (1) *nutritional* (Goodman and Rose, 1991) , resulting from famine (Zhou, 1995), a reorientation of diet (Lambert, 1993; Larsen and Hutchinson, 1992; Hutchinson and Larsen, 1990, 1988 ; Simpson et al., 1990), and weaning stress (Lanphear, 1990; May et al., 1993; Goodman et al., 1987; Goodman et al., 1984; although see Blakey et al., 1994) and (2) *environmental*, resulting from disease stress (Goodman et al., 1980; Larsen and Hutchinson, 1992; May et al., 1993; Simpson et al., 1990).

The methods for measuring the developmental timing of enamel hypoplasias vary widely among researchers (Skinner and Goodman, 1992). Many measure from the occlusal margin or the center of the defect to the dento-enamel junction and then convert this measurement into an age at occurrence by using already published enamelization schedules, noted by Swärdstedt (1966) to differ by study and to be a potentially significant source of error. Recent studies have also addressed quantifying the duration of stress episodes using the breadth or area of a lesion (Ensor and Irish, 1995; Larsen and Hutchinson, 1992; Simpson et

al., 1990), although a better method for estimating duration of an episode may be to count the perikymata contained within a lesion (Guatelli-Steinberg, 2000). Also of interest is differential susceptibility within and between tooth classes (Wright, 1997; Condon and Rose, 1992; Goodman and Armelagos, 1985) and the amount of lateral versus cuspal enamel in the tooth type. Traditional anthropological means of estimating the age of occurrence of enamel surface defects have failed to take into account variable enamel deposition; instead, it has been assumed that tooth crowns can be divided into equal portions for comparing frequencies of hypoplasia in these portions of the crown (see Goodman and Rose, 1990). Goodman and Armelagos (1985) demonstrated this intra-tooth variation in susceptibility to defects by dividing the tooth crowns into thirds and documented the prevalence of hypoplasia in each third. The middle third of tooth crowns was found to be most affected, followed by the cervical and then the cuspal thirds. Problems arise in comparing defect prevalence in *equal* thirds of a tooth given that the rate of crown growth, specifically elongation, is *non-linear* (Sciulli, 1992; Shellis, 1984) and that the "equal thirds" divisions have no actual developmental correlates.

A number of conditions have been implicated in the etiology of enamel hypoplasia in living populations. Pindborg (1982) summarized studies of enamel defects associated with specific causes, including: genetic and chromosomal conditions such as Amelogensis imperfecta, Ehlers'-Danlos' syndrome and Trisomy 21; congenital conditions such as heart disease, facial hypoplasia, and facial hypertrophy; congenital metabolic disorders such as galactosaemia, phenylketonuria, alkaptonuria, erythropoietic porphyria, and hyperoxaluria; neonatal disturbances, including premature birth, hypocalcemia (see Nikiforuk and Fraser 1981), hemolytic anemia, and congenital allergies; viral infectious diseases such as rubella

and bacterial infections such as syphilis and tetanus; neurological disorders; endocrinopathies such as hypoparathyroidism and diabetes mellitus; nutritional deficiencies inlcuding vitamin, mineral, and protein deficiencies; kidney disorders; enteropathies including non-specific diarrhea, coeliac disease, and lymphangiectasia; liver diseases; intoxications with tetracycline, thalidomide, vitamin D, and Pica-related substances; local mechanical trauma, burns, and irradiation; and local infections such as periapical osteitis, neonatal maxillitis, and odontodysplasia.

In modern developed nations, enamel hypoplasia affects only about 10% of the population (3-15% according to Pindborg, 1970). The clinical implications of hypoplastic lesions are important to note because hypoplastic lesions are ideal sites for attack by carious bacteria. The result is a penetrating carious lesion, possible tooth loss, and often chronic periapical abscesses (Infante and Gillespie 1977).

A high prevalence of enamel hypoplasia in living populations has been shown to be clearly positively correlated with poor socioeconomic status. Enwonu (1973) found a clear correlation between hypoplasia prevalence in deciduous teeth and poor socioeconomic status. Nigerian children aged 0-7 years from a high socioeconomic group showed no defects, while malnourished children from families living in poor village conditions showed defect prevalences between 6-21%. Retarded body height and weight were also documented in these children, revealing growth disturbances in other tissues of the body. Further evidence for the role of undernutrition in hypoplasia etiology is provided by Goodman et al.'s (1991) study of linear enamel hypoplasia (LEH) prevalence in 84 adolescents from the rural Aztec Indian community of Mezonteopan, Mexico. Half of the children in the study had received nutritional supplements to their regular diets since birth, while the other half were not

supplemented. Those children receiving the supplements showed LEH prevalence in the range of 28.6-50.4% (of individuals affected), versus the non-supplemented grow with prevalence in the range of 64.7-84.1%. Both groups showed a peak age at formation of ca. 2-2.5 years, but the non-supplemented group had a wider range of ages at occurrence. The authors concluded that undernutrition during enamel formation is causally linked to linear hypoplasia formation (Goodman et al. 1991).

In a study of enamel hypoplasia in village children from Solis, Mexico, Goodman et al. (1992) found that children with defects had lower body weight and shorter stature than those without defects. The synergistic relationship between diet, disease, and environment is difficult to tease apart in determining defect causation. Malnutrition and infection were problems common to populations from Polynesia, Southeast Aisa, Central America, and Africa where linear enamel hypoplasia was prevalent in deciduous teeth at rates of 14-85% (Infante and Gillespie 1977). In the United States, Infante (1974) found that between 19 and 39% of a sample of 96 Apache children showed hypoplastic defects. All of these children were living in poverty or near-poverty and were malnourished, as determined by poor quality protein intakes and low energy, calcium, and vitamin intakes. However, it is important to remember that the defects formed during enamel development. The conditions present *during the time of the study* are not relevant to defect etiology. Enamel does provide a permanent record of developmental stress because, unlike disturbances in bone from which children can recover, e.g., catch-up growth, enamel does not remodel. No analogous system of recovery of normal structure exists in enamel, so defects persist throughout life.

Infections are another large-category etiology of enamel hypoplasia. Sweeney et al. (1969) argued that neonatal infections in Guatemalan children between the ages of 2 and 3

years accounted for the prevalence of linear enamel hypoplasia found in the deciduous incisors of 42.5% of the sample. The most prevalent infections were conjunctivitis, thrush, and diarrhea, and they occurred often in the first month postpartum (Sweeney et al. 1969). Parasitic infections are also responsible for enamel growth disruption. Suckling et al. (1986) found that inducing parasitism in sheep damaged the function of ameloblasts, the cells that produce enamel. The sheep suffered severe diarrhea, weight loss, and weakness, and postmortem examination histological sections of growing incisors revealed that hypoplastic severity (missing enamel) was related to the serverity of disturbance to ameloblastic activity.

While, malnutrition and unsanitary living conditions are correlative to enamel hypoplasia prevalence in all of these cases, neither is directly implicated in defect etiology. The synergistic relationship between undernutrition and infection is implicated by Infante and Gillespie's (1977) of rural Guatemalan children with linear enamel hypoplasia. The study revealed that children aged 2-7 years with hypoplastic lesions in their anterior teeth had caries prevalences in their posterior teeth two to four times greater than children without dental defects. The authors concluded that the mechanisms that underlie hypoplasia formation, undernutrition and infection, also may predispose the posterior teeth to excessive carious attack (Infante and Gillespie 1977), conditions that are more prevalent in economically underdeveloped countries.

Bioarchaeological studies of enamel defects have, in general, reflected the findings from studies on living peoples in terms of correlation with under- or malnutrition and disease prevalence. Historic skeletal samples, especially in comparison to earlier groups, experienced a higher prevalence of hypoplastics defects, as diets changed to include more processed agricultural foods, as population densities increased in town settlements, and as infectious

disease prevalence increased (see Larsen, 1999 for review of the changes that accompany agricultural subsistence shifts). And demographic data have showing that individuals with defects have higher mean frailty than non-affected individuals from the same population (Boldsen, 2007; see also Thomas, 2003).

Pathological striae of retzius (Wilson bands)

Growth disruption is also recorded by microdefects known as *pathological or accentuated striae of Retzius*. Pathological striae result from a sudden change in the direction of the enamel rods, associated with atypical rod morphology (Hillson, 1996; although see FitzGerald and Saunders, 2005), and they appear as thick, brown bands. An accentuated stria of Retzius is often classified as *pathological*, although there is no inter-observer agreement regarding when normal morphology becomes pathological. Attempts have been made at classifying striae of Retzius based on morphology (Risnes, 1990; Rose, 1977; Wilson and Schroff, 1970); however, no standardized classification exists.

Pathological striae of Retzius are commonly observed as histological correlates of enamel surface defects like hypoplasia, but a temporal association between microdefects and macrodefects does not always occur. And these microdefects are unlike enamel hypoplasias in that they can be seen and quantified throughout the enamel, from cusp to tip. The lack of association in many cases suggests that macrodefects and microdefects have different etiologies. Acute, non-chronic stress may result in pathological striae, while chronic stress may be responsible for hypoplastic lesions on the tooth's surface (Goodman and Armelagos, 1990; Wright, 1990). Pathological striae, like enamel hypoplasia (see Swärdstedt, 1966) have also been shown to correlate negatively with socioeconomic status (Mifsud and Marks, 1998).

The most well-known accentuated stria of Retzius is the neonatal line. The neonatal line is a stria that often appears darker and wider that the striae surrounding it, due to environmental changes that occur with birth. It differentiates pre- from post-natal enamel (Schour, 1936; Weber and Eisenmann, 1971; Antoine et al., 2009). Noren (1983) documented the location and appearance of the neonatal line in both normal weight and low birth-weight Swedish infants and found that more enamel disturbances, including hypoplasia, were prevalent in the low birthweight group.

Accentuated striae have been positively correlated with chronic and repetitive acute disease episodes in infants. In a study of 19 forensic cases, Teivens et al. (1996) found that Swedish infants with an antemortem history of internal organ inflammation and infections had a higher prevelance of pathological striae than infants with no recorded antemortem health problems. Thomas (2003) correlated accentuated striae with a higher risk of death after age 7 in a medieval Danish skeletal assemblage from Tirup. The data on positive correlations between skeletal indicators of nutritional and disease stress and higher prevalence of pathological striae is less abundant, but nonetheless convincing (Rose et al., 1981; Rose et al., 1985). Additionally, pathological striae are, not surprisingly, positively correlated with low socioeconomic status. In a study of microscopic enamel defects in a Middle Woodland Native American skeletal sample, Cook (1981) found that defects were more prevalent in individuals buried in low-status graves (as determined by burial location, body preparation, grave furnishings, as well as differential arthritis patterning, trace element composition of the ribs, and stature). Cook (1981) also noted that age at death strongly affects the apparent frequency of defects in the sample, and that stress experienced before age three years had a negative impact on later survival. It is useful to note that sex differences in

pathological striae prevalence appear to be negligible (Simpson 1999, Cook 1981, etc.); however, one might expect differences by sex to occur in populations with extreme status differences in males and females (or extreme dichotomies in division of labor).

CHAPTER III

METHODS

SUBJECTS AND SITE HISTORY

Site background

Two medieval cemetery sites, the Black Friars Square site and the Gray Friars Place site, were chosen because they comprise large samples of medieval skeletal individuals. Approximately 979 skeletal individuals were originally identified in the cemetery at the Black Friars site and 591, from the Gray Friars cemetery. These cemetery sites were also intriguing because they theoretically represent a good cross-section of individuals (in terms of socioeconomic class) living in and around the town. Both skeletal collections were analyzed for basic demographic data on age and sex as well as suitability for dental sampling.

Black Friars Square Cemetery (Sortebrødre Torv Kirkegård)

The Black Friars monastery in Odense, one of 19 mendicant monasteries established in Denmark during the Middle Ages, flourished from ca. A.D. 1239 to 1539. The friary was located in the northeastern corner of the medieval town (now the central part of Odense). Previous excavations of the friary site have revealed the foundations of the friary church, known as St. Peter's, and connected cloisters that served as the kitchen and housing areas for the friars themselves. Archaeological excavations in the 1970s also revealed that the friary also maintained a water mill and millhouse, a garden, and multiple sewer lines. All of the friary buildings were destroyed shortly after the Lutheran Reformation in Denmark, although the cemetery continued to be used through the 17th century. The Black Friars Square

cemetery was first excavated in 1978 by a team of archaeologists from Møntergården, Odense's city museum, prior to the installation of a modern sewer line at the site (Urth 1978). Excavations continued into 1979 and 1981.

A total of 661 graves were excavated in 1981 (comprising about one-half of the total cemetery), most of which could not be dated to any specific time within the 300 year medieval timespan. Well-preserved skeletal remains from the graves were removed and housed with the Antropologiske Database Odense Universitet (ADBOU) in Odense.[2] The graves surround the St. Peter's Church foundation and extend to the southwest towards modern Overstræde and Claus Bergsgade (Figure 1). The density and disturbance of burials, often one intruding on another, made assigning dates to them by stratigraphic layer difficult.[3] Moreover, medieval Christian burials rarely contain grave goods that might be used to establish dates or even social status (exceptions are the few graves that contained rosary beads, coins, and seals – and aristorcratic ceremonial grave goods in only two cases). The remnants of wooden coffins and nails were found in many burial features; although many appear also to have been deliberate coffin-less burials, these could not always be distinguished from coffin burials with wood that did not survive. Both wooden coffin (most common at SBT) and coffin-less burial types were common in the medieval period and give

[2] It should be noted that not all skeletal material was collected from the site – anatomists involved in the excavation efforts left poorly preserved skeletal material at the site in favor of whole or well-preserved material.

[3] Dendrochronology was not used to date the remains of wooden coffins because coffin wood was often recycled from prior structures. Archaeologists used the stratigraphic relationships of graves intruding on one another to try to date groups of graves – still, this method yielded no clear results. A final attempt to date the burials was made by examining body position within each grave. According to medieval Catholic burial customs, nearly all bodies were laid out in a supine position with the head to the west and feet to the east (such that on Judgement Day, the dead could rise from their graves and look eastwards). This pattern was found in all but a handful of atypical burials (Arentoft 1991). Attempts were made to date the graves by burial position, specifically arm position, but no consistent pattern was found (Becher 1999).

no indication of class or social status within the cemetery. A small number (14) of a third burial type, stone-walled graves, provide a slightly narrower timeframe for relative dating, as walled graves were most common in the 13th century (Kieffer-Olsen 1993). Of these graves, 3 were located inside of the church, and 11 were near the church. The proximity of these burials to the church may be more indicative of social status. For example, the 3 walled burials were located in the floor of the church itself. These burials were highly different from typical churchyard graves because they contained ceremonial graves swords. Such elaborate, expensive grave goods are highly suggestive of aristocratic burial; indeed, burial inside of medieval churches was often reserved exclusively for the aristocracy and high level clerics**.** Burial outside of, but in close proximity to, the church is also common indicator of higher social status in medieval European cemeteries; however, there is no direct evidence of this pattern in the Black Friars cemetery.[4]

Social status was nearly impossible to determine within the Black Friars cemetery. The cemetery was likely used to inter people from all social strata during the Middle Ages (although written sources indicate that the cemetery was used to bury primarily the indigent after the Reformation ca. 1540). The primary tasks of the friars were to preach to those living in the town and to care for the dead – including indigents whom the friars had taken in as well as middle and upper class townspeople who paid to be buried in the friary churchyard. Men, women, and children were interred in the cemetery, although no clear pattern of sexual division of burials was discernable (Becher 1999).[5] More men than women were interred in

[4]That pattern is based on the idea that rainwater dripping from the eaves of the church would fall onto the graves below and thereby "bless" the dead over and over again (Kieffer-Olsen 1993).

[5] The women were once thought by scholars to be wives or mistresses of the friars, but it is more likely that they were the relatives of lay-friars or townspeople who had contracted to be buried in the cemetery (Kieffer-Olsen 1993).

the cemetery (see Becher 1999), attributable first and foremost to the 300 year existence of the friary as a male institution (and concomitant burial of the friars in the cemetery). It is also possible that a portion of the cemetery that has been excavated was reserved for men, in light of the fact that the sample comes from only ca. one-half of the total cemetery. Becher (1999) contends that the presence of people of both sexes and all ages is a testament to the ability of the friary to intertwine itself with the lives (and deaths) of the population of Odense.

Gray Friars Place Cemetery (Gråbrødre Plads Kirkegård)

The Gray Friars Place friary in Odense was a medieval Franciscan friary site dating from ca. 1280 to the Reformation in A.D. 1539. The site is located in the central portion of modern Odense. In the Middle Ages, the friary stood at the northern edge of the town, with open landscape to its northern and western sides. The first excavations of the site, directed by Odense's Town Museum (Odenses Bysmuseem, Møntergården), took place in 1982, when the local municipality wished to renew the area by planting new trees on the site. The test excavations yielded not only skeletal material from medieval burials, but also the foundations of the porch of the friary church (Arentoft 1991).

Archaeological excavations associated with the renewal project continued in 1990, when renewal plans had expanded to include a piece of "vandkunst," or water art, in addition to the trees. The areas to be excavated were divided into 7 squares, labeled A through G on site maps (Figure 2). These areas chosen for excavation were the areas chosen by the municipality for replanting and building of the water installation and its accompanying water line. Burials were found only in squares A through D, all completely inside the boundaries of the medieval cemetery with the exception of B.[6] Archaeologists recorded 418 graves in 1990.

[6] Square A, chosen for the water art installation, comprised a 24m^2 excavated area; a significant portion of the area was disturbed and contained several destroyed graves. Square B was comprised of 23m^2

Few grave goods were recovered (as expected in a medieval Catholic cemetery); although, a few graves contained coins or medieval bone or amber beads, presumably from rosaries. The burials themselves gave no indication of the social status of the interred. Coffin type was not useful, either, in determining social status within the cemetery. Excavations revealed that 212 of the burial features showed traces of wooden coffins (either traces of nails or the wood itself). Burial density was very high in all of the squares, such that coffin-type even in the most well-preserved features was indeterminable. In a few cases, archaeologists identified coffins as either trapezoid-shaped or as bar/slatted form (Arentoft 1991).

While provisional determinations of age and sex were made in the field, no systematic demographic analysis of the skeletal material was made until 1999. Additionally, anatomists from the Institute of Cytology and Anatomy at Odense University rejected (and disposed of) some of the skeletal material because it was too meager in quantity or too poorly preserved to be useful in teaching. Loose bones (not associated with a particular grave), with the exception of crania, were discarded (Arentoft 1991).

In A.D. 1279, King Erik Clipping (r.1259-1286), the original patron of the Gray Friars in Odense, granted the friars the plot of land in Odense on which to build a friary. The king's intent was to use the new friary church for the interment of his family (his daughters were eventually interred inside the church). A second episode of royal patronage came at the end of the medieval period, when King Hans (r.1482 -1513) and his family chose the friary church as their final resting place. Han's queen, Queen Christine, endowed the friary church with altar furniture that survives today in St. Knud's, Odense's cathedral church. The

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excavated area that revealed remnants of the friary's porter's lodge as well as cemetery burials in the most southerly part of the square. Square C, circa 48m^2, contained part of the foundation and buttress for the church chancel, as well as churchyard graves. Square D contained an excavated area of only 56m^2 due to the presence of a live power line. This square only contained remnants of the churchyard cemetery.

majority of the people served by the friary, however, were not royalty, but were either townspeople who made arrangements to be buried in the churchyard or were indigents who died in the care of the friars (Arentoft 1991).

After the Reformation, the friary itself was converted to a hospital and was used as such until 1866. The friary church, however, lost its status as a parish church in 1618, and the church was eventually demolished between 1817 and 1819. The last burials in the churchyard are provisionally dated to ca. 1800 (Arentoft 1991). Today, a small portion of the original cloister walkway and cemetery yard still exists; however, most of the original site is covered by a new church and a retirement home and associated grounds.

Preliminary osteological analysis

Age determination

The ages of the skeletal individuals excavated from the Black Friars and Gray Friars cemeteries were derived using a variety of techniques. Skeletal ages were determined using ADBOU's own written descriptions of the stages of pubic and auricular age estimation in conjunction with classic osteological methods of pelvic age estimation from Todd's (1920) and the Suchey-Brooks (Brooks and Suchey 1990) descriptions of pubic symphysis stages and Lovejoy and author's (1985) descriptions of iliac auricular surface age estimation. Children and young adults' ages were determined using Ubelaker's (1978) deciduous and permanent tooth development and eruption stages, as well as age at epiphyseal closure in cranial elements (Meindl and Lovejoy 1985, Mann et al. 1987) and postcranial elements (White 1990) and postcranial element lengths (Fazekas and Kosa 1978, Ubelaker 1978). Many of these methods were conveniently reprinted and redrawn in Milner (1994).

Sex determination

The biological sex of the skeletal individuals was determined for individuals displaying secondary sexual characteristics in the skeleton (young adults usually over age 15 years and adults). The method used for sex determination of adult pelves was based on several features of the pubis, including the ventral arc, subpubic concavity, and medial aspect of the ischiopublic ramus (Phenice1969), as well as the angle formed by the greater sciatic notch and the depth of the preauricular sulcus. Helpful drawings of all of these characteristics by P. Walker appear in Milner (1994). The presence of dorsal pubic pitting, often an indicator of pregnancy and parturition, was used to designate remains as female. Cranial characteristics were also assessed for sex association, with the help of graded drawings by P. Walker and descriptions of robusticity of the nuchal crest, mastoid process, glabella, supra-orbital margin (all graded on a 1-5 scale), and mandibular robusticity of the mental eminence and gonial angle (Milner 1994).

Selection of dental sample

Mandibular canine. A sample of 410 canines (n = 234 Black Friars, n = 176 Gray Friars) were chosen to be evaluated for enamel hypoplasias (one per individual) from the inventory of individuals who retained at least one fully mineralized, intact permanent mandibular canine. From these canines, a subsample of n = 33 teeth (each representing one individual) from the Black Friars collection and n = 30 from the Gray Friars collection were chosen randomly to be sectioned for the histological defects analysis.

A second subsample of canines were also chosen from the original sample to derive a population-based model of enamel growth. Six crowns were originally chosen, although three were ultimately used (representing individuals aged 11.5 to 27 years) for the model based on visibility of SOR throughout the crown. Determining a standard for the timing of

growth in different parts of the adult canine crown (e.g. cusp versus cervix), as well as the total time to crown completion, required that the crowns used for the standard be as complete as possible. Therefore, the sample used to create the endogenous growth standard was chosen with the following criteria: (1) each crown was fully mineralized, (2) there was little to no enamel attrition, (3) there was no damage to cervical enamel along the DEJ (which serves as the basepoint for measurements), and (4) SOR were visible throughout the crown.

RESEARCH DESIGN

Procedures

Dental sample preparation

The crowns of whole teeth were rinsed in water and lightly swabbed with an ethanol solution to remove adhering dirt and debris. This cleaning procedure was performed before analyzing whole crowns for external defects as well as to prepare a subsample of the teeth for molding and sectioning.

Molding

Molds were made of each crown in the samples to be thin-sectioned in order to preserve the crowns' exterior features and dimensions.

Negative molds. Each crown was cleaned with 95% ethanol prior to the molding process. Negative molds of the crowns were produced with Provil® novo (Light Body, Fast Set), a silicone impression material used to make high quality surface replicas. The base and catalyst were mixed together in a 1:1 volume ratio and applied to each crown with a wooden stir stick. The material cold-cured within 5 minutes, and the crowns were freed from the mold negatives by gently pulling on the roots and moving them back and forth, out of position. Good molds retained all of the detail of the crown surface and were left for several hours before filling with resin, to ensure a completely dry set.

Resin. Araldite epoxy resin was chosen as the medium, as it produces excellent detail for SEM studies. The resin consisted of three chemicals, a hardener, resin, and accelerator in the following ratios: 75g DDSA (hardener): 100g Araldite 502 (resin): 1.9g DMP-30 (accelerator). The DDSA and Araldite 502 were combined under a hood vent and placed on a magnetic stirring platform for 45 minutes, or until the mixture appeared smooth. The accelerator was added to this, producing a darkened orange color, and mixed for another 15 minutes. The final product was poured into negative molds of the crowns and used for embedding the teeth themselves.

Positive molds. Each negative mold was filled with resin under a hood vent. A slim applicator stick was inserted into each mold to release air bubbles that might have become trapped as the resin was added. The molds were transferred to a 60° C oven to cure for 24 hours. The curing molds were checked every hour for the first 3-4 hours for air bubbles. The final product, a positive resin cast of each crown, was revealed by simply tearing away the negative mold. The molds were stored as a permanent record of the external appearance and dimensions of the crowns destroyed by sectioning, and they and may be used for future scanning electron microscopic analysis of perikymata and enamel hypoplasias.

Embedding

Once positive molds had been produced of all the crowns chosen for sectioning, the canines themselves were embedded and prepared for thin sectioning. Each tooth was cleaned prior to embedding in successive 15-minute baths of 70%, 95%, and 100% ethanol and placed into a Peel-A-Way® 1”x1” disposable plastic tissue embedding mold. A label identifying each specimen’s provenience was added to each mold as well. Enough resin to cover the crown and cervical portion of the root of each tooth was poured into the molds. The

molds were placed into a 60ºC oven for at least 24 hours, or until the resin hardened completely. The hardened blocks were removed from the molds and readied for sectioning.

Sectioning

Thick sections. To produce a midline thin section in the labio-lingual plane, each whole block was placed in an irregular saw chuck, mounted onto the saw arm of a Buehler Isomet sectioning saw, and oriented to be cut slightly off-midline. Thick sectioning resulted in two thick sections, each containing half of the canine. Each thick section was rinsed, bathed in 95% ethanol, and gently swabbed with a .1M hydrochloric acid solution to decalcify the enamel and expose the rods more clearly.

Thin sections. Sections approximately 250-300μm thick were cut from one of the thick section blocks for each individual. For each section, the saw micrometer was turned ca. 12 increments, moving the saw blade approximately 300μm from the face of the slide. The position of the blade was recalibrated for each section, to account for differences in adhesive thickness. All thin sections were rinsed in water and acid-etched in .1M hydrochloric acid solution for 4 seconds to reveal rod structure. It should be noted that the sections were not polished by machine. Sections were permanently mounted using a clear epoxy resin and temporarily coverslipped[7] using a 30% ethanol solution or oil for examination under a light microscope. All sectioning was performed by the author in the Simpson Laboratory in the Department of Anatomy at Case Western Reserve University School of Medicine and in the Wright Laboratory in the Department of Pediatric Dentistry at the University of North Carolina School of Dentistry.

[7] A few slides were coverslipped permanently using Permamount.

ANALYSIS

Identifying defects

Hypoplasias

All of the sampled canine crowns (n = 234 from Black Friars site; n = 176 from Gray Friars site) were evaluated for surface defects at a magnification of 10x on a dissecting light microscope. Specific criteria for defining surface defects (hypoplasias) were used after Hillson and Bond (1997) and included: (1) furrow-form defects that interrupt the normal spacing of perikymata and show occlusal and cervical walls bordering the defect floor, (2) pit-form defects in bands or clusters, representing clusters of ameloblasts that have ceased enamel formation, and (3) exposed-plane form defects, with a wholly exposed growth line plane representing a cessation of enamel production.

The breadth of each hypoplasia as well as the distance from its cuspal edge to the cervix of the root was measured in millimeters with the ocular micrometer.

Pathological Striae of Retzius

PS were only scored in the canine subsample chosen for thin sectioning (n = 33 from Black Friars site, n = 30 from Gray Friars site). PS were positively identified based on at least two of the following three criteria: (1) stria appears darker and wider than surrounding striae, extending clearly from the DEJ to the enamel surface, (2) the stria exhibits rod disorganization on examination at 1000x magnification, and (3) the stria has a corresponding darkened stria in the lingual enamel. All sections were examined and scored for PS on a transmitted light microscope at progressive magnifications from 40x to 1000x. Suspected PS were examined by a second observer for verification in a number of cases. Agreed upon striae were denoted as pathological; however, disagreement on PS identification resulted in those striae not being recorded as pathological. A small number of thick sections with

suspected PS were also examined with Scanning Electron Microscope – only a few cases of disrupted enamel were verified with SEM. Stria meeting the criteria for pathology were noted, and photographs of the sections at 40x were scanned and examined digitally with ImageTool 3.0 biomedical imaging software (available as shareware from the University of Texas at San Antonio). The absolute locations of PS were measured in millimeters from the DEJ to the cervical enamel.

Determining the timing of defects

Endogenous growth standard

The length of the DEJ was measured digitally in photographs of thin sections of the unworn mandibular canine crowns using ImageTool 3.0 and also MetaMorph miscropic image analysis software.

Each DEJ was divided into 10% increments along its length using the regions function in MetaMorph (Figure 3), and counts of the intersecting striae of Retzius were taken within each increment (Reid and Dean, 2000). Counts were taken the entire length of the DEJ – covering both imbricational and cuspal enamel – to determine total enamel formation and to document the location of any cuspal pathological striae (per Simpson, 1999). To promote visibility of these intersections, a photomontage was created of each thin section (Figure 4). Each montage consisted of 4 to 7 photographs taken at 40x magnification through a light microscope. The montages were scanned in pieces and reassembled digitally using Adobe Photoshop and analyzed with MetaMorph software.

Two types of data were sought by counting striae of Retzius. First, counts per increment provided data on the *rate* of enamel formation. This method of determining enamel growth pattern relies on the critical assumption that the cross striation repeat interval (periodicity) is 8 (after Reid et al, 2002). Reid and Ferrell (2006) found that the modal value

in 49 Danish medieval mandibular canines was 8 (with a range of 7-11 days, normally distributed), but periodicities do vary within the enamel. For the purpose of this research, a periodicity of 8 was selected as the model standard. The average daily rate of enamel secretion was also assumed to be 3 μm (Schour and Poncher 1937). Second, the total number of striae for any given canine provided information of the *total time* of enamel growth.

Total formation time was calculated by counting all the striae intersecting with the DEJ (after Simpson, 1999). Formation time in days was summarized by the following formula:

$$\text{Number of striae} \times 8 \text{ (days)} = \text{time to formation (days)}$$

Making internal and external defect timing comparable: Converting external to internal formation time

Enamel growth pattern was defined in two ways: (1) the relationship between the location of the origins of normal striae along the DEJ and their corresponding terminations at the enamel surface, summarized by a regression equation and (2) the mean total time to crown completion, determined by counts of striae of Retzius throughout the enamel.

Simpson's (1999) method for converting internal to external defect timing (thus, making the distributions comparable) is duplicated here. The course of 79 striae of Retzius in the Black Friars sample and 84 in the Gray Friars sample (163 total) were recorded by measuring the internal intersection of each SOR with the DEJ and the external location of each from its surface manifestation to the cervico-labial junction, along the external surface of the tooth. The relationship between each pair of data points (internal and external positions) was analyzed using MS Excel and SAS software, resulting in non-linear regression equations useful for translating external to internal positions (or vice-versa). The location (in mm) of each defect from its cuspal, or leading, edge to the cervical margin of the tooth was

measured with a dissecting light microscope. Each defect location was plugged into the sample-specific distance function to derive a corresponding internal location along the DEJ. The conversion allowed direct comparison of the distributions of hypoplasias and pathological striae. It is expected that, based on enamel geometry, hypoplasias will be seen in imbricational only; but PS may occur at any position in the crown. A different mean distribution of PS from that of hypoplasias may suggest that the pathologies do represent different etiologies, *i.e.*, acute and chronic. The equation itself represents the relationship between the internal and external architecture/growth of the enamel, a relationship which cannot be assumed to be the same in different (genetic) populations. The converted internal locations were plotted in 10% increments per the population model to determine distribution by location within the tooth crown.

Standardizing for tooth size (for inter-tooth comparability)

All sampled whole canine crowns were measured to standardize for differences in overall tooth size. Crown dimensions were measured with electronic calipers to the nearest hundredth of a millimeter in labio-lingual and mesio-distal planes. Maximum and midline crown heights were also measured with calipers to evaluate.

Locations of pathological striae along the DEJ in thin sections of canines were standardized by dividing the absolute location in millimeters by the labiolingual breadth of the sectioned tooth (taken at the cervical enamel margins) (after Simpson 1999).

ANALYSIS: COMPARISON WITH PUBLISHED SUMMARY DATA

The first hypothesis, that the mean prevalence of pathological striae is greater in the medieval study sample than in archaeological samples from the New World, was summarized as:

$$H_1: d_m > d_f$$

where *d* indicates mean defect prevalence, *m* indicates the medieval study sample, and *f* indicates comparative historic and prehistoric samples from northern Florida (representing AD 1- 1704) (Simpson 1999).

The second hypothesis, that pathological striae occur in the "hidden" cuspal enamel of children ca. 6 to 18 months old, expanding the age range of defect chronology to include early infancy, was summarized as:

$$H_2: a_m < a_f$$

where *a* indicates mean peak age range at occurrence, *m* indicates the study sample, and *f* indicates comparative samples from northern Florida in 50% of internal defects occurred in the cuspal half of the crown and before the age of 18 months. The age distributions of each type of defect (determined by using the population model of growth) was expected to differ, with PS not only occurring in infants, but also occurring on a different schedule from enamel hypoplasias (reflecting different ages at occurrence).

The third hypothesis, that the pattern of enamel growth is unique to the study sample, was summarized as:

$$H_3: g_m \neq g_f$$

where *g* indicates growth, *m* indicates the medieval sample, and *f* indicates the archaeological population from northern Florida, USA analyzed by Simpson (1999). The medieval Danish study sample and the northern Florida samples represent genetically distinct populations, so their growth models may differ. The regression equations for crown growth were compared between the samples for statistically significant differences at the $p<.0001$ level.

CHAPTER IV

RESULTS

OVERALL DEMOGRAPHIC STRUCTURE OF THE CEMETERIES

The overall demographic structure of both cemetery samples is similar. Figure 5 shows that adult males (n = 227) comprise 50% of the individuals excavated from the Gray Friars cemetery (n = 455). Adult females (n = 127) make up 28% of the sample, while adults of unknown sex (n = 33) comprise 7%. Juveniles (n=68), defined as individuals less than 16 years old, comprise 15% of the sample. The Black Friars cemetery sample (n = 557) is comprised of mainly adults, with males (n= 235) at 42%, females (n=179) at 32%, and adults of unknown sex (n=48) at 9% (Figure 6). Juveniles (n=95) accounted for 17% of the entire skeletal sample. The dearth of juveniles in both samples is not entirely unexpected, as children in medieval Scandinavian cemeteries were often buried together (but apart from their families) in a separate part of the churchyard (Sellevold, 2008). However, it should be noted that no caches of child burials have been found at either cemetery.

PREVALENCE OF ENAMEL DEFECTS

Surface defects (hypoplasias)

The sex and age breakdown of the skeletal individuals scored for surface defects is reflected in Table 1, while Table 2 describes surface defect prevalence in both cemetery samples. One tooth per individual was scored, with left mandibular canines selected preferentially. A total of 234 canines (representing 234 individuals – 209 adults and 25 juveniles under age 15 years) were scored from the Black Friars cemetery. One or more

surface defects (SD) were detected in 218 of those, resulting in a SD prevalence of 93%. In the Gray Friars sample, 176 teeth were scored (156 adults and 20 juveniles), with 174 found to have one or more defects (see Figure 7). The prevalence in the Gray Friars dental sample was 99%, similar to that of the Black Friars. When the samples are combined (n=410), the prevalence is 96% (392 afflicted).

Surface defect prevalence was also determined for afflicted teeth (Table 3) and for all teeth scored (Table 4). The mean number of surface defects per tooth in the afflicted Black Friars sample was 2.69, with a standard error of .10 and standard deviation of 1.43. The maximum number of defects for any one tooth was 7. In the Gray Friars sample, the mean number of SD's per tooth in the afflicted sample was even higher, at 3.72 (SE = .14, SD=1.80). The maximum number of defects per tooth in the Gray Friars sample was 9, also slightly higher than the Black Friars sample. When the cemetery samples were combined, the mean number of defects per tooth for afflicted teeth was 3.15 (SE=0.09, SD=1.68).

SD prevalence was also scored for all teeth in the sample, afflicted or not. The mean number of SD's per tooth declined very slightly for both cemeteries. The mean for the Black Friars sample was 2.51 (SE=.10, SD=1.54), while that for the Gray Friars sample was 3.68. When the samples were combined, mean number of SD per tooth was 3.01 (SE=0.09, SD=1.77).

The prevalence data indicate that: (1) individuals from both cemeteries had an extremely high prevalence of surface defects, (2) the mean number of defects per tooth was also high in both samples, with the Gray Friars sample experiencing more bouts of illness than the Black Friars, and (3) the population measure of illness as measured by mean number of defects per tooth for all teeth in the samples is similar to the rate for afflicted samples;

both are high. The number of individuals who got sick in these populations was high, and they had relatively frequent bouts of illness.

Pathological striae

Table 5 denotes pathological stria (PS) prevalence in both cemetery samples. One mandibular canine per individual was scored, with left canines sampled preferentially. From the Black Friars sample, 33 individuals were scored for PS. Of those, 10 were afflicted with one or more defects, resulting in a prevalence rate of 30%. In the Gray Friars sample, 30 individuals were scored, with 6 showing one or more defects (Figure 8), indicating a prevalence rate of 21%, slightly lower than the rate in the Black Friars. A scanning electron micrograph image of rod disruption at high magnification clearly shows rod disruption (Figure 9).

The PS prevalence per tooth for the afflicted dental samples is described in Table 6. The mean number of PS per tooth in the afflicted Black Friars sample was 1.6 (SE=.34; SD=1.07). The Gray Friars sample showed a slightly lower rate per tooth, at 1.16 (SE.17; SD=.41). When the samples are combined, the mean number of PS per tooth was 1.43 (SE=.22; SD=.89). The maximum number of PS per tooth in the afflicted samples was 4 in the Black Friars group, in contrast to 2 in the Gray Friars sample.

The population measures of PS prevalence per tooth are less than half of those in the afflicted samples. Table 7 details PS prevalence per tooth for all teeth scored. The mean number of PS per tooth in the Black Friars sample was .48 (SE=.16; SD=.94), while the Gray Friars sample showed a mean number at half of that figure at .24 per tooth (SE=.09; SD=.51). When the samples were combined, the mean number of PS per tooth was .37 (SE=.10; SD=.77).

The prevalence data for PS indicates that the number of individuals experiencing illness that resulted in PS was relatively low, as was the frequency of illness. The values are lower than expected for a historic medieval skeletal sample, particularly in comparison to surface defect prevalence.

MANDIBULAR CANINE DEVELOPMENT: A MODEL

The distribution of striae was calculated for 6 mandibular canines (representing 6 individuals), 4 from the Gray Friars cemetery and 2 from the Black Friars. Three of those teeth yielded adequate visibility of striae through the tooth and were included in the population model. The age, sex, and site affiliations of the teeth used in the model are described in Table 8. Two juveniles from the Gray Friars cemetery ages 11.5 and 14 years and one juvenile age 13 years from the Black Friars sample comprise the model, based on preservation and completeness of the crowns as well as highly visible SOR in thin-sections. One showed evidence of pathological striae, while all had surface defects.

Figure 10 shows the number of striae that were counted in 10 percent sections along the DEJ, from the cusp (starting at 0%) to the cervico-enamel junction (100%). The pattern of stria distribution is similar in each – few striae are seen in the cuspal 3 tenths (0-30%) of the teeth, while the number of striae peak at 65% to 85% of DEJ length. The standard error ranged from over 3 in the 10-20% and 60-70% deciles, to under 1 from 80% to the cervix.

Table 9 details the duration of crown formation in the 3 teeth of the sample. The total number of SOR for the teeth ranged from 187 to 193, with a mean of 190 (SE=2.03; SD=3.51). Assuming that periodicity (counts of cross-striations between adjacent striae) for the model was 8, crown growth was calculated to occur over a period of 1520 days, or 50.6 months (see Table 10). The overall development of the mandibular canine in 10% increments of the DEJ is modeled in Figure 11. Growth clearly slows as the cervical portion of the crown

is reached (similar to Reid and Ferrell, 2006). Figure 12 shows that the cumulative duration of crown development over the growth period is non-linear and slows as growth progresses, and full crown height is achieved. No development is shown in the first ten percent of the DEJ as the mean number of countable striae in this section was 0.

ESTABLISHING TIMING OF ENAMEL DEFECTS

Scatter plots of the raw data from each cemetery sample (Figs. 8and 9) reveal, as expected, a curvilinear relationship between the internal and external ends of SOR. The regression equation that best fits the Black Friars sample is a quadratic equation written as:

$$y = -.353 + .970x - .027x^2$$

where y is a location along the DEJ and x is a location from the cerivo-labial junction along the external surface of the tooth.

The equation was based on 84 pairs of data points (Table 11). Curve-fitting with a quadratic equation for the Gray Friars resulted in the following function:

$$y = -.038 + .831x - .009x^2$$

The quadratic regression was based on 79 pairs of data points (Table 12). Both plots reveal that error increases as SOR occur further from the cervical margin (the zero point at both axes). Figure 13 reveals that this pattern is more exaggerated in the Black Friars sample. A 3 df test of equality was conducted on the parameter pairs between the distance equations of the both the Black and Gray Friars samples. The test showed that they are significantly different, with a p- value < 0.0001. For the equality test of the 3 coefficients separately, the p-value increases and ranges from .04 (x^2) to .22 (y). The issue here is whether the statistical significance is meaningful functionally and contributes to real location differences.

The data used to produce the combined Black and Gray Friars distance function model included 163 pairs of data points. The quadratic regression on the combined samples (Figure 14) is written as:

$$y = -.052 + .832x - .011x^2$$

The standard error was 0.343, and correlation coefficient was 0.980. The quadratic equation was chosen so that the data could be compared to published data from other skeletal samples. The medieval distance functions are summarized in Table 13.

A 3 degrees of freedom test of equality of the combined sample distance function to Simpson's (1999) function for the northern Florida populations again yielded significantly differences between the equations at the $p<0.0001$ level. But original data points were not compared as the Simpson (1999) function came from a published source.

COMPARING SD AND PS POSITIONS ALONG THE DEJ

Surface defects

The absolute position of each surface defect was determined in both cemetery samples using sample-specific regression equations. Tables 14 and 15 detail the derived internal locations of surface defects along the DEJ in millimeters in the Black and Gray Friars dental samples, respectively. The locations of defects ranged from 0.31 to 7.55 mm from the cervico-enamel junction (mean 3.42 mm) in the Black Friars sample and 0.50 to 8.33 mm in the Gray Friars (mean 3.55 mm).

Pathological striae

The absolute locations of PS as measured along the DEJ from the cervix in the Black and Gray Friars samples are noted in Tables 16 and 17. The mean absolute locations of PS in the Black and Gray Friars samples were nearly identical, at ca. 4.75mm from the cervix. The relative locations, standardized by dividing by the cervico-enamel junction breadth, were

correspondingly similar at .68 and .69 respectively (Figure 15). (The combined mean absolute location for both cemeteries was .68.) This indicates similar patterns in the average timing of morbidity in both groups that resulted in microdefects based on positional measurements.

Distribution and timing of SD versus PS

Figure 16 shows the distribution of the number of SD by percent of DEJ crown height in the Black Friars Cemetery. The majority of SD in the sample, 174 defects, occurred in the 60-70th percentile of DEJ crown height, followed by the 70-80th percentile, with 144 defects, and the 50-60th percentile at 123 defects. According to the population model, the peak in prevalence would occur at chronological age 30 months (2.5 years), given that canine development begins at age 4.5 months.

The pattern of defect distribution in the Gray Friars sample (Figure 17) is similar, with the largest number of SD, 172, occurring in the 70-80th percent of the DEJ crown height, followed extremely closely by the 60-70th percentile with 170 defects, and the 50-60th percentile with 116 defects. The corresponding ages at peak prevalence in the Gray Friars is between 30 and 40 months (2.5 – 3.3 years). The majority of defects occurred after 40% of the DEJ crown height was achieved.

The distribution of number of PS by percent DEJ crown height is depicted for the Black Friars sample in Figure 18. It should be noted that the range of defects per percentile was narrow, at 0 to 2. Prevalence peaks at the 50-60th percentile at 7 defects and, according to the population model, corresponds to an age of 21 months (1.75 years). Defects were also detected in the cuspal enamel, even in the first 10-20th percent of DEJ crown height (0.8 months).

The distribution of number of PS by DEJ percentile in the Gray Friars sample is depicted in Figure 19. Prevalence peaks at 2 defects in the 50^{th} to 80^{th} percentiles, which correspond to chronological ages 21 to 40 months (1.75 – 3.3 years). Defects were also detected in the cuspal enamel at the 20^{th} -30^{th} percent of DEJ height, corresponding to age 6.9 months.

CHAPTER V

DISCUSSION

The research presented here tested the following hypotheses: (1) the prevalence of stress as defined by enamel microdefects called pathological striae of Retzius is significantly higher than in pre-urban skeletal samples assessed with *similar methods*, (2) stress affected children as young as 6 to 18 months as evidenced by the pathological striae in cuspal enamel, and (3) the pattern of canine crown growth is unique to the study population.

OVERVIEW OF MAIN FINDINGS

Prevalence of pathological striae

The first main finding was that the prevalence of PS was lower than that of the northern Florida dental samples (assessed with a similar method) and lower than in other medieval samples, while surface defect prevalence was extremely high. Unexpectedly, the prevalence of microdefects, or PS, was lower than in Simpson's (1999) prehistoric and groups, despite using the same criteria for identifying the defects. While the Black and Gray Friars samples showed individual prevalence of microdefects at 30% and 21% respectively, Simpson's (1999) early and later preagricultural groups showed greater frequencies of individuals afflicted, at 67% and 36% respectively. For the preagricultural groups combined, Simpson (1999) reported an average of 1.0 PS per tooth, for all teeth combined. This contrasts to the population value for all of the medieval teeth scored, at 0.37 per tooth which does not support the first hypothesis, that the medieval groups would have higher PS prevalence. Simpson's (1999) early contact and mission samples had even higher prevalence

of PS in individuals, at 54% and 83% afflicted, and for all canines scored in each sample, at 0.9 and 1.4 respectively. The general pattern in these groups shows an increase in micro-indicators of growth disruption through time that is not unexpected with aggregation and missionization. What would account for the relatively low prevalence of microdefects in the medieval friary cemetery samples? Intraobserver error could account for some differences, although most of the defects were verified independently by two observers. For example, if one observer saw rod changes at 1000x and the other did not, the stria was not recorded as pathological. This could result in an undercounting of microdefects, given the strict criteria that were used to identify microdefects. Additionally section preparatory and microscopy protocol was similar to Simpson (1999), the sections were produced by the author and not by an independent laboratory, which may have resulted in a compromise in the optical quality of some sections. Another plausible explanation lies in the type of stress experienced by the medieval children of Odense, that they experienced relatively low levels of acute stress but extremely high levels of chronic stress, but that does not explain the extreme disparity in population PS prevalence levels. *It may be more likely that the strict criteria for identification of PS resulted in fewer positive identifications*. FitzGerald and Saunders (2005) argue that there are no fundamental differences in the crystalline structure of Wilson Bands (irregular striae) and SOR, and they refrain from giving a minimal definition of a Wilson band that would reflect all physiological stress events.

Interestingly, the prevalence of hypoplasias (surface defects) in mandibular canines (representing individuals) is much higher in the present study samples (96%) than in other medieval Scandinavian samples, for example, Hanson and Miller's (1997) study of mandibular canines (17.5-21.5%) from medieval Norway, Denmark, Iceland, and Greenland.

Based on these figures, Hanson and Miller concluded that early childhood (defined here as ages 4 to 7 years) was a stressful time for medieval Scandinavians; however, the ages derived in this study are based on published crown development schedules and may be erroneous for populations other than modern Americans.

Thomas (2003) found that linear enamel hypoplasia occurred as the least common enamel microdefect, at a population prevalence of close to 55%, surpassed by weak and strong Accentuated Striae (over 85%) in a sample of canines from the medieval Danish village of Tirup (AD 1100-1350). In additional research on Tirup, Boldsen (2007) found that 120 of 458 skeletal individuals who survived to at least age 6 showed hyploplastic defects (26.2%) and that adult males and young adult females with linear hypoplasia experienced higher mortality those those without hypoplasia. These comparative data point to a disease pattern in medieval Odense in which chronic stress events were extremely common and affected much, if not all, of the juvenile population. These data do not support the first hypothesis, that microdefects would be more prevalent in the medieval Danish samples than in the northern Florida samples described by Simpson (1999).

The relationship between enamel hypoplasias and other stress indicators has been investigated in several medieval Scandinavian skeletal assemblages. Brinch (1959) noted that enamel hypoplasias occurred in the dentition of several individuals buried at the medieval Salernitan Augustinian monastery of Æbelholt, ca. AD. 1175-1561, near Copenhagen on the island of Sjælland. The individuals buried on the monastery grounds are assumed to include abbots, friars, and lay-brothers, as well as inhabitants of the nearby countryside and the sick who were treated by monks in the monastery's infirmary. The majority of the individuals with hypoplastic enamel lesions exhibited only faint transverse lines in the incisors and canines.

However, Brinch notes that the absence of more severe hypoplastic lesions may be due to the significant degree of wear on the teeth.

Swärdstedt's (1966) dissertation on the odontology of the skeletal individuals from medieval Westerhus (Västerhus) churchyard, AD 1025-1375, in Sweden is perhaps the most exhaustive dental study of a medieval Scandinavian population. Dental caries, calculus, attrition, ante-mortem tooth loss, and enamel hypoplasia were analyzed and quantified in individuals with well-preserved dentition (n=132). Enamel surface defect prevalence in the Westerhus dentition was found to be "high." Defects occurred commonly as bilateral transverse bands, the majority of which were formed between the ages of 2.5 and 4 years. When the skeletal population was broken down by age category, hypoplasias were the most prevalent in individuals who died young. Mature individuals (ages 40-60 years) exhibited the least number of hypoplasias; however, attrition may have had a role in obscuring actual hypoplasia prevalence in the mature individuals.

Microdefects

The second main finding was that microdefects occurred in the friary samples in individuals prior 18 months of age. The timing of defects was also different between the medieval samples and those of the northern Florida groups. Simpson (1999) found that PS in peaked in children prior to age 24 months, while SD peaked at age 45 months. Using the same methods, this study found that PS peaked at age 21 months in the Black Friars and at ages 21 to 40 months in the Gray Friars sample, while SD peaked at between ages 30 - 40 months in both medieval dental samples. The pattern of a peak in PS prevalence earlier with a later peak in SD prevalence suggests, as did Simpson (1999), that the defects do have different etiologies. This in itself is expected, as hypoplasias occur only in lateral (later forming enamel); however, the PS can occur anywhere in the enamel but were not found to

peak in coincidence with hypoplasias. PS occurred in the medieval groups between the 10th and 30th percentiles of DEJ crown height (up to 7 months), while most SD occurred after the 40th percentile. The prevalence of microdefects in cuspal (hidden) enamel extends the defect chronology into infancy. These data do support the second hypothesis of the study, that enamel defects will be detected in children prior to 18 months of age when microdefects are assessed. FitzGerald and authors (2006) have clearly documented microstructural enamel defects in nearly 40% of their deciduous dental sample from Imperial Roman infants. These data suggest the importance of looking at microstructural defects to establish a more accurate picture of stress profiles of infants and young children in the past.

The total duration of mandibular canine crown formation based on total stria counts also differed in the medieval and northern Florida samples. While the crown was found to develop in 50.6 months (1520 days) in this study, Simpson's (1999) analysis of the northern Florida groups revealed that the crown developed in 60-63 months. He denoted that this duration of development is shorter than the 78 month period used in some published samples (see Goodman and Rose, 1990). While the duration of crown growth in the friary samples appears comparatively short, Reid and Dean (2006) found that mandibular canines (n=67) from the medieval Danish village of Tirup (AD 1100-1350) completed formation, given an initiation age of 200 days, in 62.3 months (formation time was based on separate calculations of cuspal and lateral enamel formation using average daily secretion rates and perikymata counts). The lateral enamel completed formation in 1588 days, or ca. 53 months. Reid and Ferrell (2006) found a similar imbricational crown formation time based on SOR numbers and periodicities in their sample of mandibular canines (n=49) from Tirup. The average formation time was 1594 days, or 53.1 months. The mean number of SOR in the

imbricational enamel of the Tirup canines was 190.3 (range 142 - 257), while the total number of SOR for the entire course of the enamel along the DEJ in this study was 190 (range 187 – 193). This suggests that the duration of crown formation in the mandibular canine may be somewhat plastic among and within populations, that methodology (including sample size or sectioning method) may be yielding different crown formation estimates, or that decussation in the cusp tip may be preventing identification of some striae. Watt and Lunt (1999) found variation in the age estimations of different teeth in juveniles from the medieval Whithorn site in Scotland, regardless of the aging standard used (see Massler et al., 1941; Ubelaker, 1989; Demijirian et al., 1973, 1976; Smith, 1991). Because the variation was directional, the authors decided to test whether the relative stages of development in the medieval group were different for those in modern North Americans, on whom the aging criteria was based. Using the first molar as a reference tooth against which the development of other teeth were compared, Watt and Lunt (1999) compared the development of the Whithorn dentitions to that of Native Americans (Moorrees et al., 1963) and modern French Canadians (Tompkins, 1996).

Mandibular canine crown geometry

The third main finding was that the hypothesis that the pattern of mandibular canine crown geometry is unique to populations was weakly supported. The external –to-internal distance regression equations of the medieval Scandinavians and the prehistoric and historic northern Florida samples described by Simpson (1999) were significantly different, but the differences may not be functionally significant. For example, a 6mm external defect distance from the cervico-enamel junction would yield and internal distance of 4.68mm with Simpson's (1999) population equation and 4.65mm with the combined medieval Danish model. This suggests that, at least in this case, there is weak evidence for genetic or

environmental inter-populational variation in the geometry of how growth lines are manifested from DEJ to surface. The third hypothesis, that the medieval populations are unique in their canine crown growth pattern, was only weakly supported.

Sources of childhood stress in medieval Scandinavia

The setting: The medieval town of Odense. The study sample is derived from two friary cemeteries, dating from ca. A.D. 1250 to 1536, in the Danish town of Odense.[8] At the beginning of this period, Odense was experiencing a new urban phenomenon that was sweeping all of Europe - the rise of the urban monastery and the birth of the Catholic friars-preachers. The town was a crucial element in the success of the friary movement because it allowed the friars to preach to hundreds of people in one, densely populated location (Lawrence, 1994; Lynch, 1992). The friaries brought new hope for salvation to the townspeople – and an alternative to parish church burial. In part because of its status as an ecclesiastical center, Odense was a growing town in the early high medieval period, with a population estimated to be over one thousand inhabitants by A.D. 1300 (Sawyer and Sawyer 1993).[2] A merchant class had emerged in towns all over Scandinavia by this time, fostering specialized production of goods for seasonal markets and trade with the Scandinavian kingdoms, the Germans, the Baltic, and other parts of Western Europe (Sawyer and Sawyer, 1993). Odense was governed by a town mayor (*Danish*., borgmester) and council. And the city had all the accoutrements of a typical northern medieval town, including an open sewage

[8] The medieval period in Denmark extends from A.D. 1050, the end of the Viking Age, to A.D. 1536, the time of the Lutheran Reformation. The skeletal sample spans a period of 300 years, from ca. A.D. 1250 to 1536, and dates from the high to late Middle Ages in Scandinavia. The medieval period in Scandinavia is significantly later than in continental Europe.

[2] The Danish medieval population was at its maximum between A.D. 1250-1300 at just under 1 million people, although this estimate is considered inaccurate by some scholars (Benedictow 1993). The exact population of Odense is unknown, although the largest towns in medieval Scandinavia were Stockholm, Sweden and Bergen, Norway, with ca. 7000 inhabitants each (Sawyer and Sawyer 1993).

system, an irrigation system, numerous wells, cobbled and dirt roads, home gardens and pens, wooden and brick residences, watermills, municipal buildings, workshops for smithies and cobblers, hospitals, schools, churches, and cemeteries (Christensen, 1988). The appearance of the Black Plague in Denmark in A.D. 1348, coupled with famines throughout the fourteenth century, effected the demise of smaller towns in Scandinavia. But Odense survived and even prospered into the 15th and 16th centuries, enlarging the fortified areas of the town with inner and outer gates, building more ecclesiastical institutions and enlarging a city hospital. The Lutheran Reformation in A.D. 1538 brought with it changes in the use of, and in some cases, disestablishment of, Odense's ecclesiastical buildings. Otherwise, the appearance that the town took on in the 16th century was maintained by in large for the next several hundred years, into the mid-20th century (Christensen, 1988).

MEDIEVAL SCANDINAVIAN DISEASE ENVIRONMENT

What diseases were prevalent?

Bioarchaeological analyses of skeletal remains are a major source of information about morbidity and mortality in Scandinavia. Some of the earliest paleopathological research in Denmark was conducted by Vilhelm Møller-Christensen (1958), who described the pathological skeletal lesions of those buried in the churchyard at Æbelholt Kloster , a 12th century medieval Augustinian monastery located in Sjælland, DK. He found evidence of localized trauma, arthritis and subluxations, dental caries, and scurvy as well as skeletal indicators of chronic diseases such tuberculosis, non-venereal syphilis, and ergotism ("St. Anthony's Fire"). There is no doubt that leprosy was also prevalent in Scandinavia; the leper hospital cemetery site known as Skt. Jørgensgård in Odense shows clear evidence of the skeletal changes associated with leprosy (see Anderson, 2000). Evidence of acute, fast-acting diseases is much more rare archaeologically (hence, a need for analyzing pathological striae

in the enamel). English sources describe diseases such as cholera, typhoid fever, malaria ("the ague"), smallpox ("red plague"), diptheria, dysentery ("bloody flux"), diarrhea ("squirt"), scurvy, and anthrax ("black bane") (Bishop, 1968; Hays, 1998), which have no skeletal correlates. Additionally, some chronic conditions result only in non-specific stress indicators (like enamel hypoplasia). This means that these pathologies are not attributable to a specific disease process but are indicative of generalized metabolic stress. Swärdstedt (1996) found that dental indicators of stress in the form of enamel hypoplasias were most common in the individuals believed to be slaves, while Hölder-men (land tenants) exhibited fewer hypoplasias and the highest ranking group, Länder-men (literally, "landed men," or land owners), exhibited the least lesions. Swärdstedt (1966) suggested that differences in diet, housing conditions, and infectious disease prevalence affected health status of the different social groups, possibly reflected in differential hypoplasia prevalence. Milk may have been a food reserved for higher social groups, and a number of skeletal individuals from Westerhus show signs of malnutrition, including rickets. The role of infectious disease in producing health stress is also assumed to be important given that 50% percent of the 364 individuals from the churchyard died before age 7 years. Swärdstedt attributed this high juvenile mortality to infectious disease, which children from the slave class would be least able to combat. Unlike Swärdstedt (1966), Tkocz and Brøndum (1985) calculated the prevalence of enamel hypoplasias in the skeletal individuals from a Franciscan cemetery (ca. A.D. 1236) in the medieval town of Svendborg on Fyn, Denmark, and found no relationship between social status and defect prevalence. Unfortunately social class has not been established in the Black and Gray Friars skeletal assemblages, so distinctions in disease prevalence by guild or other social grouping cannot be made.

Disease in urban environments

Susceptibility to epidemic disease characterizes the difference between disease risk in urban versus rural medieval Scandinavian communities. Boldsen (1984) compared the demographies of a skeletal population from the 11th-12th century rural agrarian cemetery site of Löddeköppinge in Scania, Sweden to that from the 12th-16th century urban parish site of Lille Sct. Mikkelsgade in Viborg, Denmark. He found that age-specific mortality rates were high among children and young adults from the urban site, while mortality rates were higher among mature and older people from the rural community. These results support the idea that urban endemic disease effectively kills children and young adults, while occasional epidemics impacting more sparsely populated rural groups affect all parts of the population equally (resulting in the higher adult mortality rates at Löddeköppinge).

Medieval diet

Barley was of major dietary importance to the majority of people living in medieval Scandinavia. Barley was the staple cereal in the medieval diet and was used to brew ale, the staple drink for adults and children, to make porridge, and, along with rye, to bake bread. Wheat was also produced, but in much smaller quantities, as only the wealthy consumed it (Skaarup 1993). Written sources make little mention of large-scale vegetable agriculture, but there are references to cabbage, beets, onions, peas, beans, and endives (all suited to growing in cooler climates).

Herbs were grown in medieval Scandinavia, including dill, parsley, horseradish, cress, mint, thyme, and marjoram (Skaarup, 1993). Spices were available via importation, but were expensive. Hops and bog myrtle were used to flavor beer while honey and sugar were used commonly as sweeteners. Vinegar, mustard, and salt were also common additives to foods. Wild foods were also an important part of the diet. They included wild fruits and

berries such as apples, pears, cherries, blueberries, sloes, strawberries, and raspberries, as well as hazelnuts and wild caraway. Fruits like figs, oranges, and grapes could be imported, but, like spices, were expensive and probably not a regular component in the diets of commoners (Skaarup 1993).

Brinch (1959) and Sagne (1990) agree that attrition during the medieval period in Scandinavia was directly correlated with the denaturalized state of foodstuffs consumed and that, not unexpectedly, the level of dental attrition in the Middle Ages was much higher than that in modern Scandinavian populations. Furthermore, Brinch (1959) notes that grain grinding was "primitive" in the Middle Ages and that neither refined sugar nor potatoes existed in Scandinavia at the time.

Medieval Scandinavians also consumed a variety of animal products. Generally faunal remains in urban areas in medieval Scandinavia (located within agricultural lands) indicated the slaughter and consumption of cattle, followed by sheep and pigs. These animals were often slaughtered in the fall to avoid the cost of feeding them through the winter. Fowl provided a protein source for winter (hens, geese, ducks) in the form of meat and eggs. Milk, cheese, and other dairy products were processed from cows, sheep, and goats (Skaarup, 1993).[3]

A major transition in the diet of medieval Danes occurred with the arrival of the plague and the agrarian crisis and periodic famines in the 14th century. Kjersgaard (1978) hypothesized that the amount of cultivated land decreased through the 1300s and 1400s, accompanied by a shift in diet from equal portions of grains to meat in the 1200s to an increasing dependence on meat and livestock raising in the 14th and 15th centuries (such that

[3] Although regular fasting requiring abstinence from meat compromising about 180 days of the year, children weren't usually subject to the same restrictions (Orme 2001).

the consumption ratio of grains to meat became 1:2). Others sources indicate an increase in cattle farming in the 14th and 15th centuries (Sawyer and Sawyer, 1993; Fenger, 1993). Fenger (1993) notes that the emphasis on cattle husbandry may explain, in part, farm abandonment prior to the plague epidemics.

Even in cities maintaining large numbers of livestock and local garden plots, medieval Scandinavians were still dependent on outside sources for food. Thus, adequate food preservation and storage was crucial. Some vegetables and fruit were dried or preserved in honey or sugar, while meat and fish were smoked, dried or salted (Skaarup 1993).

THE LIVES OF MEDIEVAL CHILDREN IN NORTHERN EUROPE

The medieval concept of childhood

In light of the findings in this study that acute stress episodes were detected in children prior to 18 months of age and well after, it is valuable to assess what roles children played in medieval Northern European society that may have furthered their exposure to physiological and psychosocial stressors.

For the last four decades, the prevailing view of childhood in medieval Europe has centered on the arguments of Ariès (1962), that medieval society did not recognize childhood as a distinct period culturally, and that adults viewed children as little more than small, inadequate versions of themselves. Orme (2001) recently countered these views, suggesting that medieval Europeans viewed childhood as a distinct phase(s) of life. This notion relies in part o the medieval concept of the ages of man, which recognized the ages of three and seven as particularly important for children because they marked the time of weaning and the end of infancy. In addition, Orme (2001) presents documentary evidence that legal and religious entities recognized childhood as separate from adulthood in medieval England by A.D. 1200. In the later Middle Ages, English children aged seven and up were deemed by the church and

by the law to be old enough to be formally charged with crimes, to be engaged to be married, to be tonsured as clerks, and in the case of some girls, to be sexually active. Historical evidence documenting these circumstances is unusual, possibly indicating that the age of seven may have been more symbolic than actual in marking the transition from infancy to childhood (Orme 2001). In support of this modern concept of childhood in medieval Europe, Hanawalt (1993) argues that, in the 13th and 14th centuries, the English became preoccupied, and even obsessed, with preparing their children for passage into the adult world, as evidenced by the number of advice manuals for child-rearing and the change in English laws to include children. Sentimentality for youth is also evident by the existence of medieval English phrases that refer to "the tender years of youth" (Hanawalt, 1993). And medieval adults appear to have recognized a distinct culture of young children that included significant amounts of playtime and an accompanying material culture of toys and children's furniture (Orme, 2001).

Infancy and early childhood

The birth of children in Britain and Scandinavia was a notable event in all households and celebrated with elaborate baptismal and naming ceremonies, involving both parents and godparents (Hanawalt,1993; Jacobsen, 1993). Children had, in fact, layers of identity that began in the baptismal ceremony with their christening. The godparents and possibly a nurse or wet nurse (in wealthier families) were all children's first social network outside of the family, followed by the members of the parish in which their baptism occurred, and later the family of apprenticeship or service (Hanawalt, 1993). Jacobsen (1993) reports that numerous ceremonies surrounded the birth of a child in medieval Scandinavia, including the "kvindegilde," a celebratory feast for the female helpers at the birth. From the 13th to 19th centuries, the kvindegilde was known to be a rowdy affair, with eating, drinking, dancing,

and mocking a straw figure dressed as a man. Baptism feasts including men and women followed shortly after birth. And after a recovery period, the new mother participated in the "kirkegang" (church-going), a religious ceremony celebrating her recovery and sometimes demonstrating her social status.

In England, urban children had an extended system of adult care in the proximity of neighbors and parishioners, despite the health risks that other facets of city life entailed. An infant's mother was its primary caretaker, along with a nurse and other members of the household. For the first year of its life, a child would be kept at home, swaddled in a cradle. Infants were nursed by their mothers or by a wet nurse hired by the family (Hanawalt, 1993).

English infants were weaned from breast feeding between ages one and three years (Hanawalt, 1993). The analysis of diet in juvenile skeletal remains from the medieval Swedish site at Westerhus (A.D. 1100-1350) and from 12th century medieval Schleswig, northern Germany supports this assertion. According to trace element analyses, Iregren et al. (1993) and Hühne-Osterloh and Grupe (1989) conclude that weaning likely occurred before age two years. After age 2, children were fed with plant-based foods. Orme (2001) notes that English mothers and nurses would masticate food and then feed it to children without teeth. Babies were also fed a kind of gruel called "pap" made from animal milk or water mixed with meal or bread. Dietary transitions like this could potentially account for the prevalence of defects seen in the Odense populations between infancy and early childhood. At age three to four years, children were given small beer (a second brewing of ale) because it was believed to be more nutritious than water. Bread, hard-boiled egg yolks, and pared apples were also recommended for toddlers (Orme, 2001). Beer and wine were common drinks at all meals, even for children. Hanawalt (1993) notes that moralists of the time dictated that

children were to have "only two or three drinks of wine or beer during a meal because beverages deformed their minds and caused an unreasonable diet" (1993: 73). It was far more likely that wine was had by wealthier families, and beer, the standard for the less fortunate. For example, the 1513 household book of the Percy earls of Northumberland recorded that the older children were to have for breakfast household bread, wheat bread, two pints of beer, and meat in the form of chicken or boiled mutton bones. The children in the nursery were given wheat bread, only one pint of beer, and boiled mutton bones. Dinner would include butter and fresh fish. The child servants in the earl's chapel (in keeping with their lower social status) were fed a different diet. They were given household bread only - probably a barley rye bread - beer, and boiled beef for breakfast. And servant children ate salt fish, never fresh fish, on Fridays and at dinner (Orme 2001).

Children remained in and around the home through their first few years, eventually contributing to running the household. Children as young as age 4 were given chores such as fetching water for the household from nearby water sources or helping in other simple chores (Hanawalt, 1993; Orme, 2001).

Later childhood: Children as economic contributors to medieval society

Children actively contributed to the economic well-being of medieval society as apprentices and servants. By age 12 years, most children left their natal homes to live with and work for their employers. Both apprenticeship and service were life stages, transition periods between childhood in the natal home and independent adulthood during which adolescents learned skills and accumulated wealth for their future roles as adults. Entering an apprenticeship meant working in a shop all day, learning a craft (such as shoemaking, smithing, or baking) from a master, and preparing ultimately for life as a full-fledged guild

member (Hanawalt, 1993).[4] The majority of merchant and craft guilds were established in Scandinavian cities in the 14th and 15th centuries (Jacobsen, 1993). Apprenticeships were privileged positions because they offered an opportunity for upward mobility, stable income, and a secure future. Apprentices had an important role in the direct production of goods and made a significant contribution to the economy. In some cases, an apprentice married into his master's family and succeeded him in the business (Hanawalt, 1993).

Adolescents whose families could not afford to make them apprentices sent them into service instead. Although servants, like apprentices, had a contract with their masters normally lasting 7 to 10 years, they far outnumbered apprentices in the city. Younger servants hoped to work out their contractual term in their masters' households to accumulate dowry money for marriage or enough money to return to their villages outside the city to set up their own households. This was not commonly achieved by less skilled servants, and some become career servants, working as scullery maids, house and shop cleaners, etc. Those most skilled worked, for example, as journeymen or skilled sewers who waited personally on the master; more moderately skilled servants worked in baking, brewing, and selling food and drink. It is important to note that service drew people from a wide range of social backgrounds, although children going into service were generally from less well-to-do families than those entering apprenticeships (Hanawalt, 1993).

Service became particularly important after the Great Plague of AD 1348-49. As population decreased, the labor demand increased; and apprentices and servants were in shorter supply. Masters in medieval London went to extremes to secure child servants, from

[4] Guilds are defined as corporations of merchants and craftsmen. They developed in medieval Europe as an urban phenomenon.

kidnapping them to enticing them to break existing contracts, both of which were prosecutable offenses (Hanawalt, 1993).

Other insights into children's risk of death

Naming practices may give some insight into the risk of death for children in medieval England. It was not uncommon for two or three children in a family to bear the same Christian name - evidence to some historians that parents were aware of the likelihood that one or more of their children would die before adulthood and that naming two children the same would ensure at least one child might survive to preserve the name (Hanawalt, 1993).[5]

How a child was treated and whether he or she lived or died may have been dependent, in part, on the child's sex. Hanawalt (1993) cites an increase in female mortality, according to wardship cases in London in the fourteenth and fifteenth centuries. Hanawalt suggests that male children in medieval London may have been given better care because of their higher social value; males brought wealth into the family through marriage while female children required dowry from their families for marriage. Hanawalt (1993) cites a division of labor by sex attached to children aged 2-3 years, as evident from urban and rural coroner's inquests of accidental deaths; most boys were killed outside the home, while girls died inside. By age 4, parents began to discipline children to make them fit for proper society. Corporeal punishment was standard and even recommended in some cases, although maliciously beating a child without reason was discouraged (Hanawalt, 1993).

[5] Hanawalt (1993) presents an alternative to this sentimentality for names. The most common naming practice in medieval England was for a child's god-parents to name him or her. Two children in a family may easily have different god-parents with the same name, especially if the name was common.

CONCLUSIONS

This dissertation presented a justification for using a population-specific method for assessing stress via macrodefects and microdefects in two skeletal samples from medieval Denmark. A method developed by Simpson (1999) was employed to increase the accuracy of the timing estimation of defects by determining locations along the DEJ for both internal and external defects.

The methods presented here offer three distinct advantages:

(1) "Hidden" cupsal enamel is taken into account, along with lateral enamel to assess growth disruption via enamel defects. This lengthens the window of time available for assessing stress that analyses of surface defects, or hypoplasias. Canine crowns present a significant period of time that allows evaluation of stress in the prior to age 1.5 years if the cuspal enamel is taken in to account.

(2) By employing a histological method (after Simpson, 1999) that uses counts of regular growth lines along the dento-enamel junction from the cusp tip to cervix, a population-specific model of crown duration and rate of growth can be generated. This solves two problems – (a) it circumvents the problem of grafting on a model from an entirely different population, which may not be accurate and (b) it allows for much greater accuracy in terms of timing normal growth and episodes of growth disruption in days that linear crown development models and radiographic studies do not have. This is not to say that standards can't be developed, or that every skeletal collection requires its own model for growth and pathology timing – it is important to recognize that even genetically rigid growth may vary through time and space, that it can be contextual and that we can do something more to elucidate the variation that likely existed in children who experienced stress hundreds or even thousands of years ago.

(3) By using a population-specific method for timing defects that was not as labor- or time-intensive as other methods for determining duration and rate of canine crown development (for example, the method for calculating cuspal formation time using periodicities and measures from the dentin horn, added to imbricational enamel formation using histological SOR or surface perikymata counts in a large sample of teeth) this method is potentially more user-friendly in non-dental laboratories. The value lies in using a small sample size that utilizes counts of SOR throughout the crown, in cuspal, mid-crown, and cervical portions. This makes the method potentially useful for bioarcheological analyses which may have neither the time nor the budget to do large scale dental studies that require sending dental samples to another source for processing and analysis.

Overall the hypotheses of this research were only partially supported by the findings: (1) PS did occur in infancy, expanding the stress profile to include ages that would not be included with analysis of surface defects only; (2) using the same method for estimating crown development duration, timing, and criteria for defect identification as Simpson (1999) , PS prevalence was unexpectedly found to be much lower (and surface defects, higher) in samples from the Odense friaries versus those of northern Florida (Simpson, 1999); and (3) comparison of the distance functions (representing enamel growth geometry) were found to be significantly different in the Odense and northern Florida samples. While statistically significant, the differences may be not be functionally significant.

One potential issue with the method for calculating crown development may come to the lack of being able to detect and count striae of Retzius in the first 10% of DEJ length from the cusp, thereby compacting (in small part) the full time to complete development. The crown grows very quickly in this area, and the SOR may be difficult to detect due to

decussation (Risnes, 1986). It may be useful in the future to compare total cuspal enamel formation time estimated by the method of Reid and Dean (2006) to counts of SOR in the Black and Gray Friars samples to determine how much time is not accounted for in the first 10-20% of DEJ height. Other suggestions for future research include: (1) as periodicities may vary in different parts of the crown (Reid and Ferrell, 2006), it may be beneficial to determine periodicities by decile rather than using a modal value as in this study; (2) a larger sample size for a population model may be necessary, as the range of SOR counts was narrow; and (3) re-evaluation or a relaxation of the criteria for determining what constitute an accentual, irregular, or pathological stria of Retzius may be useful, if the conjectures of Fitzgerald and Saunders (2005) are correct.

What do the methodological contributions tell us ultimately about childhood health in medieval Odense?

Dietary transitions in infants and toddlers: Weaning stress

It is critical to note that the use of these methods helps to highlight the prevalence and timing growth disruption in medieval Danish children to enhance the understanding of how medieval children in Odense coped with the tumultuous time period between AD 1250 and 1539. Stress during childhood in the groups examined here can be described as nearly *universally chronic* (as represented by enamel hypoplasia), with much fewer acute episodes (as represented by PS), in comparison. While traditional arguments of stress have attributed peak stress to the "weaning period" of age 2-3 years in children in a number of archaeological populations, historical sources have indicated that weaning occurred between 1 and 3 years in, for example, medieval England (Hanawalt, 1993). It was also not surprising that surface defects were peaked at ages 2.5 to 3 years in the Odense friary groups, as the enamel that develops along the first 30% of the DEJ from the cusp is hidden and never

reaches the surface to manifest defects. In comparison, hypoplastic defects in Slavonic populations from the 9th to 10th centuries AD had a prevalence rate of over 80% in the permanent dentition of children. Because these children had skeletal age determinations of 2 to 4 years, the defects were described by the authors to indicate weaning stress (Velemínský et al., 2009). Similarly, Saunders and Keenleyside (1999) found the linear enamel hypoplasias peaked in the mandibular canine at 3.5 years in the 19th century St. Thomas Anglican Church cemetery sample; but they argued that this peak timing had no correlation to the cessation of breast feeding or to the weaning process in general, based on isotopic and demographic data that suggested that breast feeding duration was 14 months and exogenous food was first introduced to infants at circa 5 months. This research demonstrates that one should be careful in assigning surface defects at ages 2.5 – 3 years causally to weaning stress only, although it certainly may be an important factor.

The earliest PS at 5.3 and 6.9 months, may possibly be attributable to weaning stress, although it is not known when exogenous foods began to be introduced into the diets of nursing infants in Odense. The mixing of cereal with potentially contaminated water to form a gruel during weaning certainly could have made the dietary transition for infants more taxing. Weanling diarrhea, one of the most common causes of acute infant morbidity and mortality in living populations, could have resulted in acute disruptions to enamel growth (see Simpson, 1999). The data on peak prevalence of PS suggest that children just under 2 years old experienced acute stressors in the Black Friars sample while children just under 2 years to 3.3 years old in the Gray Friars sample experienced acute episodes. They all survived these episodes and manifested surface defects as well, at a mean age peak of 2.5 to 3.3 years, overlapping with mean ages at peak PS prevalence. Acute stressors at these ages

may be associated with weaning stress, or even the cessation of breastfeeding itself (Dirks et al, 2010). Hanawalt (1993) suggests medieval mothers weaned their children between 1 and 3 years old, so weaning stress, in combination with a host of other factors, including disease and nutrition, could have resulted in a combination of acute and chronic stress indicators.

Infectious disease stress and starvation

While it commonly thought that the plague was the most important determinant of population devastation and social and economic breakdown in northern Europe, Ormrod and Lindley (1996) argue that medieval society had a level of familiarity to the natural and social disease of plague, having lived through multiple famines and periods of starvation and malnutrition. While the first devastating episode of plague hit in AD 1348, a second outbreak in England occurred in AD1360 and was named the "Children's Plague,' after the section of the population that it hit hardest. Overall, the death rate of this second plague outbreak was less than the first, and the population growth was essentially halted Given the specific timing of plague outbreaks and the high mortality rate, it is unlikely that any of the enamel defects in the Odense populations can be attributed to these specifically to these events. More likely to have been critical in causing acute and chronic stress in Denmark are the periodic famines like the Great Famine of AD 1315-1317 and the many famines that followed in the 14 century, which entailed crop failure, farm abandonment, and sometimes child abandonment. Famine itself is a risk factor for a host of other negative health effects and may have been more significant overall than disease waves in impacting the health of children. The effects of starvation on nursing mothers' ability to produced milk and feed their children is certainly a possible cause of infant and toddler nutritional stress that should be considered in the timing and prevalence of enamel defects in the Odense groups.

Despite this continual risk of disease, malnutrition, and starvation, more than 85% of the skeletal individuals comprising the Odense friary samples *survived childhood*, providing a window for bioarchaeological research on the types of stress they were enduring and surviving (continuously chronic with comparatively fewer acute episodes).

Issues for future research

One potential issue with the method for calculating crown development may come to the lack of being able to detect and count striae of Retzius in the first 10% of DEJ length from the cusp, thereby compacting (in small part) the full time to complete development. The crown grows very quickly in this area, and the SOR may be difficult to detect due to decussation (Risnes, 1986). It may be useful in the future to compare total cuspal enamel formation time estimated by the method of Reid and Dean (2006) to counts of SOR in the Black and Gray Friars samples to determine how much time is not accounted for in the first 10-30% of DEJ height. Other suggestions for future research include: (1) as periodicities may vary in different parts of the crown (Reid and Ferrell, 2006), it may be beneficial to determine periodicities by decile rather than using a modal value as in this study; (2) a larger sample size for a population model may be necessary, as the range of SOR counts was narrow; and (3) re-evaluation or a relaxation of the criteria for determining what constitute an accentual, irregular, or pathological stria of Retzius may be useful, if the conjectures of Fitzgerald and Saunders (2005) are correct that significant rod disorganization is not necessarily a component of PS. Finally, a potentially fruitful area of research in the future would be to determine if there is a correlation between skeletal pathologies and enamel defect prevalence in the friary assemblages to shed further light on the types of physiological stressors that medieval children encountered. Analysis of microdefects in the deciduous

dental assemblage would also provide a clearer picture of the timing and etiology of stress, particularly in infancy.

Appendix A

Tables

Table 1. Sex and age of individuals from both cemeteries scored for surface defects

Cemetery sample	No. Adult male	No. Adult female	No. adult unknown	No. juvenile (< 15y)	Total
Black Friars	102	100	7	25	234
Gray Friars	74	79	3	20	176
Combined	176	179	10	45	410

Table 2. Surface Defect Prevalence per Cemetery Sample

Cemetery Sample	No. Individuals Scored (n=)[a]	No. afflicted with 1+ defect	% afflicted with 1+ defect
Black Friars	234	218	93
Gray Friars	176	174	99
Combined	410	392	96

[a] One mandibular canine per individual was scored; left canines were sampled preferentially.

Table 3. Surface Defect Prevalence per Afflicted Teeth

Cemetery sample	Mean No. *SD* per tooth	*SE*	*SD*	Min. No. *SD* per tooth	Max No. *SD* per tooth
Black Friars	2.69	0.10	1.43	1	7
Gray Friars	3.72	0.14	1.80	1	9
Combined	3.15	0.09	1.68	1	9

Table 4. Surface Defect Prevalence per All Teeth Scored

Cemetery sample	Mean No. *SD* per tooth	*SE*	*SD*	Min. No. *SD* per tooth	Max No. *SD* per tooth
Black Friars	2.51	0.10	1.54	0	7
Gray Friars	3.68	0.14	1.83	0	9
Combined	3.01	0.09	1.77	0	9

Table 5. Pathological Stria (PS) Prevalence per Cemetery Sample

Cemetery sample	No. individuals scored (n =)[a]	No. afflicted with 1+ defect	% afflicted with 1+ defect
Black Friars	33	10	30
Gray Friars	29	6	21
Combined	62	16	26

[a] One mandibular canine per individual was scored; left canines were sampled preferentially.

Table 6. Pathological Stria (PS) Prevalence per Afflicted Teeth

Cemetery sample	Mean no. PS per tooth	*SE*	*SD*	Min. No. PS per tooth	Max No. PS per tooth
Black Friars	1.6	0.34	1.07	1	4
Gray Friars	1.16	0.17	0.41	1	2
Combined	1.43	0.22	0.89	1	4

Table 7. Pathological Stria (PS) Prevalence per All Teeth Scored

Cemetery sample	Mean no. PS per tooth	*SE*	*SD*	Min. No. PS per tooth	Max No. PS per tooth
Black Friars	0.48	0.16	0.94	0	4
Gray Friars	0.24	0.09	0.51	0	2
Combined	0.37	0.10	0.77	0	2

Table 8. Sample canines comprising the population model for canine growth

Grave no	Age	Sex	Burial location	# PS	# SD
GP90-090	11.5	J	Square C	0	3
GP90-254	14	J	Square D	0	9
SBT81-015	13	J	West Wall	1	2

Table 9. Striae of Retzius counts in 10% percent increments of DEJ crown height

Grave no.	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90	90-100
SBT81-015	0	10	9	10	17	22	36	33	34	22
GP90-090	0	0	3	11	23	26	27	40	35	25
GP90-254	0	0	6	6	17	23	38	39	35	23
Mean # SOR	0	3	6	9	19	24	34	37	35	23

Table 10. Canine crown development timing based on population model from Gray and Black Friars Samples

% DEJ length	10	20	30	40	50	60	70	80	90	100
Months	0.00	0.80	1.60	2.40	5.07	6.40	9.07	9.87	9.33	6.13
Cumulative months	0.00	0.80	2.40	4.80	9.87	16.27	25.33	35.20	44.53	50.67
Chronological age (months)	—	5.30	6.90	9.30	14.37	20.77	29.83	39.70	49.03	55.17

Table 11. External and internal positions (mm) of striae of Retzius in the Black Friars sample

External position	Internal position
1.38	0.9
2.09	1.49
3.57	2.67
2.44	1.88
3.49	2.65
1.92	1.43
5.9	3.87
3.53	2.79
4.57	3.12
4.78	3.7
1.91	1.51
6.03	4.43
1.93	1.68
1.46	0.99
2.33	1.69
5.22	4.27
2.61	2
3.26	2.47
2.75	2
5.47	3.7
3.95	3.26
7.57	4.71
5.46	4.07
2.37	1.86
4.4	3.43
3.18	2.64
1.68	1.16
2.65	1.94
8.12	6.32
3.94	2.98
4.3	3.41
4.48	3.5
6.96	4.44
5.51	4.64
8.71	5.42
5.87	4.34
2.61	1.97
6.86	4.75
5.17	4.39
3.57	2.37
2.76	2.21

External position	Internal position
4.55	3.48
4.76	3.78
5.04	3.87
7.76	4.83
5.92	4.8
6.38	4.66
4.42	3.65
6.02	4.74
3.75	2.46
4.39	3.45
5	3.84
4.97	4.06
6.01	4.4
7.05	5.81
4.65	3.84
7.58	5.58
4.38	2.92
5.59	4.27
5.59	4.15
5.67	4.65
7.85	6.13
4.88	4.06
6.06	3.71
6.23	4.78
6.48	4.71
6.13	4.92
8.63	6.64
6.51	4.23
6.98	5.2
7.06	5.12
6.68	5.33
8.79	5.03
7.66	5.69
8.02	5.55
7.35	5.73
5.01	3.32
7.73	5.74
8.96	5.96
7.92	6.15
9.22	6.14
8.63	6.36
9.63	6.32
10.05	7.09
2.01	1.42

External position	Internal position
2.2	1.97
6.27	3.99
1.4	1.2
1.34	1
5.33	3.63
3.59	2.99
3.33	2.72
2.84	2.25
3.89	2.94
3.99	3.33
1.13	0.85
6.73	4.7
4.18	3.49
8.07	6.56
2.03	1.26
1.84	1.41
2.72	2.02
3.57	2.41
3.01	2.48
2.86	2.39
1.8	1.5
1.57	1.23
4.51	3.46
3.94	3.2
5.27	4.64
2.65	2.27
4.33	3.26
5.52	4.41
9.2	7.15
3.67	2.76
2.16	1.62
4.68	3.7
2.77	1.84
5.03	4.14
3.47	2.94
3.87	3.26
2.12	1.72
5.38	3.91
4.43	3.65
9.18	6.76
3.09	2.56
2.64	2.06
6.36	5.06
9.83	7.68

External position	Internal position
9.18	6.27
2.58	1.87
7.11	5.41
4.72	3.02
8.29	6.33
6.33	5.4
4.48	3.66
2.74	2.33
7.31	4.86
4.94	4.16
3.28	2.7
7.71	5.06
7.68	5.77
10.23	7.76
2.8	2.15
9.26	6.8
8.7	6.46
6.89	5.62
6.79	4.9
3.53	3.03
7.21	5.62
10.58	8.04
3.35	2.6
11.94	8.33
7.02	5.04
5.93	4.63
7.42	5.82
4.16	3.33
7.09	5.04
8.1	6.4
8.38	6.64
4.75	3.98
5.8	4.65
5.92	4.72

Table 12. External and internal positions of striae of Retzius in the Gray Friars sample

External position	Internal position
2.01	1.42
2.2	1.97
6.27	3.99
1.4	1.2
1.34	1
5.33	3.63
3.59	2.99
3.33	2.72
2.84	2.25
3.89	2.94
3.99	3.33
1.13	0.85
6.73	4.7
4.18	3.49
8.07	6.56
2.03	1.26
1.84	1.41
2.72	2.02
3.57	2.41
3.01	2.48
2.86	2.39
1.8	1.5
1.57	1.23
4.51	3.46
3.94	3.2
5.27	4.64
2.65	2.27
4.33	3.26
5.52	4.41
9.2	7.15
3.67	2.76
2.16	1.62
4.68	3.7
2.77	1.84
5.03	4.14
3.47	2.94
3.87	3.26
2.12	1.72
5.38	3.91
4.43	3.65
9.18	6.76
3.09	2.56

External position	Internal position
2.64	2.06
6.36	5.06
9.83	7.68
9.18	6.27
2.58	1.87
7.11	5.41
4.72	3.02
8.29	6.33
6.33	5.4
4.48	3.66
2.74	2.33
7.31	4.86
4.94	4.16
3.28	2.7
7.71	5.06
7.68	5.77
10.23	7.76
2.8	2.15
9.26	6.8
8.7	6.46
6.89	5.62
6.79	4.9
3.53	3.03
7.21	5.62
10.58	8.04
3.35	2.6
11.94	8.33
7.02	5.04
5.93	4.63
7.42	5.82
4.16	3.33
7.09	5.04
8.1	6.4
8.38	6.64
4.75	3.98
5.8	4.65
5.92	4.72

Table 13. Distance functions for each sample and combined samples

Sample	Quadratic regression	σ	r
Black Friars	y = -.353 + .970x - .027x2	0.347	0.970
Gray Friars	y = -.038 + .831x - .009x2	0.292	0.988
Combined samples	y = -.052 + .832x - .011x2	0.343	0.980

Table 14. External and converted internal locations (mm) of enamel surface defects (SD) in the Black Friars dental sample using the regression formula developed from Black Friars striae of Retzius data

Individual	# *SD*	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int
SBT78-010	2	3.80	2.94	4.60	3.54										
SBT78-011	0														
SBT78-017	0														
SBT78-018	3	0.70	0.31	4.00	3.10	5.45	4.13								
SBT78-022	3	2.25	1.69	3.36	2.60	4.74	3.64								
SBT78-025	0														
SBT78-027	1	5.78	4.35												
SBT78-028	2	4.55	3.50	5.77	4.34										
SBT78-029	3	1.10	0.68	2.91	2.24	4.98	3.81								
SBT78-031	3	4.00	3.10	4.30	3.32	5.40	4.10								
SBT78-037	3	2.30	1.74	4.43	3.41	5.30	4.03								
SBT78-044	2	3.10	2.39	4.82	3.70										
SBT78-047	4	1.62	1.15	2.14	1.60	4.25	3.28	5.00	3.82						
SBT78-049	2	5.30	4.03	6.85	5.02										
SBT78-051	3	1.75	1.26	3.90	3.02	6.00	4.50								
SBT78-055	3	2.45	1.86	3.00	2.31	5.75	4.33								
SBT78-056	1	3.80	2.94												
SBT78-083	0														
SBT78-085	2	2.78	2.13	8.25	5.81										
SBT78-200	2	3.82	2.96	6.75	4.96										
SBT78-201	1	4.75	3.65												
SBT78-202	2	4.71	3.62	7.02	5.13										
SBT79-003	1	1.63	1.16												
SBT79-004	0														
SBT79-005	6	2.31	1.74	2.90	2.23	4.90	3.75	6.05	4.53	8.00	5.68	9.33	6.35		
SBT79-009	6	2.48	1.89	4.55	3.50	5.35	4.06	5.91	4.44	6.65	4.90	7.30	5.29		
SBT79-011	3	4.35	3.36	5.40	4.10	6.06	4.53								
SBT79-014	3	4.07	3.15	4.86	3.72	6.10	4.56								
SBT79-017	2	5.55	4.20	6.25	4.65										
SBT79-018	2	5.17	3.94	8.95	6.17										
SBT79-020	3	3.86	2.99	6.35	4.72	6.67	4.92								
SBT79-021	4	2.30	1.74	2.90	2.23	3.65	2.83	4.80	3.68						
SBT79-022	1	5.40	4.10												
SBT79-027	2	3.32	2.57	5.13	3.91										
SBT79-029	3	1.29	0.85	4.05	3.13	6.37	4.73								
SBT79-030	3	2.95	2.27	5.45	4.13	5.90	4.43								
SBT79-031	1	4.10	3.17												

Individual	# *SD*	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int
SBT81-001	2	6.40	4.75	7.48	5.39										
SBT81-005	2	4.05	3.13	7.27	5.27										
SBT81-015	2	4.50	3.47	6.17	4.60										
SBT81-019	1	4.17	3.22												
SBT81-023	0														
SBT81-034	1	4.17	3.22												
SBT81-035	1	7.13	5.19												
SBT81-047	1	1.75	1.26												
SBT81-050b	2	4.05	3.13	6.03	4.51										
SBT81-053	4	3.17	2.45	4.71	3.62	5.75	4.33	6.91	5.06						
SBT81-054	2	4.25	3.28	5.70	4.30										
SBT81-056	5	2.40	1.82	5.67	4.28	6.84	5.02	7.09	5.17	9.07	6.22				
SBT81-060	2	3.59	2.78	4.85	3.72										
SBT81-062	2	2.11	1.57	4.60	3.54										
SBT81-069	4	2.00	1.48	3.15	2.43	4.60	3.54	6.00	4.50						
SBT81-075	1	4.89	3.74												
SBT81-078	1	5.01	3.83												
SBT81-085	1	7.28	5.28												
SBT81-089	3	4.30	3.32	6.43	4.77	8.50	5.94								
SBT81-094	2	4.59	3.53	6.30	4.69										
SBT81-099	3	2.65	2.03	3.21	2.48	4.30	3.32								
SBT81-100	3	2.00	1.48	4.88	3.74	8.39	5.88								
SBT81-104	3	3.61	2.80	5.23	3.98	6.00	4.50								
SBT81-108	1	6.51	4.82												
SBT81-117	0														
SBT81-119	2	3.20	2.47	4.31	3.33										
SBT81-132	3	3.62	2.80	5.15	3.93	6.45	4.78								
SBT81-134	2	2.95	2.27	3.72	2.88										
SBT81-153	3	2.50	1.90	3.59	2.78	5.05	3.86								
SBT81-160	1	4.30	3.32												
SBT81-168	2	2.81	2.16	4.05	3.13										
SBT81-183	0														
SBT81-188	1	3.35	2.59												
SBT81-193	2	2.90	2.23	5.15	3.93										
SBT81-195	1	5.28	4.02												
SBT81-203	2	2.92	2.25	3.90	3.02										
SBT81-205	2	4.10	3.17	5.20	3.96										
SBT81-207	1	2.50	1.90												
SBT81-212	3	2.10	1.56	4.20	3.24	5.20	3.96								
SBT81-214	4	1.60	1.13	3.50	2.71	5.45	4.13	6.50	4.81						

Individual	# *SD*	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int
SBT81-215	2	2.91	2.24	5.71	4.31										
SBT81-218	5	2.45	1.86	3.30	2.55	4.55	3.50	6.45	4.78	7.85	5.60				
SBT81-218x	2	3.20	2.47	4.85	3.72										
SBT81-219	2	3.85	2.98	5.04	3.85										
SBT81-222	4	1.85	1.35	4.50	3.47	6.35	4.72	7.95	5.65						
SBT81-223	5	0.71	0.32	1.32	0.88	3.30	2.55	5.50	4.17	6.75	4.96				
SBT81-231	1	4.20	3.24												
SBT81-234	3	2.20	1.65	3.10	2.39	4.15	3.21								
SBT81-235	2	3.95	3.06	7.65	5.49										
SBT81-236	3	4.25	3.28	5.91	4.44	8.45	5.92								
SBT81-241	1	2.44	1.85												
SBT81-244	4	2.39	1.81	4.55	3.50	6.14	4.58	6.83	5.01						
SBT81-248	5	2.95	2.27	4.25	3.28	4.90	3.75	6.20	4.62	6.74	4.96				
SBT81-251	0														
SBT81-253	1	4.99	3.81												
SBT81-256	3	2.60	1.99	3.69	2.86	5.22	3.97								
SBT81-266	3	3.82	2.96	5.72	4.31	6.08	4.55								
SBT81-271	4	1.10	0.68	1.70	1.22	5.70	4.30	6.90	5.05						
SBT81-281	1	3.87	3.00												
SBT81-282	0														
SBT81-283	2	4.50	3.47	5.60	4.23										
SBT81-289	3	2.22	1.67	3.21	2.48	3.51	2.72								
SBT81-291	2	3.05	2.35	6.60	4.87										
SBT81-292	3	2.00	1.48	2.92	2.25	4.20	3.24								
SBT81-294	2	2.83	2.18	5.32	4.04										
SBT81-295	4	3.20	2.47	4.18	3.23	5.52	4.18	6.70	4.93						
SBT81-296	4	1.20	0.77	2.50	1.90	3.70	2.87	5.05	3.86						
SBT81-297	4	2.10	1.56	3.35	2.59	4.35	3.36	6.00	4.50						
SBT81-299	2	2.51	1.91	3.15	2.43										
SBT81-307	3	1.92	1.41	2.83	2.18	4.20	3.24								
SBT81-308	6	0.90	0.50	2.58	1.97	3.20	2.47	3.60	2.79	4.10	3.17	5.12	3.91		
SBT81-310	3	2.45	1.86	2.92	2.25	4.70	3.61								
SBT81-312	1	2.45	1.86												
SBT81-313	3	2.15	1.61	4.01	3.10	5.95	4.46								
SBT81-314x	3	3.20	2.47	4.60	3.54	12.50	7.55								
SBT81-316	2	3.98	3.08	5.85	4.40										
SBT81-320	1	4.41	3.40												
SBT81-326	0														
SBT81-328	3	3.60	2.79	5.80	4.36	7.85	5.60								

Individual	# *SD*	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int
SBT81-331	2	2.20	1.65	5.75	4.33										
SBT81-334	4	4.10	3.17	5.00	3.82	7.70	5.52	10.00	6.65						
SBT81-337	4	2.00	1.48	3.30	2.55	4.50	3.47	5.00	3.82						
SBT81-339	1	6.00	4.50												
SBT81-340	2	1.00	0.59	3.65	2.83										
SBT81-344	4	2.68	2.05	4.20	3.24	5.71	4.31	7.10	5.17						
SBT81-345	1	3.20	2.47												
SBT81-346	1	5.90	4.43												
SBT81-349	2	3.31	2.56	4.50	3.47										
SBT81-351	0														
SBT81-353	1	3.00	2.31												
SBT81-354	1	3.75	2.90												
SBT81-355	2	5.75	4.33	6.45	4.78										
SBT81-357	2	2.10	1.56	3.30	2.55										
SBT81-358	2	3.30	2.55	4.35	3.36										
SBT81-359	2	2.80	2.15	4.95	3.79										
SBT81-362	2	4.70	3.61	6.25	4.65										
SBT81-367	3	4.60	3.54	6.30	4.69	8.10	5.73								
SBT81-371	3	2.40	1.82	3.05	2.35	6.53	4.83								
SBT81-372	6	3.70	2.87	4.70	3.61	5.50	4.17	6.35	4.72	7.15	5.20	7.95	5.65		
SBT81-374	2	2.47	1.88	4.60	3.54										
SBT81-378	5	2.20	1.65	3.60	2.79	5.60	4.23	6.70	4.93	7.62	5.47				
SBT81-381	1	2.50	1.90												
SBT81-382	2	4.20	3.24	5.50	4.17										
SBT81-383	4	3.45	2.67	5.10	3.89	6.63	4.89	7.42	5.36						
SBT81-386	4	2.65	2.03	3.81	2.95	5.30	4.03	6.20	4.62						
SBT81-395	3	2.25	1.69	3.95	3.06	5.00	3.82								
SBT81-400	1	4.40	3.39												
SBT81-403	7	2.92	2.25	4.10	3.17	4.36	3.36	5.35	4.06	6.60	4.87	8.25	5.81	8.90	6.14
SBT81-409	5	2.75	2.11	3.60	2.79	4.40	3.39	5.22	3.97	5.78	4.35				
SBT81-434	2	2.15	1.61	3.10	2.39										
SBT81-437	1	4.10	3.17												
SBT81-439	1	2.50	1.90												
SBT81-442	2	3.60	2.79	5.80	4.36										
SBT81-446	5	1.50	1.04	2.35	1.78	3.15	2.43	4.50	3.47	5.90	4.43				
SBT81-457	3	2.61	1.99	5.75	4.33	7.69	5.51								
SBT81-459	6	4.02	3.11	5.00	3.82	6.00	4.50	6.95	5.08	8.10	5.73	9.10	6.24		
SBT81-460x	1	4.45	3.43												
SBT81-462	3	3.60	2.79	3.80	2.94	4.48	3.45								
SBT81-464	2	4.45	3.43	5.30	4.03										

Individual	# *SD*	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int
SBT81-465	2	1.40	0.95	4.68	3.60										
SBT81-468	2	4.00	3.10	5.72	4.31										
SBT81-470	7	2.50	1.90	3.25	2.51	4.49	3.46	6.10	4.56	6.80	4.99	7.65	5.49	9.90	6.60
SBT81-471	1	6.60	4.87												
SBT81-472	1	4.45	3.43												
SBT81-478	2	3.10	2.39	7.25	5.26										
SBT81-479	1	5.00	3.82												
SBT81-483	3	2.60	1.99	5.03	3.84	6.10	4.56								
SBT81-484	0														
SBT81-491	3	3.80	2.94	5.20	3.96	6.43	4.77								
SBT81-496	1	5.50	4.17												
SBT81-499	4	1.72	1.24	7.00	5.11	7.60	5.46	9.30	6.33						
SBT81-502	1	3.30	2.55												
SBT81-503	2	5.02	3.84	6.35	4.72										
SBT81-505	4	2.50	1.90	3.10	2.39	5.02	3.84	6.70	4.93						
SBT81-506	2	4.21	3.25	5.35	4.06										
SBT81-511	1	4.20	3.24												
SBT81-513	1	1.30	0.86												
SBT81-515	1	2.91	2.24												
SBT81-516	4	2.90	2.23	4.50	3.47	5.60	4.23	7.10	5.17						
SBT81-519	3	2.30	1.74	4.31	3.33	5.31	4.04								
SBT81-522	1	4.21	3.25												
SBT81-524	4	2.12	1.58	3.28	2.54	4.31	3.33	6.22	4.64						
SBT81-525	0														
SBT81-530	3	1.65	1.17	3.18	2.46	5.50	4.17								
SBT81-531	3	3.59	2.78	5.50	4.17	7.10	5.17								
SBT81-534	2	3.70	2.87	5.84	4.39										
SBT81-538	7	2.50	1.90	3.45	2.67	4.50	3.47	5.01	3.83	5.35	4.06	6.84	5.02	7.90	5.62
SBT81-546	2	5.40	4.10	8.20	5.79										
SBT81-550	2	4.35	3.36	6.81	5.00										
SBT81-552	5	0.95	0.54	2.51	1.91	3.25	2.51	5.02	3.84	6.63	4.89				
SBT81-553	4	1.25	0.82	2.00	1.48	6.35	4.72	8.25	5.81						
SBT81-559	6	3.10	2.39	4.18	3.23	4.35	3.36	4.78	3.67	5.80	4.36	7.00	5.11		
SBT81-561	2	3.20	2.47	5.20	3.96										
SBT81-562	4	2.80	2.15	3.30	2.55	4.40	3.39	7.70	5.52						
SBT81-563	0														
SBT81-564	5	0.40	0.03	1.40	0.95	2.90	2.23	4.30	3.32	5.60	4.23				
SBT81-566	1	5.00	3.82												
SBT81-567	5	1.40	0.95	2.80	2.15	3.40	2.63	4.50	3.47	5.25	4.00				
SBT81-568	1	4.71	3.62												
SBT81-570	5	0.70	0.31	1.95	1.44	4.23	3.27	5.10	3.89	6.50	4.81				

Individual	# *SD*	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int
SBT81-577	2	1.55	1.09	3.10	2.39										
SBT81-578	5	2.20	1.65	4.40	3.39	5.70	4.30	7.20	5.23	8.43	5.91				
SBT81-579	4	4.92	3.77	6.40	4.75	7.00	5.11	8.20	5.79						
SBT81-580	3	2.00	1.48	2.95	2.27	3.70	2.87								
SBT81-581	4	2.90	2.23	4.10	3.17	6.20	4.62	8.31	5.84						
SBT81-582	4	2.80	2.15	4.05	3.13	5.70	4.30	7.10	5.17						
SBT81-584	1	4.20	3.24												
SBT81-587	5	2.60	1.99	4.28	3.30	5.20	3.96	5.62	4.25	6.41	4.76				
SBT81-592	1	3.30	2.55												
SBT81-593	3	3.45	2.67	4.95	3.79	9.55	6.45								
SBT81-595	5	2.10	1.56	3.10	2.39	4.60	3.54	5.30	4.03	5.85	4.40				
SBT81-597	4	4.70	3.61	2.90	2.23	6.10	4.56	8.10	5.73						
SBT81-598	1	4.00	3.10												
SBT81-600	1	4.90	3.75												
SBT81-602	3	3.05	2.35	4.80	3.68	5.25	4.00								
SBT81-605	3	2.58	1.97	3.90	3.02	6.10	4.56								
SBT81-607	3	4.20	3.24	6.60	4.87	7.85	5.60								
SBT81-616	2	2.05	1.52	3.22	2.49										
SBT81-617	5	2.60	1.99	2.90	2.23	5.20	3.96	5.82	4.38	6.21	4.63				
SBT81-625	2	5.70	4.30	8.25	5.81										
SBT81-626	3	2.15	1.61	4.20	3.24	5.95	4.46								
SBT81-630	3	2.45	1.86	4.50	3.47	6.35	4.72								
SBT81-633	5	2.85	2.19	4.10	3.17	5.45	4.13	6.20	4.62	6.70	4.93				
SBT81-637	1	4.40	3.39												
SBT81-639	3	3.05	2.35	6.30	4.69	7.43	5.36								
SBT81-641	2	3.12	2.41	4.30	3.32										
SBT81-643	3	1.67	1.19	2.60	1.99	3.80	2.94								
SBT81-644	1	5.03	1.90												
SBT81-651	3	2.50	1.90	3.59	2.78	6.10	4.56								
SBT81-657	0														
SBT81-659	3	2.58	1.97	3.79	2.94	5.05	3.86								
SBT81-661	4	1.30	0.86	3.00	2.31	4.80	3.68	5.50	4.17						
SBT81-665	5	1.45	1.00	2.30	1.74	3.35	2.59	4.81	3.69	6.82	5.01				
SBT81-670	2	3.25	2.51	4.33	3.34										
SBT81-671	3	4.40	3.39	4.95	3.79	7.90	5.62								
SBT81-674	2	6.05	4.53	7.81	5.58										
SBT81-679	6	2.21	1.66	2.92	2.25	3.70	2.87	4.30	3.32	5.30	4.03	6.11	4.57		

Table 15. External and converted internal locations (mm) of enamel hypoplasias in the Gray Friars sample

Individual	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int	*SD*8 Ext	*SD*8 Int	*SD*9 Ext	*SD*9 Int
GBP82-003	1.9	1.49	2.7	2.11	3.9	3.03	5.45	4.16	6.3	4.75								
GBP82-006	1.1	0.85	2.5	1.96	3.5	2.73	4	3.10	5.2	3.98	6.25	4.72	6.95	5.20				
GBP82-008	4.25	3.29	7.5	5.57	8.42	6.17												
GBP82-009	1.8	1.41	2.8	2.19	4	3.10	4.98	3.82	5.5	4.19	6.93	5.19	8.8	6.42				
GBP82-014	2.6	2.04	5.2	3.98	6.6	4.96												
GBP82-019	5.1	3.91																
GBP82-020	2.54	1.99	4.6	3.54	5.32	4.06	6	4.54										
GBP82-026	3.2	2.50	3.7	2.88	4.45	3.43	5.09	3.90	5.55	4.23	5.89	4.47	7.27	5.42				
GBP82-028	1.9	1.49	5.8	4.40	6.91	5.17												
GP90-002	2.83	2.21	5.05	3.87														
GP90-011	2.85	2.23	4.2	3.25	6.35	4.79												
GP90-014	3.07	2.40	4.55	3.51	5.52	4.21	0.62	0.46	1.15	0.89	0.62	0.46						
GP90-017	3.21	2.51																
GP90-021	1.22	0.95	2.85	2.23	4	3.10												
GP90-022	2	1.57	3.63	2.82	6.5	4.89												
GP90-023	3.2	2.50	4.35	3.36														
GP90-024	1.8	1.41	3.71	2.88	6.18	4.67												
GP90-028	1.7	1.33	2.2	1.73	2.92	2.28												
GP90-029	1.52	1.19	2.31	1.81	3.5	2.73	5	3.83										
GP90-030	3.2	2.50																
GP90-034	1	0.77	2.2	1.73	3.1	2.42	7.21	5.37										
GP90-040	1.1	0.85	4.7	3.62														
GP90-041	2.1	1.65	3.2	2.50														
GP90-044	2.74	2.15	3.5	2.73	5.2	3.98	6.65	4.99										
GP90-045	2.71	2.12	3.3	2.57														
GP90-046	1.3	1.01	2.55	2.00	4	3.10												
GP90-048	0.88	0.67	1.85	1.45	3.2	2.50	4.5	3.47										
GP90-051	1.5	1.17	2.18	1.71	4.4	3.40	5.68	4.32	7.6	5.64								
GP90-053	2.9	2.27	3.98	3.09	5.1	3.91	6.1	4.61	7.4	5.50								
GP90-055	1.68	1.31	2.56	2.01	3.44	2.68	5.41	4.13										
GP90-058	2.2	1.73	3.95	3.06	4.8	3.69												
GP90-059	2.41	1.89	3	2.35	3.89	3.02	5.2	3.98	7.08	5.29								
GP90-065	2.41	1.89	3.88	3.01														
GP90-066	1.2	0.93	2.22	1.74	3.7	2.88	5.05	3.87										
GP90-070	1.4	1.09	3.2	2.50														
GP90-071	1.7	1.33	3.15	2.46	5.05	3.87												
GP90-072	2.9	2.27	4.3	3.32														

Individual	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int	*SD*8 Ext	*SD*8 Int	*SD*9 Ext	*SD*9 Int
GP90-073	1.9	1.49	3.45	2.69														
GP90-079	3.9	3.03																
GP90-088	2.75	2.15	3.4	2.65	4.55	3.51	6.23	4.70										
GP90-090	2.3	1.80	5.4	4.12	7.45	5.54												
GP90-096	2.2	1.73	6.1	4.61														
GP90-099	2.55	2.00																
GP90-100	1.75	1.37	2.4	1.88	3.75	2.91	4.63	3.56										
GP90-102	1.45	1.13																
GP90-108	2	1.57	3.1	2.42	4.7	3.62	6.25	4.72										
GP90-110	1.9	1.49	2.65	2.08	3.31	2.58	3.96	3.07	4.4	3.40	4.95	3.80	5.91	4.48	7.4	5.50		
GP90-111	2.4	1.88	3.95	3.06														
GP90-116	1.2	0.93	2.35	1.84	2.91	2.28												
GP90-124	1.1	0.85	1.8	1.41	2.13	1.67	3.79	2.94	4.21	3.26	4.86	3.73						
GP90-128	1.9	1.49	2.6	2.04	3.61	2.81	5.4	4.12										
GP90-129	2.28	1.79																
GP90-130	2.1	1.65	3.5	2.73	4.6	3.54	5.1	3.91	6.1	4.61	6.58	4.95						
GP90-131	2.2	1.73	3.1	2.42	5.1	3.91												
GP90-135	0.6	0.44	1.9	1.49	3.05	2.38	3.7	2.88	4.05	3.14	4.5	3.47	5.7	4.33	7	5.23		
GP90-138	1.35	1.05	3	2.35	4.6	3.54												
GP90-141	2.8	2.19	6.38	4.81														
GP90-143	0.91	0.70	2.42	1.90	4.25	3.29												
GP90-150	2.9	2.27	4.1	3.17	5.13	3.93	6.42	4.84	7.1	5.30								
GP90-153	2.2	1.73	3.89	3.02	5	3.83	5.7	4.33	6.9	5.17								
GP90-156	2.48	1.94	3.35	2.61	6.8	5.10	8	5.90										
GP90-157	1.1	0.85	2	1.57	3.75	2.91												
GP90-160	1.03	0.79	2	1.57	3	2.35												
GP90-170	2.03	1.59	3.3	2.57	5	3.83												
GP90-173	2.35	1.84	3.9	3.03	5	3.83	7	5.23										
GP90-174	1.4	1.09	2.9	2.27														
GP90-175	0.52	0.38	1.11	0.86	1.8	1.41	2.9	2.27	3.7	2.88	5.6	4.26	6.7	5.03				
GP90-178	2.1	1.65	3.33	2.60	4.9	3.76												
GP90-182	3	2.35	3.8	2.95	5.4	4.12	6.92	5.18										
GP90-183	2	1.57	2.6	2.04	3.32	2.59	4	3.10										
GP90-185	1.92	1.50	3.2	2.50														
GP90-191	1.4	1.09	2.3	1.80	4.2	3.25	6.29	4.75										
GP90-193	2.05	1.61	2.7	2.11	3.95	3.06	5	3.83	5.35	4.08	6.05	4.58	7	5.23				
GP90-194	2.8	2.19	3.9	3.03	5.4	4.12	7	5.23										
GP90-199	1.43	1.12	3.3	2.57	4.2	3.25	5.3	4.05										
GP90-202	0.8	0.61	2.1	1.65	3.25	2.54												
GP90-204	0.7	0.53	1.45	1.13	2.02	1.58	3.91	3.03	6.4	4.82								

Individual	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int	*SD*8 Ext	*SD*8 Int	*SD*9 Ext	*SD*9 Int
GP90-205	4	3.10																
GP90-211	2.9	2.27	3.8	2.95														
GP90-213	1.9	1.49	3.05	2.38														
GP90-216	1.33	1.04	1.79	1.40	2.41	1.89	3.25	2.54										
GP90-217	2.9	2.27	4.38	3.38														
GP90-220	3.1	2.42	5.5	4.19	6.3	4.75	7.8	5.77										
GP90-221	1.27	0.99	2.65	2.08	3.71	2.88												
GP90-224	2.2	1.73	3.57	2.78	4.05	3.14												
GP90-225	2	1.57	3.55	2.76	5.5	4.19												
GP90-227	4.94	3.79	6.2	4.68	6.89	5.16												
GP90-228	1.55	1.21	2.41	1.89	3.2	2.50												
GP90-230	1.6	1.25	2.05	1.61	3.13	2.44												
GP90-232x	3	2.35																
GP90-235	1.31	1.02	2.42	1.90	3.13	2.44	4.35	3.36	5.42	4.13								
GP90-236	0.8	0.61	1.7	1.33	5.52	4.21	7.1	5.30										
GP90-238	1.9	1.49	2.32	1.82														
GP90-241	3.1	2.42	3.75	2.91														
GP90-248	1.81	1.42	2.94	2.30	3.9	3.03	5.1	3.91	6.5	4.89								
GP90-254	1.7	1.33	2.55	2.00	2.93	2.29	3.5	2.73	4.34	3.35	5.5	4.19	6.18	4.67	8	5.90		
GP90-255	0.7	0.53	1.65	1.29	2.84	2.22	3.92	3.04										
GP90-262	3.7	2.88																
GP90-263	1.4	1.09	1.9	1.49	2.57	2.01	3.05	2.38	3.35	2.61	4.15	3.21	5.42	4.13	6.72	5.04		
GP90-267	2.03	1.59	3.3	2.57	4.4	3.40	5.4	4.12										
GP90-272	2.6	2.04	3.4	2.65														
GP90-273	2.8	2.19	3.65	2.84	4.6	3.54												
GP90-275	2.14	1.68	3.1	2.42	4.55	3.51												
GP90-279	0.4	0.28	0.9	0.69	1.52	1.19	2.5	1.96	4.95	3.80								
GP90-280	3	2.35	3.5	2.73	4	3.10	4.5	3.47										
GP90-280x	1.9	1.49	2.7	2.11														
GP90-281	3.45	2.69	4.5	3.47	5.5	4.19												
GP90-282	2.19	1.72	4.3	3.32	6	4.54												
GP90-283	2.05	1.61	5.52	4.21														
GP90-294	0.3	0.20	1.37	1.07	3.1	2.42												
GP90-296	1.1	0.85	2.1	1.65	2.9	2.27	5.93	4.49										
GP90-297	1.3	1.01	2.52	1.97	3.96	3.07												
GP90-300	0.55	0.40	1.6	1.25	2.4	1.88	3.4	2.65										
GP90-301	2	1.57	2.62	2.05	3.1	2.42	4.9	3.76										
GP90-302	1.8	1.41	3.4	2.65	4.55	3.51	5.61	4.27										
GP90-304	1.46	1.14	2.45	1.92	4.5	3.47												
GP90-309	1.45	1.13	2.43	1.90	4	3.10												

Individual	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int	*SD*8 Ext	*SD*8 Int	*SD*9 Ext	*SD*9 Int
GP90-315	2.45	1.92	3.75	2.91														
GP90-317	2.41	1.89	4	3.10	5.55	4.23	6.4	4.82										
GP90-319	1.98	1.55	3.2	2.50	3.69	2.87	4.2	3.25	4.7	3.62	5.1	3.91						
GP90-320	1.1	0.85	2.8	2.19	5.35	4.08	6.4	4.82										
GP90-321	1.3	1.01	3.15	2.46	4.5	3.47												
GP90-322	0.7	0.53	1.7	1.33	3	2.35	5.2	3.98	6.81	5.10								
GP90-324	1.6	1.25	5.4	4.12														
GP90-326	2.49	1.95	3.34	2.60	4.15	3.21	6.28	4.74										
GP90-327	2.3	1.80	3	2.35	3.41	2.66	3.91	3.03	4.71	3.62	6.7	5.03						
GP90-328	1.8	1.41	5.31	4.06														
GP90-329	1.82	1.43	2.67	2.09	3.6	2.80	5	3.83	7.2	5.37	7.6	5.64						
GP90-330	2.5	1.96	5.3	4.05														
GP90-331	2.9	2.27																
GP90-333	2.73	2.14	4.21	3.26	5.07	3.88	6.4	4.82										
GP90-334	1.7	1.33	2.95	2.31	3.55	2.76	4	3.10	5.87	4.45	7.3	5.44						
GP90-337	3.05	2.38	3.6	2.80	5.42	4.13	7	5.23	7.78	5.76								
GP90-338	1.25	0.97	1.82	1.43	2.36	1.85	4.3	3.32	7.1	5.30								
GP90-339	2.6	2.04	4	3.10	4.52	3.48	7.45	5.54										
GP90-349	2.7	2.11	3.3	2.57														
GP90-350	2.88	2.25	4.29	3.31	5.62	4.28	7	5.23										
GP90-365	2.3	1.80	2.71	2.12	4.7	3.62	5.53	4.21	6.5	4.89	7.1	5.30						
GP90-367	1.3	1.01	2.62	2.05	4.71	3.62	6.3	4.75										
GP90-376	4.3	3.32	7.51	5.58	8.3	6.10												
GP90-377	1.11	0.86	1.9	1.49	3.21	2.51	4.61	3.55	5.51	4.20	6.9	5.17						
GP90-379	1.1	0.85	2.4	1.88	4.6	3.54	6.5	4.89										
GP90-380	2.2	1.73	3.69	2.87														
GP90-382	2.3	1.80	3.1	2.42	3.6	2.80	4.61	3.55										
GP90-384	2.35	1.84	3.6	2.80	4.65	3.58												
GP90-387	1.71	1.34	3.2	2.50	4.4	3.40												
GP90-391	1.11	0.86	2.8	2.19	5.1	3.91												
GP90-395	0.8	0.61	1.9	1.49	2.7	2.11	3.15	2.46	4.25	3.29	5.22	3.99	6.7	5.03	7.8	5.77		
GP90-396	3.1	2.42	3.7	2.88	4.15	3.21	4.6	3.54	5.65	4.30	6.35	4.79	7.45	5.54				
GP90-398	1.3	1.01	2.2	1.73	3.45	2.69	5.6	4.26										
GP90-399	2.2	1.73	2.85	2.23	3.2	2.50	4	3.10	5.2	3.98	7.3	5.44	7.71	5.71				
GP90-400	0.4	0.28	1.38	1.08	2.8	2.19	3.65	2.84	4.5	3.47	5.2	3.98	5.97	4.52	8.6	6.29	9.8	7.05
GP90-401	3.4	2.65	3.8	2.95	4.82	3.70	6.2	4.68	6.9	5.17								
GP90-403	1.2	0.93	2.4	1.88	3.63	2.82	3.87	3.00	5.4	4.12								
GP90-405	1.8	1.41	3.1	2.42														
GP90-406	2.1	1.65	6.3	4.75														
GP90-410	1.12	0.87	3.4	2.65	6.5	4.89	7.7	5.70										

Individual	*SD*1 Ext	*SD*1 Int	*SD*2 Ext	*SD*2 Int	*SD*3 Ext	*SD*3 Int	*SD*4 Ext	*SD*4 Int	*SD*5 Ext	*SD*5 Int	*SD*6 Ext	*SD*6 Int	*SD*7 Ext	*SD*7 Int	*SD*8 Ext	*SD*8 Int	*SD*9 Ext	*SD*9 Int
GP90-421	1.5	1.17	4.6	3.54	5.9	4.47												
GP90-423	0.6	0.44	2.9	2.27	3.6	2.80	4.93	3.78	5.7	4.33	6.1	4.61	6.31	4.76	7.5	5.57		
GP90-440	2.6	2.04	3.71	2.88	5.13	3.93												
GP90-446	1.3	1.01	2.95	2.31	4.51	3.48												
GP90-447	0.4	0.28	0.85	0.65	1.5	1.17	2.45	1.92	3.5	2.73	4.93	3.78						
GP90-449	1.95	1.53	3	2.35	3.5	2.73												
GP90-451	2.8	2.19	3.44	2.68	5.61	4.27												
GP90-459	2.5	1.96	3.6	2.80	4.5	3.47	5.05	3.87	5.8	4.40	7.5	5.57	8	5.90	8.7	6.35		
GP90-464	11.11	7.83	2.3	1.80	2.62	2.05	3	2.35	3.36	2.62	3.72	2.89	5.1	3.91	6	4.54	6.7	5.03
GP90-473	2.4	1.88	3.1	2.42	4.1	3.17												
GP90-479	2.85	2.23	4.4	3.40	6.4	4.82	7.8	5.77	8.5	6.23								
GP90-480	4.3	3.32																
GP90-487	1.5	1.17	2	1.57	2.95	2.31	4.9	3.76										
GP90-488	1.8	1.41	4	3.10	5.6	4.26												
GP90-489	4.9	3.76																
GP90-494	0.5	0.36	1	0.77	1.8	1.41	2.8	2.19	4.35	3.36	6.05	4.58	7	5.23				

Note. External locations were measured from the cervical edge of the defect to the tooth's cervix in millimeters. External locations were transformed into internal locations by using the quadratic function for the sample. SD stands for surface defect.

Table 16. Absolute locations of pathological striae (PS) in the Black Friars sample measured from the cervico-enamel junction

ID	Age[a]	Sex[b]	No. PS[c]	Loc 1 (mm)[d]	Loc 2 (mm)	Loc 3 (mm)	Loc 4 (mm)
SBT79-029	25	M	1	1.11			
SBT81-005	27.5	M	1	3.07			
SBT81-015	13	J	1	1.88			
SBT81-056	11.5	J	4	6.81	6.12	5.73	3.65
SBT81-137	13.5	J	1	3.05			
SBT81-295	10.5	J	1	6.13			
SBT81-334	10	J	2	7.67	5.96		
SBT81-524	38	F	1	5.64			
SBT81-559	11.5	J	3	5.82	4.10	2.19	
SBT81-617	39	M	1	6.05			

[a] Age in years as estimated by traditional osteological analyses.
[b] M signifies male, F signifies female, and J signifies unsexed juvenile (defined as age 15 or under).
[c] Number of pathological striae of Retzius.
[d] Location of pathological stria measured from intersection at the DEJ to the cervix in millimeters.

Table 17. Absolute locations of pathological striae (PS) in the Gray Friars sample measured from the cervico-enamel junction

ID	Age[a]	Sex[b]	No. PS[c]	Loc 1 (mm)[d]	Loc 2 (mm)
GP90-014	16	F	1	3.03	
GP90-029	7	J	2	7.47	6.23
GP90-248	adult	M	1	4.97	
GP90-262	21	M	1	5.23	
GP90-304	45	F	1	2.52	
GP90-365	10	J	1	3.3	

[a] Age in years as estimated by traditional osteological analyses.
[b] M signifies male, F signifies female, and J signifies unsexed juvenile (defined as age 15 or under).
[c] Number of pathological striae of Retzius.
[d] Location of pathological stria measured from intersection at the dej to the cervix in millimeters.

Appendix B

Figures

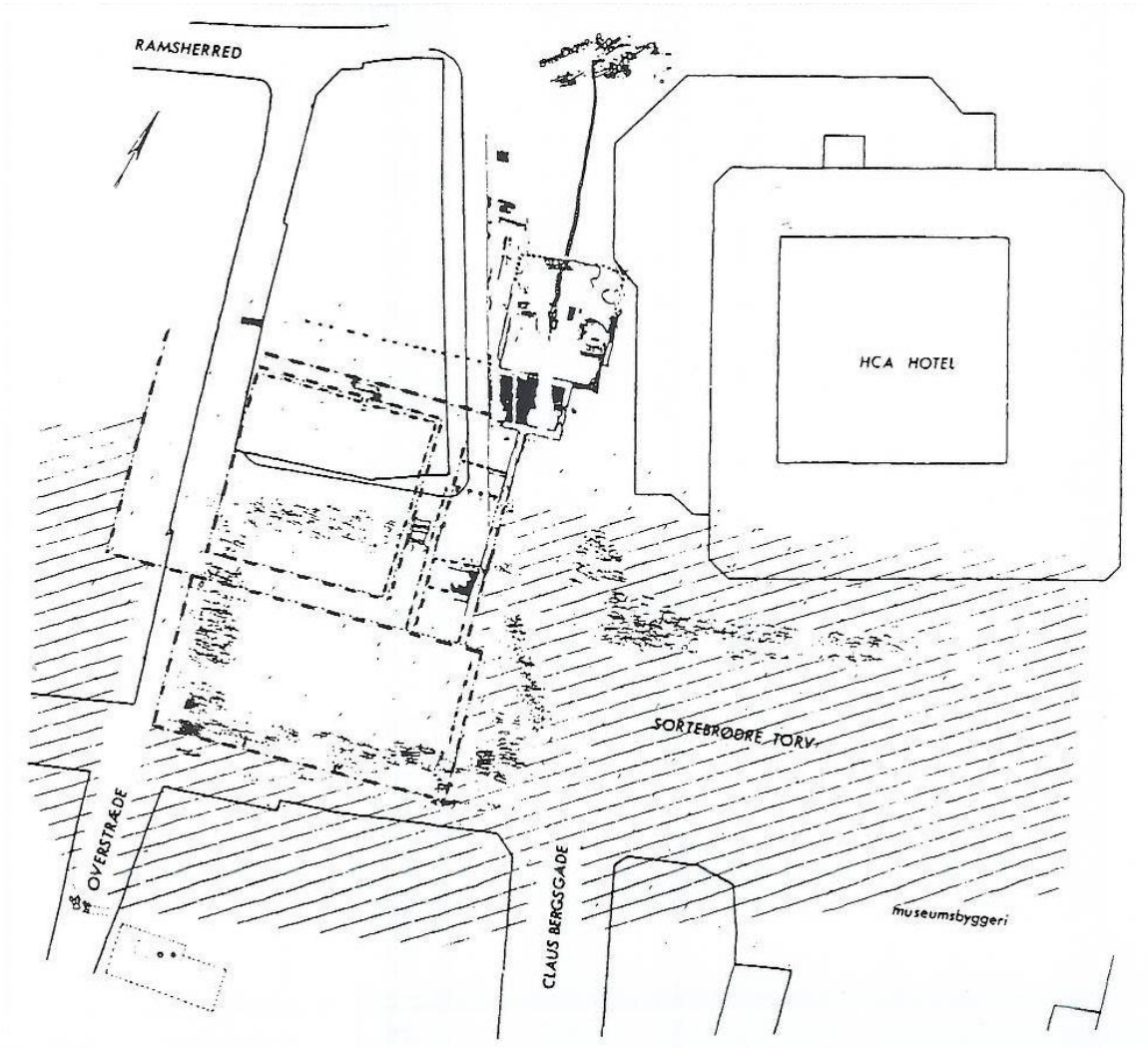

Figure 1. Drawing of the Black Friars monastery area excavations in Odense, DK from between 1972 and 1981. Stipled lines indicate monastery buildings, including the former church to the southwest. Skeletal individuals are denoted in a roughly square area around the former church, extending eastward. Printed after Becher, 1999.

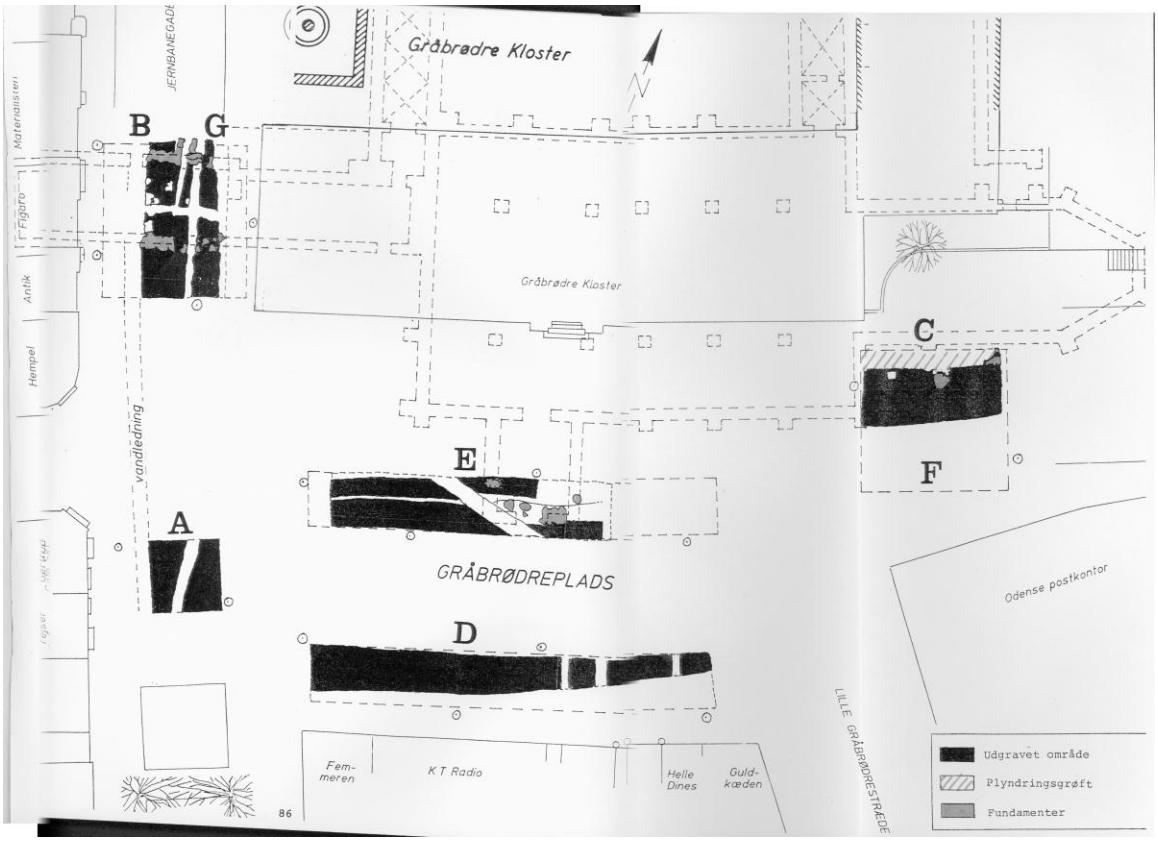

Figure 2. Drawing of the excavated areas at the Gray Friars monastery area (in black) in 1982 and 1990, including looted areas (stippled) and foundations (gray).

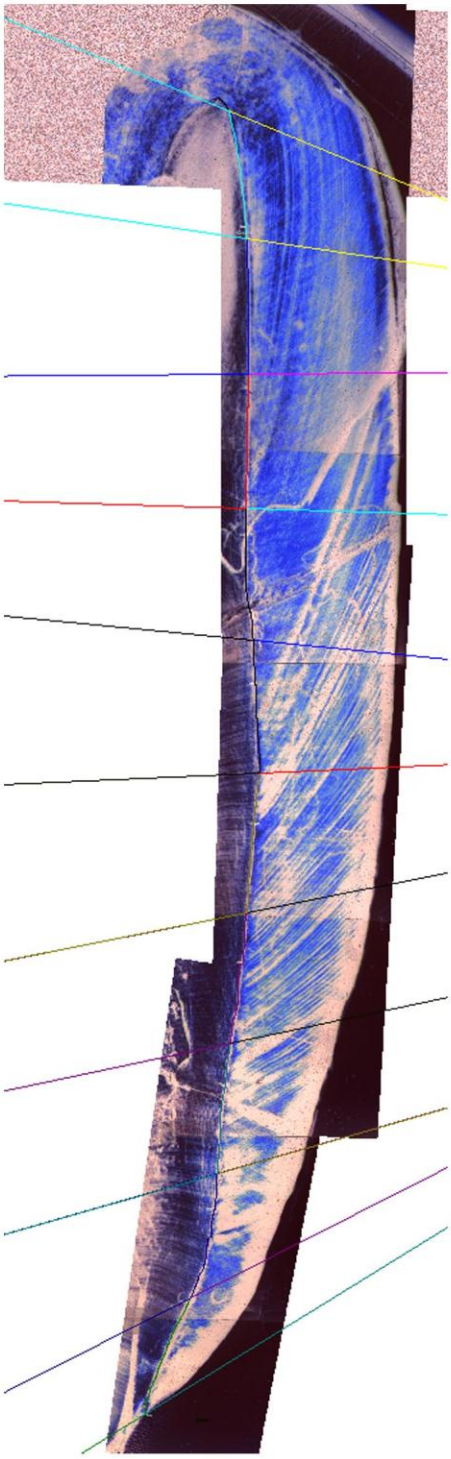

Figure 3. Photomontage of the labial mandibular canine enamel of SBT81-015, a 13 year old juvenile from the Black Friars monastery assemblage. The DEJ was divided in deciles, and intersecting SOR were counted in each decile to determine the total number of SOR.

Figure 4. Photomontage of labial mandibular canine enamel of SBT-81-137, a juvenile aged 13.5 years (+/- 1.5 years). Cuspal striae are clearly seen, as are darkened striae of Retzius. None were associated with extreme rod disorganization at higher magnification. Image taken at magnification of 40x.

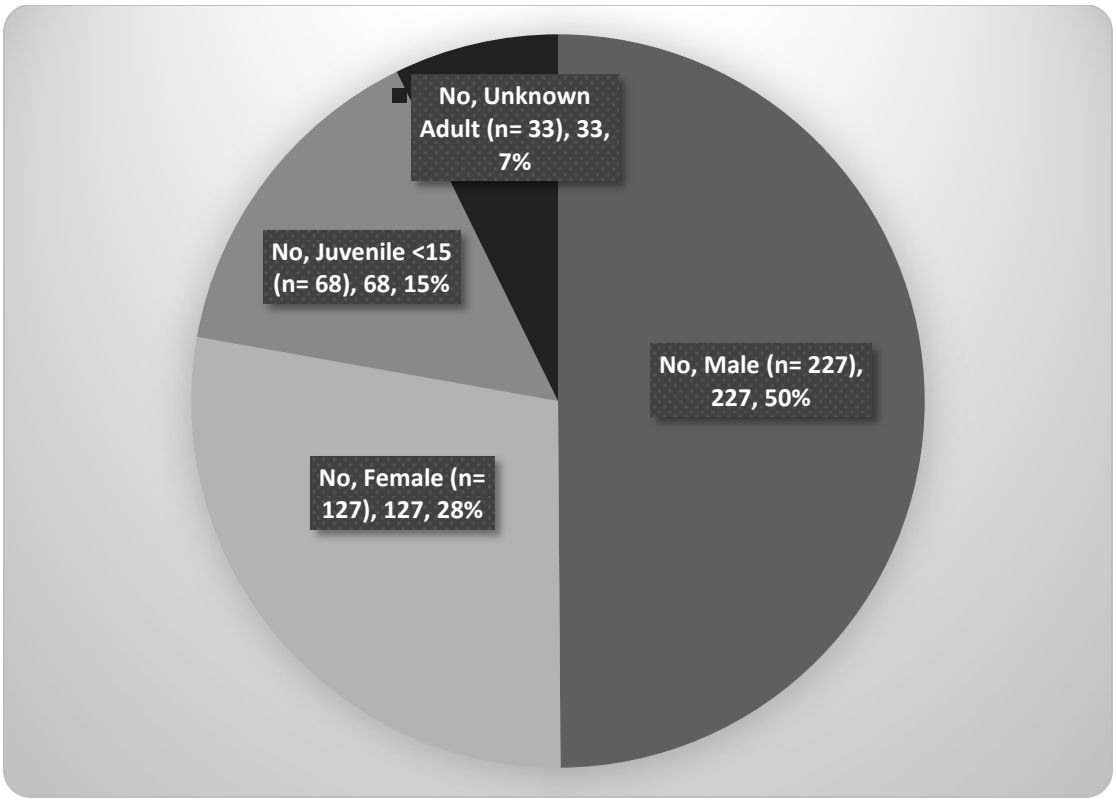

Figure 5. Sex and age of the skeletal individuals (n=455) making up the Gray Friars cemetery sample.

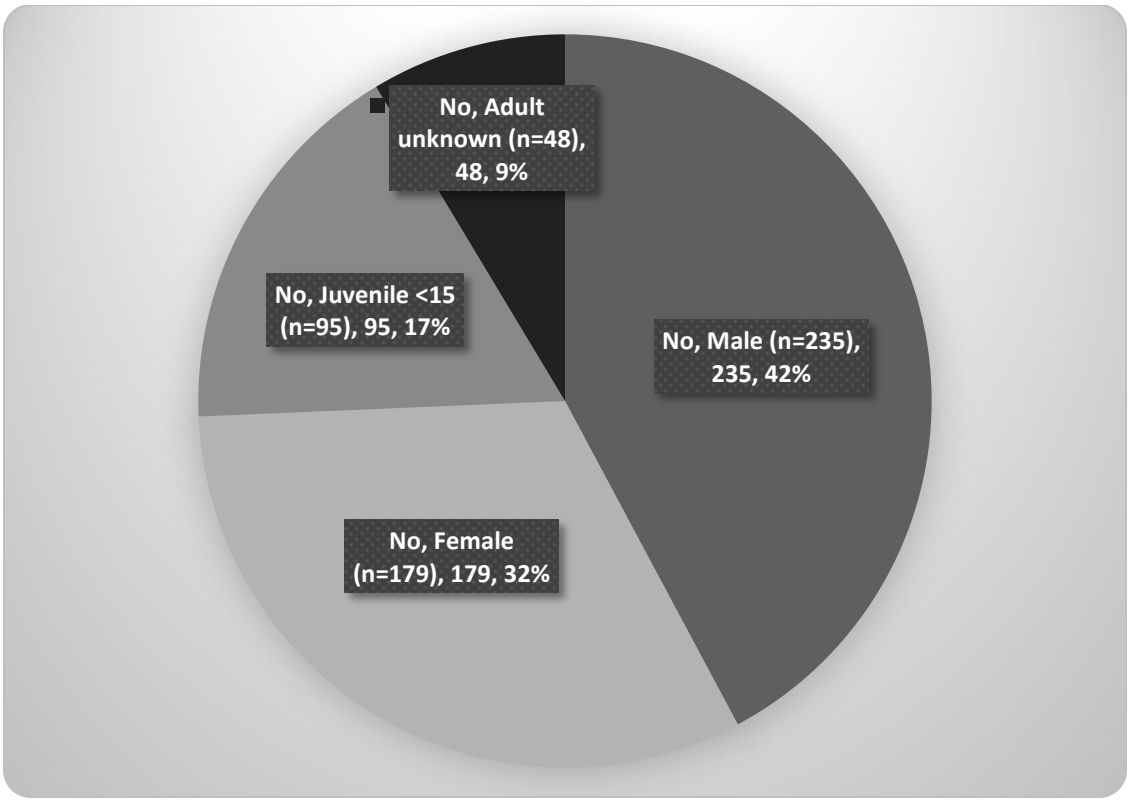

Figure 6. Sex and age of the skeletal individuals (n=557) making up the Black Friars cemetery sample.

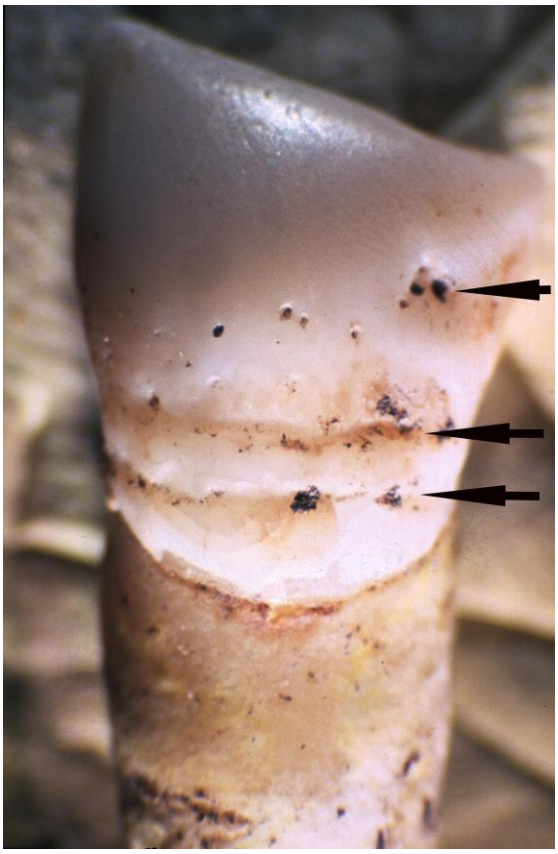

Figure 7. Hypoplastic defects (lines and pits) on the labial enamel of the left mandibular canine. The angular attrition to the cusp precludes scoring of the first-formed lateral enamel for surface defects.

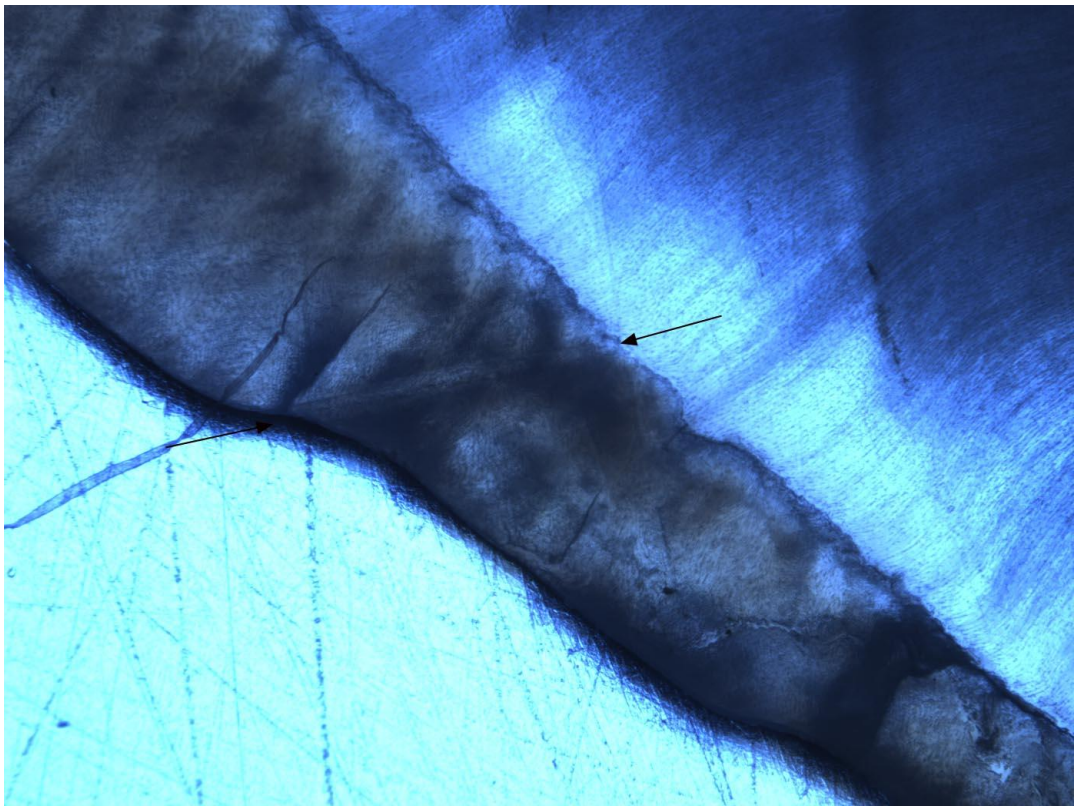

Figure 8. A thin section image of a pathological stria in at the leading edge of an enamel hypoplasia in GP90-304, a female age 45 years from the Gray Friars assemblage (square D).

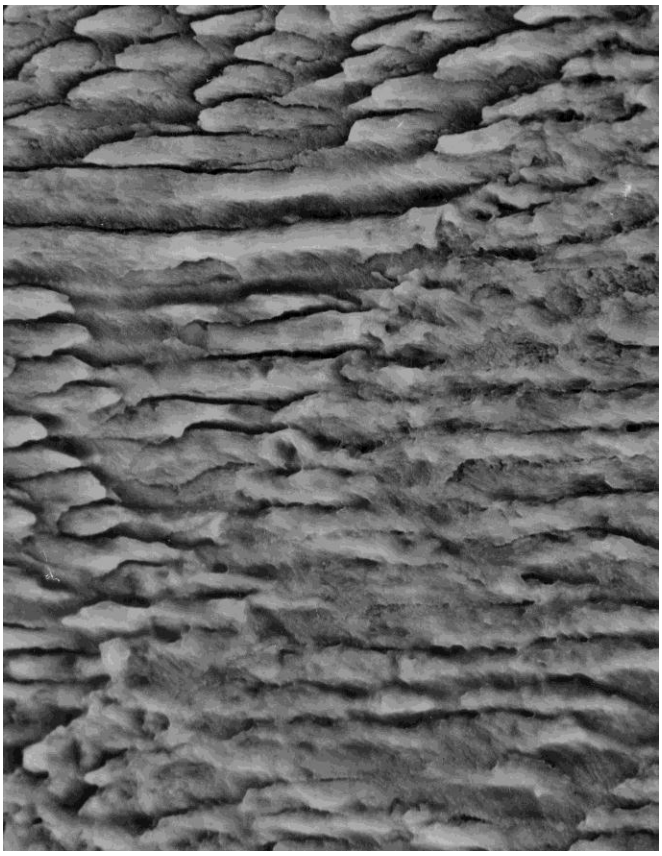

Figure 9. Scanning electron micrograph (SEM) of a pathological stria in a thick section from SBT-81-056, a juvenile age 11.5 years (+/- .5 years). Rod disruption occurs at the bottom left of the image (cervical end) and runs to the top right (cuspal end). The rod on the right side of the image are disorganized and have a melted appearance. The image was taken at a magnification of 1000.

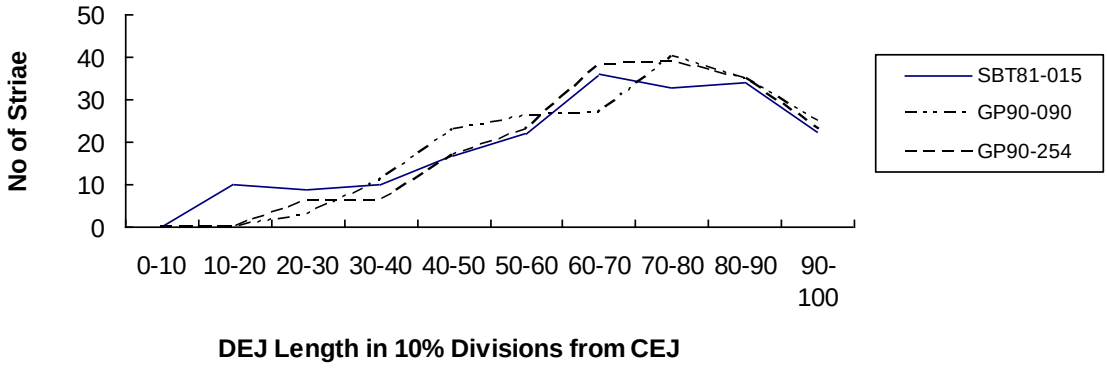

Figure 10. Growth rate in the mandibular canines chosen for the population model. The number of striae of Retzius per 10% increment is similar in each tooth. There is a general increase in the number of SOR as the cervix is reached, indicating that crown elongation is fastest at the beginning of development and slows as growth proceeds.

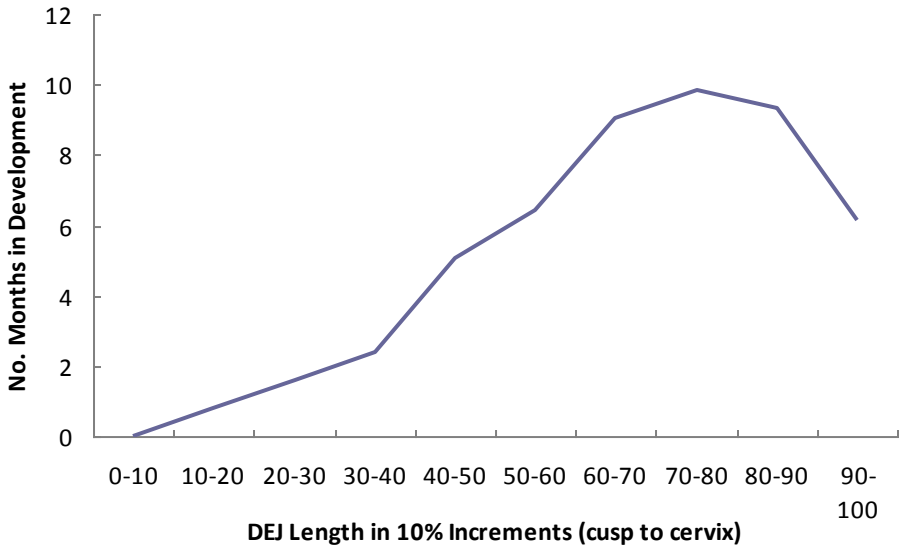

Figure 11. The timing of the development based on the population model. The number of months for development is generally increases as the cervix is reached. The dropoff at the 90th percentile reflects difficulty in seeing and counting SOR in the most cervical regions of the teeth.

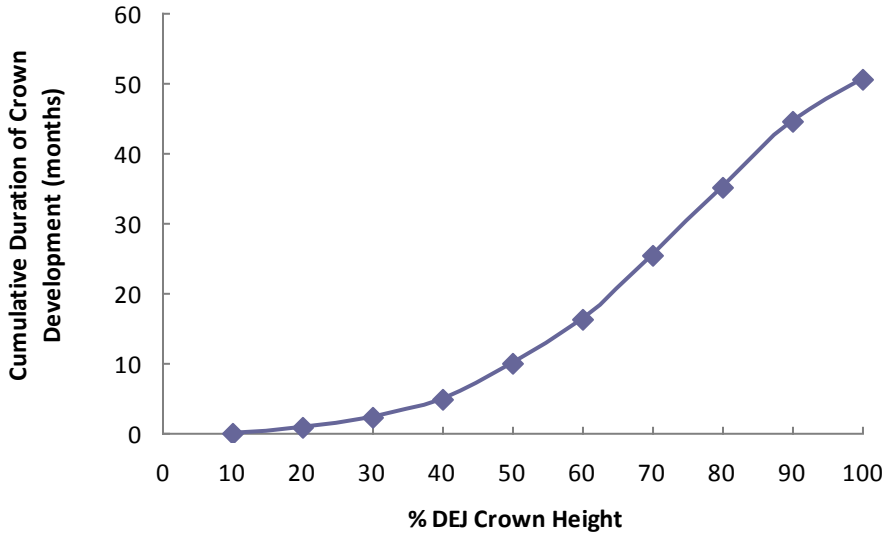

Figure 12. Non-linear growth in the population model of crown development.

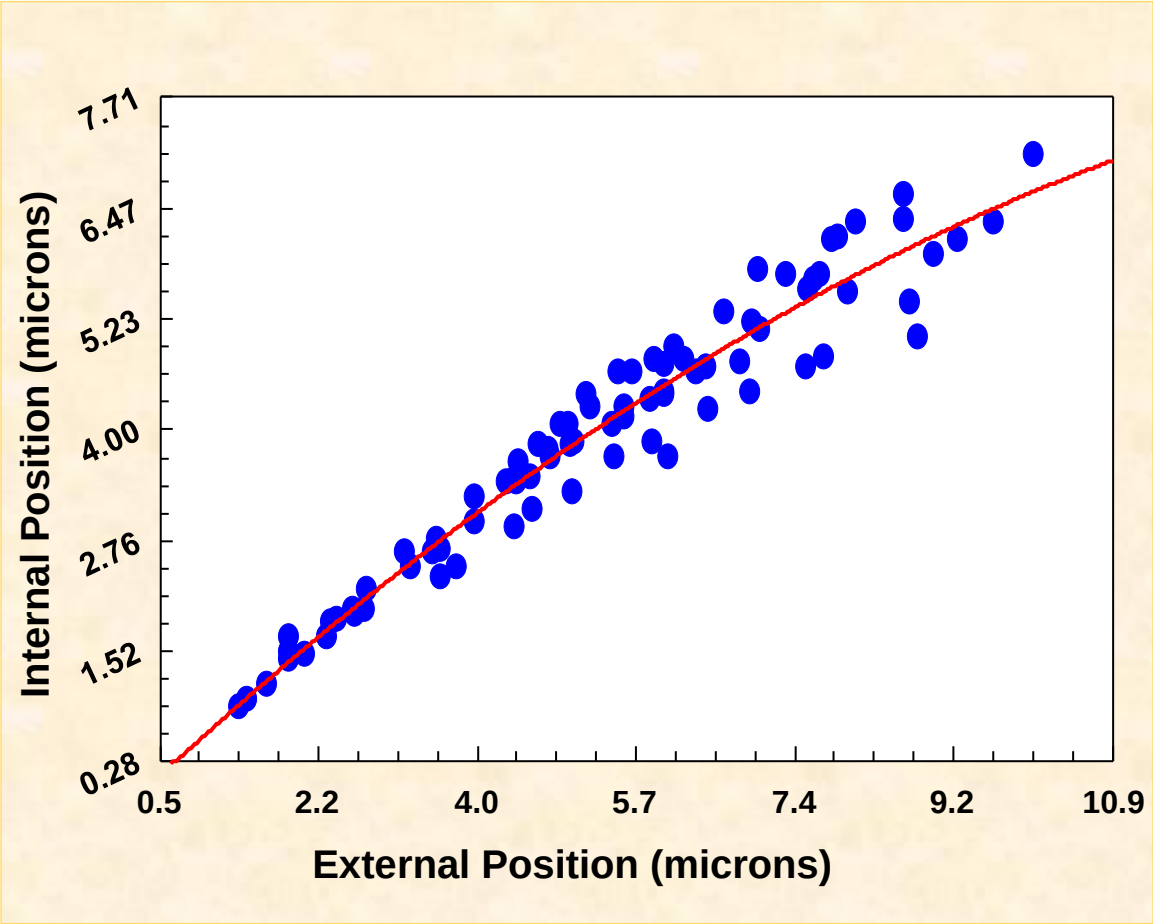

Figure 13. Distance function data for determining internal locations of external structures for the Black Friars sample. Note that the relationship between the internal and external positions of striae of Retzius is curvilinear. The regression equation that describes the relationship is $y = -.353 + .970x - .027x^2$.

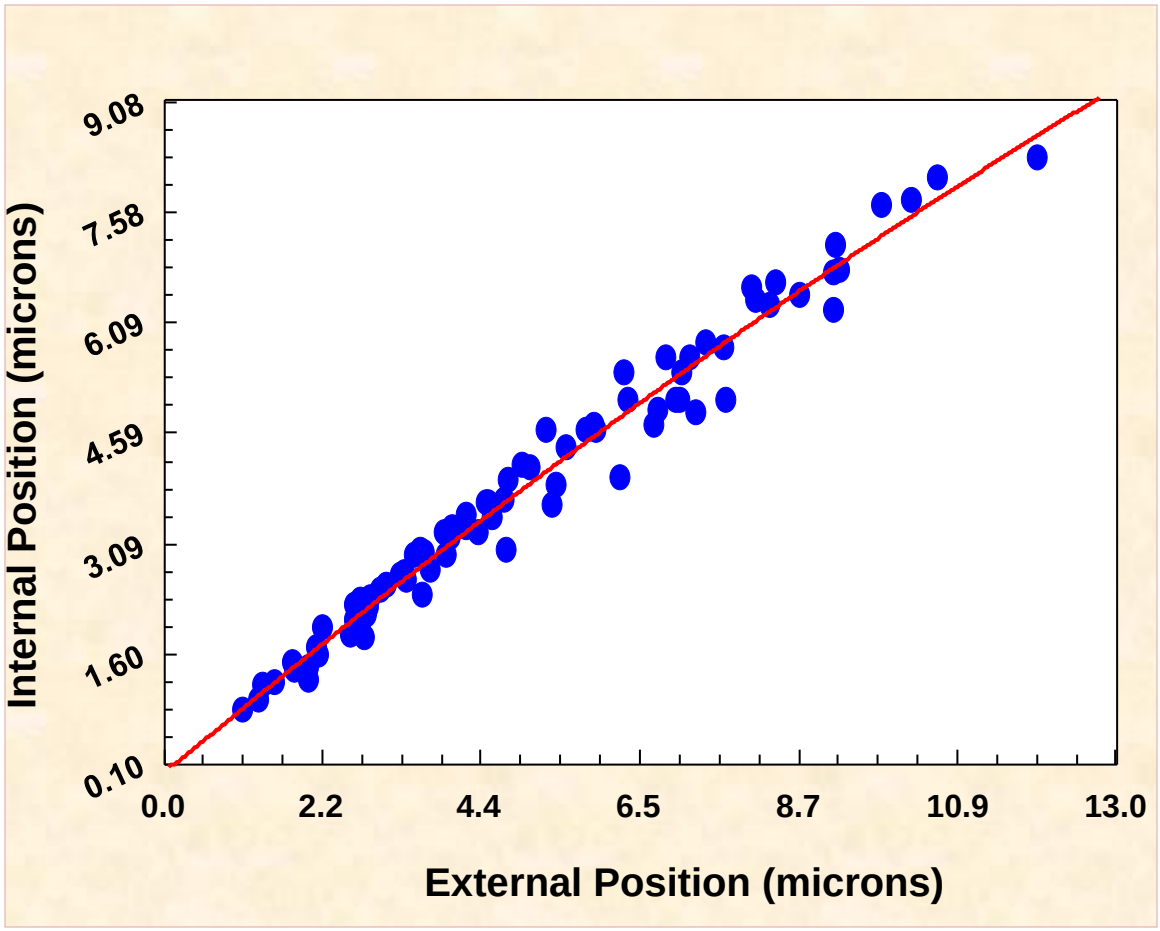

Figure 14. Distance function data for determining internal locations of external structures for the Gray Friars sample. Note that the relationship between the internal and external positions of striae of Retzius is curvilinear. The regression equation that describes the relationship is $y = -.038 + .831x - .009x^2$.

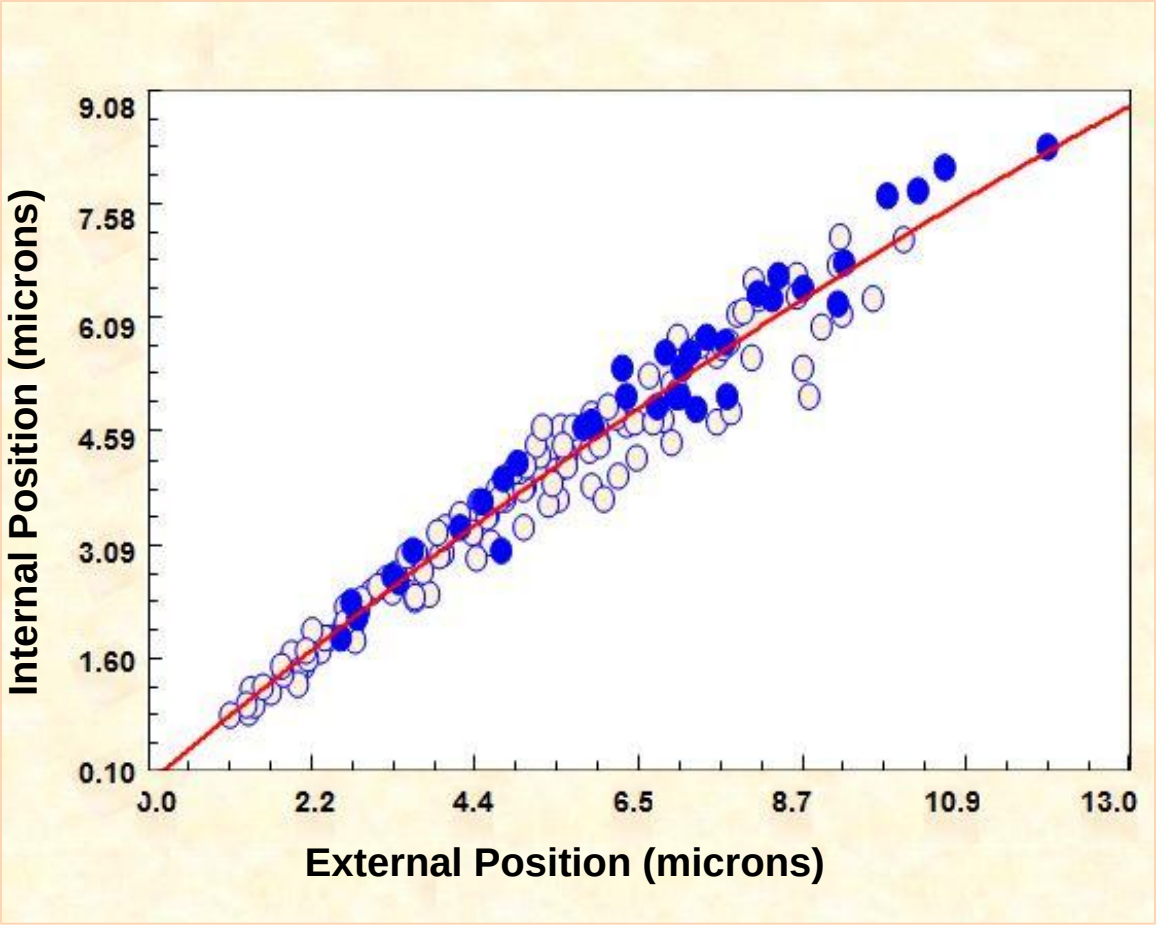

Figure 15. Distance function data for determining internal locations of external structures for the both samples combined. Note that the relationship between the internal and external positions of striae of Retzius is curvilinear. The regression equation that describes the relationship is $y = -.052 + .832x - .011x^2$.

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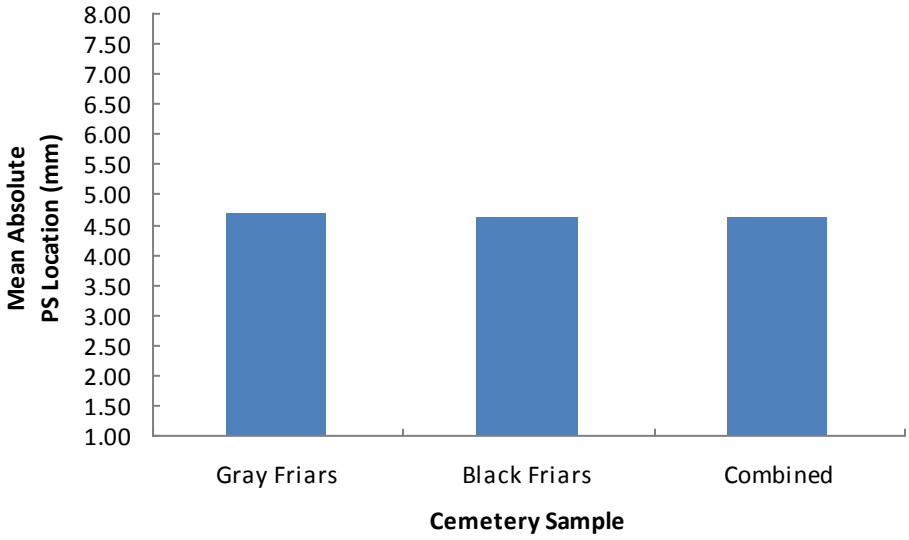

Figure 16. Mean absolute location of pathological striae (PS) in millimeters in both cemetery samples. The Gray Friars sample mean is 4.68mm, while the Black Friars is 4.61mm. The combined mean is 4.63mm.

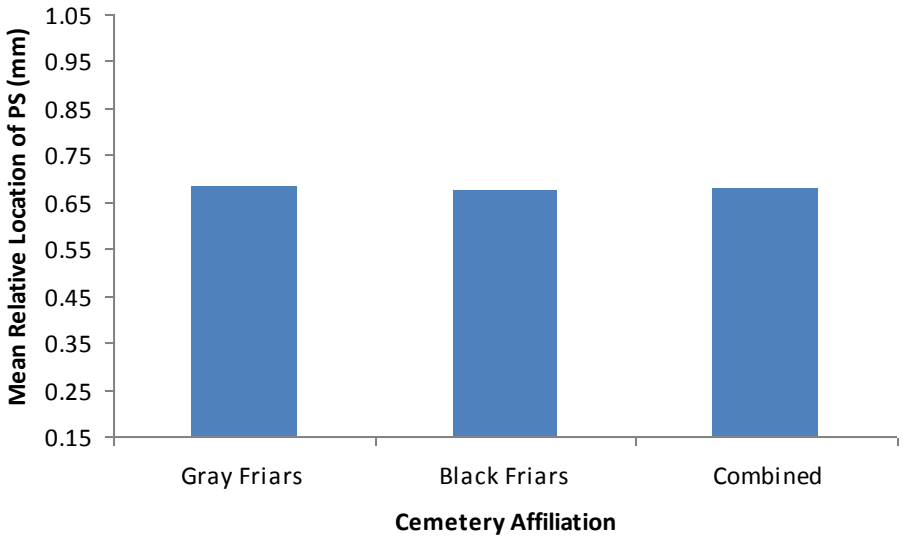

Figure 17. Mean relative locations of pathological striae (PS) by cemetery affiliation. The combined mean for both cemetery samples is 0.68.

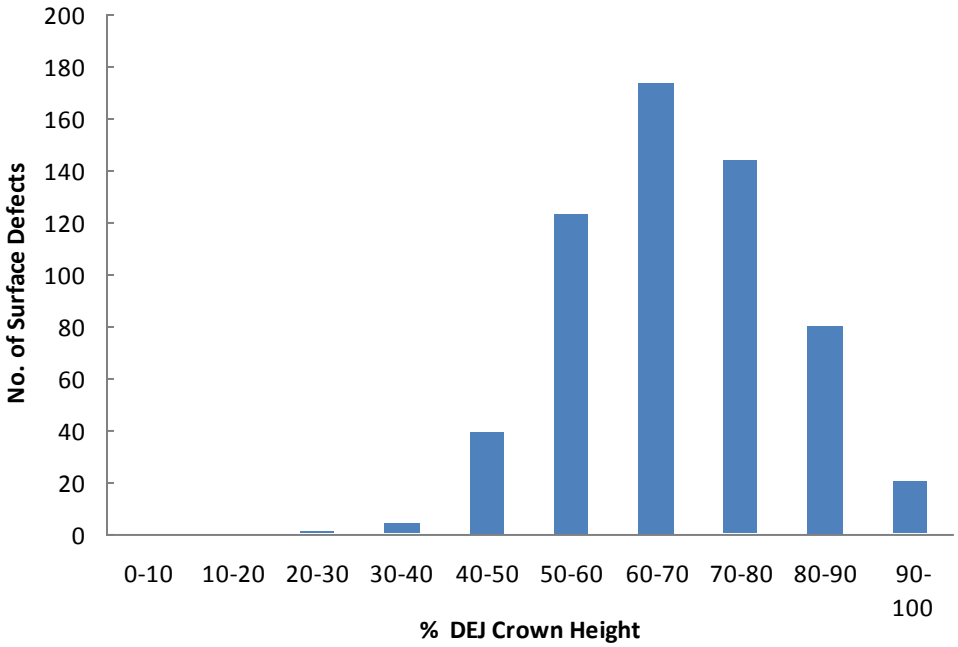

Figure 18. Distribution of surface defects in the Black Friars cemetery sample. Surface defects are most prevalent in 60-70% DEJ crown height, and few to no defects were recorded before the 40th percentile.

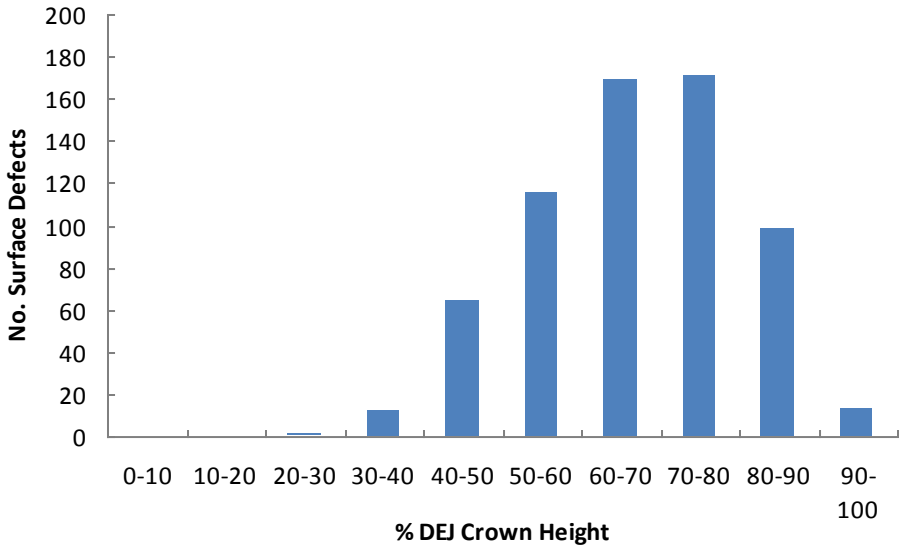

Figure 19. Distribution of surface defects in the Gray Friars cemetery sample. Surface defects are most prevalent in the 60-80% of DEJ crown height. Few to no defects were recorded in the cuspal 40th percentile.

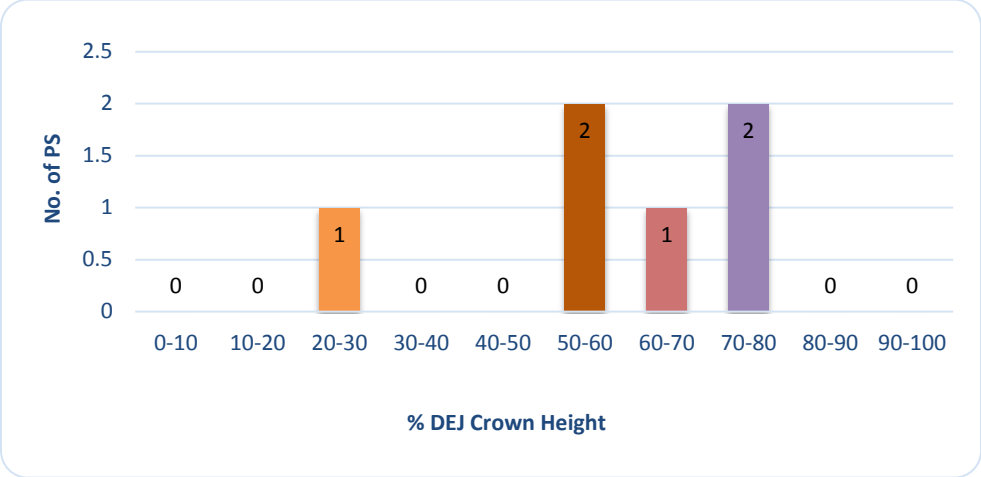

Figure 20. Prevalence of pathological striae (PS) in the Gray Friars sample in 10 percent increments along the DEJ. Prevalence peaks at the 50-60th and 70-80th percentiles.

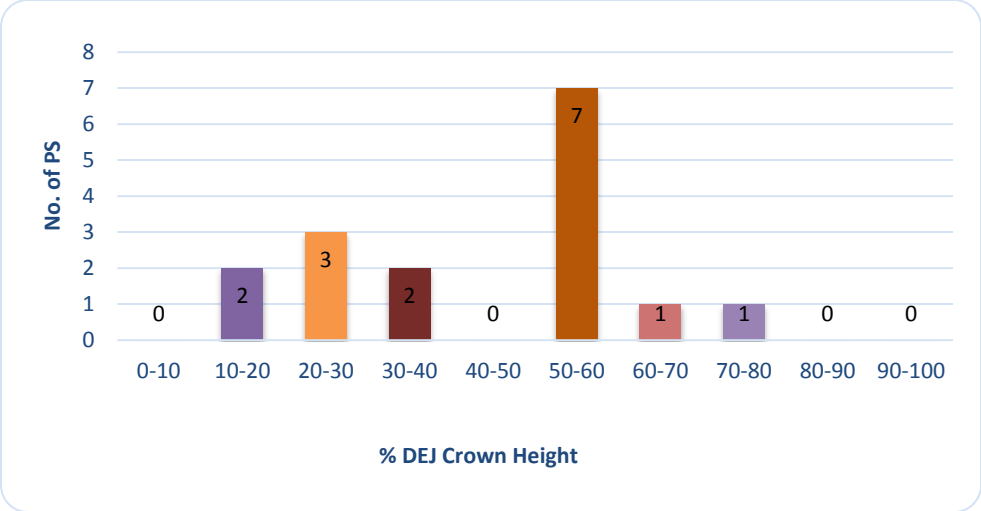

Figure 21. Prevalence of pathological striae (PS) in the Black Friars sample in 10 percent increments along the DEJ. Prevalence peaks at the 50-60th percentile.

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