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Childhood Health and Nutrition: An Exploration of Enamel Hypoplasia Studies Using the Milwaukee County Institutional Grounds Cemetery, 1882-1925

Maura K. Polli

CHILDHOOD HEALTH AND NUTRITION: AN EXPLORATION OF ENAMEL HYPOPLASIA STUDIES USING THE MILWAUKEE COUNTY INSTITUTIONAL GROUNDS CEMETERY, 1882-1925

by

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

Western Michigan University
Kalamazoo, Michigan
April 1997

ACKNOWLEDGMENTS

A project of this type requires the efforts of many organizations and individuals. Milwaukee County funded excavations of the Milwaukee County Institutional Grounds Cemetery (MCIG). Great Lakes Archaeological Research Center, Inc. and the State Historical Society of Wisconsin facilitated excavations.

Norman Sullivan and Marquette University provided access to the MCIG skeletal collection and data on the individuals. Brigitte Charaus and Lisa Iorio assisted with the laboratory work for this project. My thesis committee consisted of: Tal Simmons, Norman Sullivan, and Robert Sundick.

Authors are continually grateful to their editors for sharing their vision while restraining and restructuring their execution. I am especially grateful because I have been blessed with more than my share of talented and patient editors. Tal Simmons has fielded any question put to her with patience and encouragement, in addition to her regularly extensive roles of mentoring and editing. Norman Sullivan provided equipment, staff, data, and feedback at many points in this project. Joseph Polli supplied computer and statistical consulting at crucial and numerous points during the course of this research. Both Elizabeth McNally and Karen Atwell served as editors on

call and made extensive contributions to this paper. Karen Atwell also assisted with the figures.

On a personal note, Elizabeth McNally deserves thanks for sharing my frustration and proving that this can be accomplished by her example. I will always be grateful to Sharon Thomas who supplied my graduate experience with intellectual camaraderie and to Jennifer Pagan who, throughout my career, has unfailingly encouraged my pursuits. Family members, of course, deserve special thanks. Betty, Patrick, Jimm, Teresa, and Mike Polli were always supportive both emotionally and physically. They provided hours of clerical help, without which this would never have been finished on time. I am eternally in the debt of my father, Joseph Polli, for picking up the formatting ball when I moved on to other tasks on another continent.

To my family and friends, whose prodding did not speed the process but whose enthusiasm for my success did inspire.

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Western Michigan University, 1997

The focus of this study is the dentition of a Midwestern United States historic almshouse population, the Milwaukee County Institutional Grounds cemetery (MCIG), 1882-1925. A survey of linear enamel hypoplasia (LEH) in the anterior dentition of a subsample of this population has been conducted to demonstrate incidences of childhood stress. LEH is symptomatic of extended periods of disease or nutritional stress experienced during the formative years of tooth development.

A subsample of 140 individuals was examined for LEH. The labial surface of the anterior dentition was examined for the frequency and chronological distribution of LEH. The frequency of LEH ranges from 42.4% on the left mandibular canine to 13.7% on the right maxillary lateral incisor. 58.6% of the individuals exhibited at least one incidence of LEH. The peak age of LEH occurrence varies dependant on which age conversion is employed, a constant velocity approach or a non-linear growth scale. The MCIG results are then compared to a number of other studies.

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CHAPTER I

INTRODUCTION

The purpose of this study is to document linear enamel hypoplasia (here after referred to as LEH) rates within an historic industrial population (the Milwaukee County Institutional Grounds cemetery, or MCIG), to compare those rates by tooth type within the population, and to examine the Milwaukee data in light of other populations both within and outside of the Midwestern United States. This study makes use of an historic skeletal population recently recovered from Milwaukee County, Wisconsin. In 1991 and 1992, a substantial section of a county poorhouse cemetery, dating from 1882 to 1925, was removed to make way for expanding hospital facilities on the Milwaukee County Institutional Grounds.

Two locations within the main cemetery were for the exclusive interment of infant burials. Research on the cemetery's infant remains has produced a number of studies of infant mortality and morbidity (Charaus, et al. 1995; Hutchins 1994; Hutchins, et al. 1995; Luft 1994; Nelson 1995; Nelson, et al. 1996). The remaining portions of the cemetery were mainly adults. From the subsample selected for this research, the majority of those

individuals seem to be adult males of middle age (35-50 years). In this study there were 77 individuals in this category which amounted to 61.5% of the individuals in cases where sex had been determined.

The remainder of the study will be organized as follows. This Chapter provides a short review of dental research and the comparison populations that will be examined in light of MCIG results. Chapter II discusses the history and archaeology of the MCIG cemetery. Chapter III reviews dental etiology and histology, highlighting dental development necessary for understanding LEH studies. The literature review in Chapter IV explores foundational enamel hypoplasia research and debates about tooth development and its relationship to LEH interpretations. Chapter V discusses research methodologies, including how the MCIG sample was surveyed and the data analyzed. Chapter VI presents the results of the MCIG sample's internal and external comparisons. Conclusions and suggestions for future research are discussed in Chapter VII.

Dental Research

Current research on human skeletal populations commonly make use of a variety of dental observations. Preservation of dental material, in archaeological contexts, is often better than that of associated skeletal remains. Since dental material has a relative narrow window of growth, and

because its structure is immune to most later events (except attrition and caries development), teeth have be utilized to study developmental disturbances of childhood. The incidence of LEH is used as an indicator of stress during childhood development. Developmental stress can be the result of chronic malnutrition, severe episodes of infection or other diseases. Anything that prevents the body from receiving or processing an adequate diet can interfere with the growth of bones and teeth. Dentition is particularly useful in these incidences because, once formed, teeth do not remodel, nor do they repair any damage incurred during their formation. The resulting defects in dentition exist as a permanent record of developmental interruptions during growth of the tooth.

Comparison Populations

Previous studies of historic industrial communities in the United States have reported LEH rates above 70% (Lanphear 1990; Blakey 1987). MCIG will be compared to those populations and other collections from a variety of regions. Populations studies, used for comparison, were chosen for their representation of particular periods of history, economic development, and size of sample. Most of the comparison populations, including the Milwaukee sample, are comprised of individuals of low socio-economic status.

The Monroe County Poorhouse cemetery was located in Rochester, New York and was in use between 1826 and 1863. The population contains residents of the poorhouse, and is therefore, representative of the "poorest members of the lowest socioeconomic class" (Lanphear 1990:38). Lanphear reports a high rate of hypoplasia frequency (73.0%), from the individuals of Monroe County Poorhouse. Another New York (also New England) derived sample consists of the Quebec City prisoners' cemetery (1746-1747), which produced a collection of 44 individuals (Wood 1996).

Other eastern United States derived samples include Kelley and Angel's (1987) report on a number of Virginia and Maryland sites comprising slave populations from the eighteenth and nineteenth centuries. The compiled sites total 25 individuals from the 1700s and 34 individuals from the 1800s. Rathbun (1987) also worked with a population from a slave cemetery, in nearby Charleston, South Carolina, dating from 1840-1870. Thirty-six individuals from this sample exhibited enamel hypoplasia frequency peaks between 2 and 3 years of age. In the same year, Blakey (1987) presented the results of a survey of the First African Baptist Church cemetery in Philadelphia. The cemetery was open between 1823 and 1841 and contained individuals exhibiting an extremely high reported rate of hypoplasias at 82.2%.

Midwestern United States populations consist of the Chicago study by

Sarnat and Schour (1941) and the Hammon-Todd collection research by Goodman (1988). The Hammon-Todd collection was comprised of individuals of lower socio-economic class from the Cleveland area (1855-1913).

Prehistoric studies include the Schulz and McHenry survey of California groups, and Goodman, et al.'s (1984a) research of the Dickson Mounds collection. Historic work outside of North America is represented by the Newton Plantation cemetery of slaves from Barbados (Corruccini, et al. 1985) and Swärdstedt's (1966) work with Medieval social class distinctions from Sweden. Contemporary dental studies included are Alcorn and Goodman's (1985) work with Jordanian semi-sedentary villages and Goodman and co-workers (1987) involvement in the long term health studies of communities in rural Mexico.

CHAPTER II

HISTORY AND ARCHAEOLOGY OF THE MILWAUKEE COUNTY INSTITUTIONAL GROUNDS

Archaeological Project Background

As has often been the case over the course of the 20th century, many areas outside of cites that were considered rural in the 1910s are now engulfed by urban sprawl. In 1852, when the county board in Milwaukee decided on an institutional pathway for assisting the city's poor, an outlier farm was purchased for building facilities to contain this social service. As the growth in the community warranted, the government run property and services expanded from an almshouse and farm to an orphanage, a mental health facility, an hospital complex, a nursing college, a medical school, and multiple cemeteries (Avella 1987). By the 1990s, further expansion of the county's hospital facilities led to the development and construction of an Ambulatory Care Center on the grounds of the Milwaukee County Medical Complex (Richards and Kastell 1993).

In August of 1991, the earliest stages of construction on the Ambulatory Care Center disturbed the second of three cemeteries once used

by the county on these grounds. This disturbed cemetery was located at the NE1/4, SE1/4, SW1/4, NW1/4 of Section 28, Township 7N, Range 21E, in the City of Wauwatosa, Milwaukee County, Wisconsin (Figure 1). The most recently used cemetery closed in 1974 and had been well marked during its use. The two earlier cemeteries, however, had been forgotten, and the location and extent of interments were not known.

Upon discovery of the second cemetery, the size of the hospital expansion, the state of construction, and problems and costs involved in potentially redesigning the Ambulatory Care Center convinced the director of the State Historical Society of Wisconsin (SHSW), H. Nicholas Muller III, to grant permission for excavation and analysis of the human remains. The Milwaukee County Medical Complex and the Milwaukee County Department of Public Works then contracted the excavations to a local archaeological firm, Great Lakes Archaeological Research, Inc., (GLARC). The estimation of the remaining number of graves was determined to be several hundred individuals and archaeological mitigation began on September 4, 1991. The scope of the project was continually enlarged as more areas of the hospital grounds were impacted by construction (Richards and Kastell 1993).

Original discovery of the cemetery occurred at the west end of construction, on the south edge of the Milwaukee County Institutional

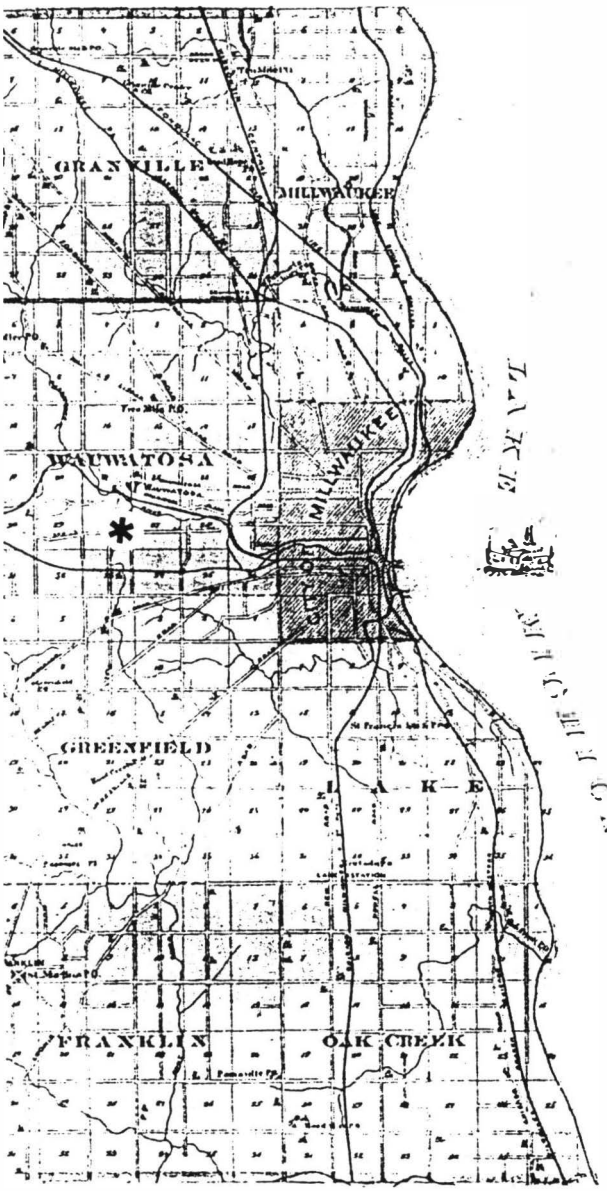


Figure 1. 1876 Map of the Project Location in Wauwatosa, to the West of Milwaukee, Wisconsin (after Olson 1987:6).

Grounds, approximately 40 meters to the west of the School of Nursing building. Ultimately, the cemetery grounds were determined to extend beneath two roads and a parking lot, almost ringing the landscaped grounds of the School of Nursing. The excavations culminated in the removal of 1649 burials, of which 588 were subadults.

A knoll of burials to the west of the School of Nursing was removed for the construction of a parking ramp. Interments probably extend further west of these excavations, but are on Froedtert Hospital property and remain undisturbed. This cemetery location has been registered with SHSW (Overstreet and Sverdrup 1992a). The earliest cemetery associated with the poor farm, in the southeast portion of the grounds at West Wisconsin Avenue and Windsor Court, has also been catalogued with SHSW (Overstreet and Sverdrup 1992b).

The cemetery boundaries continued around the south end of the School of Nursing, north of three artificial ponds at this edge of MCIG. The final burials excavated were located under a parking lot, north of the School of Nursing building. The layout of the burials suggests that the School of Nursing Residence probably impacted a number of the graves when it was erected in 1933 (Figure 2). West of the School of Nursing, construction of a road and a parking/drop-off area at the building's main entrance may have additionally impacted graves. Between the previous modification of the

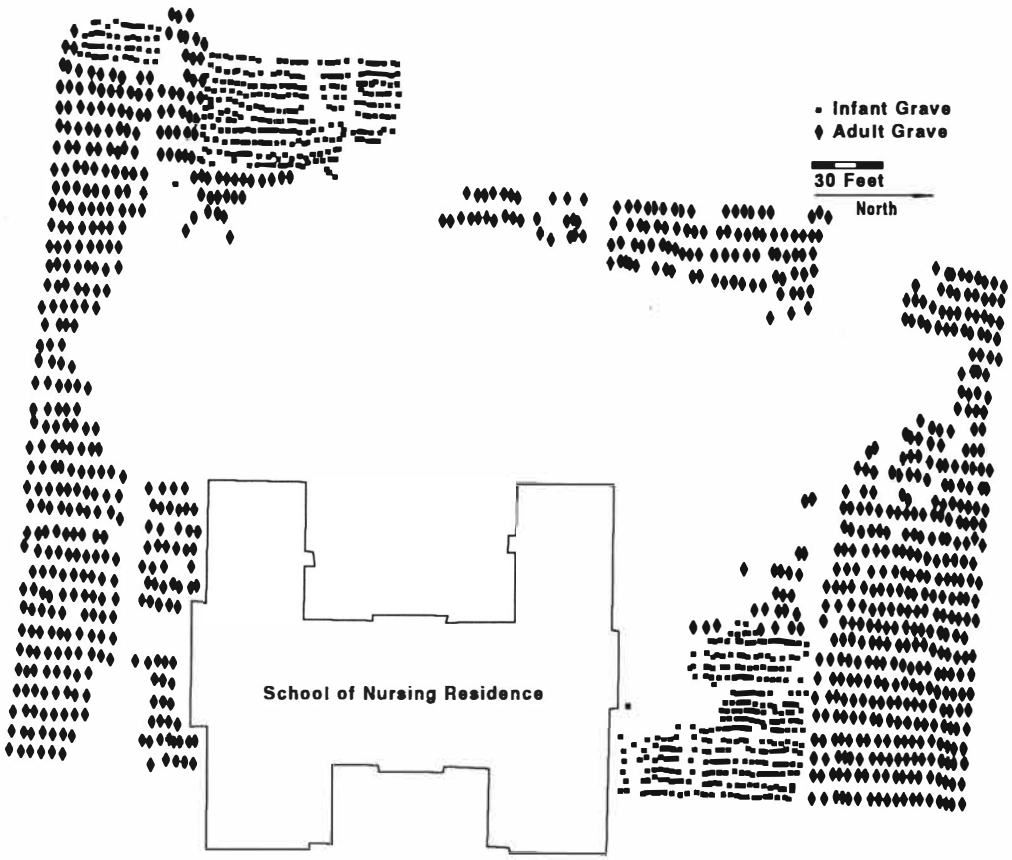


Figure 2. Map of the Location of Excavated Graves in Relation to the MCIG School of Nursing Residence (after Richards and Kastell 1993:57).

landscape and the still protected area of the cemetery, the actual number of interments originally in this cemetery could easily be double the recovered number of individuals. Richards and Kastell (1993) estimated that 2985 burials were disturbed prior to the 1991 and 1992 excavations.

Fieldwork for the removal of graves from MCIG was completed on November 16, 1992, followed by the transfer of human skeletal remains to

Marquette University in January of 1993. Dr. Norman Sullivan, Associate Professor, Department of Anthropology, is the Principal Investigator for the osteological analysis of the collection. For the term of Marquette's analysis of this material, Dr. Norman Sullivan made the collection available to the author for research purposes.

History of the Milwaukee County Institutional Grounds

In 1833, southeastern Wisconsin was opened to land speculators after the Black Hawk War and subsequent land cessions. By 1835, the United States government had begun selling land in the townsite area of Milwaukee (Conzen 1976; Still 1965). Milwaukee's history is of immigrant populations practically from its inception. By the time ethnic backgrounds were recorded in 1848 census, over half of Milwaukee's population were immigrants, mainly from Germany, England, and Ireland, in addition to its French-speaking settlers and American pioneers. In 1835, the population of Milwaukee was 125. By 1860, Milwaukee had a total resident population of 45,264, with 50.5% being foreign born (Conzen 1976).

In 1835, treatment of the poor reflected national expansion and the belief in American independence. Ideology of the period held that as a young country, anyone wanting to better themselves could not only provide for themselves and their families, but could possibly "get ahead". Persons

not able to take care of themselves were perceived as failing to take advantage of appropriate opportunities. The proscribed cure was to find work in order to become an independent and contributing member of the society. Particular circumstances that left one in poverty were not of importance, only the route to regain independence. The solution to aid those in need was to provide work first and, failing that, to provide charity. Charity, however, was to be as distasteful as possible in order to motivate people to productive work (Avella 1987).

With the development of Milwaukee's administrative programs on the state and county levels of government, social programs to deal with the poor were among the community's earliest. The beginning of public aid was a two-fold system administered by Milwaukee County and the City of Milwaukee. County provisions were put toward an "outdoor" relief system that provided the poor with food and occasional lodging, even going so far as paying for widows' and orphans' fees for travel to relatives out-of-state. Milwaukee County also had a Superintendent of the Poor appointed on an annual basis. At the same time, the City of Milwaukee constructed a poor house to which the Milwaukee City Medical Association sent physicians (Avella 1987; Leavitt 1982). By the time Wisconsin became a state in 1848, the jurisdiction of poor relief had been delegated, and the county retained responsibility. The existing almshouse in the city was given to the county to

facilitate.

A cholera epidemic from 1848 to 1850 added to the problems of poor relief, and the numbers of destitute individuals increased. Options for the poor evaluated by the country included renovating the almshouse in the first ward or purchasing land for housing and a farm. Since the almshouse was considered too rundown to be reclaimed, a 160 acre farm was purchased in 1852 from County Board Supervisor Hendrick Gregg. Later acquisition of surrounding areas, totaling 1200 acres, settled the county facilities in Wauwatosa (Avella 1987).

Outdoor relief was not replaced by the permanent structures of social welfare in Milwaukee, but continued concurrently with the indoor program with the intent of meeting the needs of those not as desperate. Soup kitchens were supported by private donations and, in early 1858, some type of food supplements were being provided for 720 individuals. Families broken up by the Civil war in the 1860s also required assistance, and voluntary collections were taken up by the Milwaukee Relief Society to purchase wood, tea, sugar, soap, and candles for the city's poor (Still 1965).

The poor farm concept was based on a New York model. Creation of an undesirable indoor relief system was intended as the only solution for those who could not survive on their own. At the same time, the indoor relief program provided a method for residents to contribute to their care by

farming or engaging in small industry. The farm purchased by Milwaukee County was considered ideal, in part, because it had "crops in the field" that could be tended by residents (Avella 1987:201).

Milwaukee County's facility became a multipurpose dwelling that took care of the poor, the sick, the insane, and the children of prisoners or the poor. Managing all of these groups in a single facility proved to be a problem, and the County Board made numerous inspections and complained of inadequate conditions (Richards and Kastell 1993). The first step in separating the various constituents of the county poor house was to build a separate hospital building to care for those who were ill (erected in 1860). By 1898, a Home for Dependant Children had been added to the county grounds.

Mentally ill residents became another problem when they began to overrun the almshouse. Milwaukee County sent inmates to state facilities in Madison and Winnebago, but eventually those facilities became crowded, and debates over Milwaukee exceeding their quota of referrals ensued. Facilities in one wing of the original almshouse in Wauwatosa were not adequate, and in 1880, the Milwaukee County Insane Asylum was opened on the county property. Crowding at this facility was inevitable, and, in 1889, Milwaukee County developed separate programs for the incurably insane (at the Asylum for the Chronically Insane) and the supposedly curable cases of

insanity (at the Milwaukee County Hospital for the Acute Insane). The "curable" patients continued working on the County farm (Avella 1987).

History of the Milwaukee County Institutional Grounds Cemeteries

The first record of a cemetery being used on the Milwaukee County grounds is an 1872 death certificate of an infant buried at the poor farm. The Milwaukee County Board of Supervisors expressed concern in 1878 with the cemetery's unsuitable location. Most of the land set aside for the cemetery was in marshy ground, and the small portion of high ground was already covered in graves. An 1876 map of Milwaukee County has this cemetery recorded with a cross in the middle of Section 28, at the intersection of Wisconsin Avenue and 84th Street (Figure 3). A register of burials was started in 1882 and notes interments in a "new cemetery" in August of that year (Richards and Kastell 1993).

A Works Progress Administration map from the 1920s illustrates the existence of the second cemetery southeast of the Insane Asylum buildings (Figure 4). This cemetery was used until a third cemetery was created in 1925, north of Watertown Plank Road, which remained open until 1974. The second cemetery was disturbed during the 1991 construction of the Ambulatory Care Center of the Milwaukee County Hospital. The western portion of the cemetery remains intact on Froedtert Hospital property. The

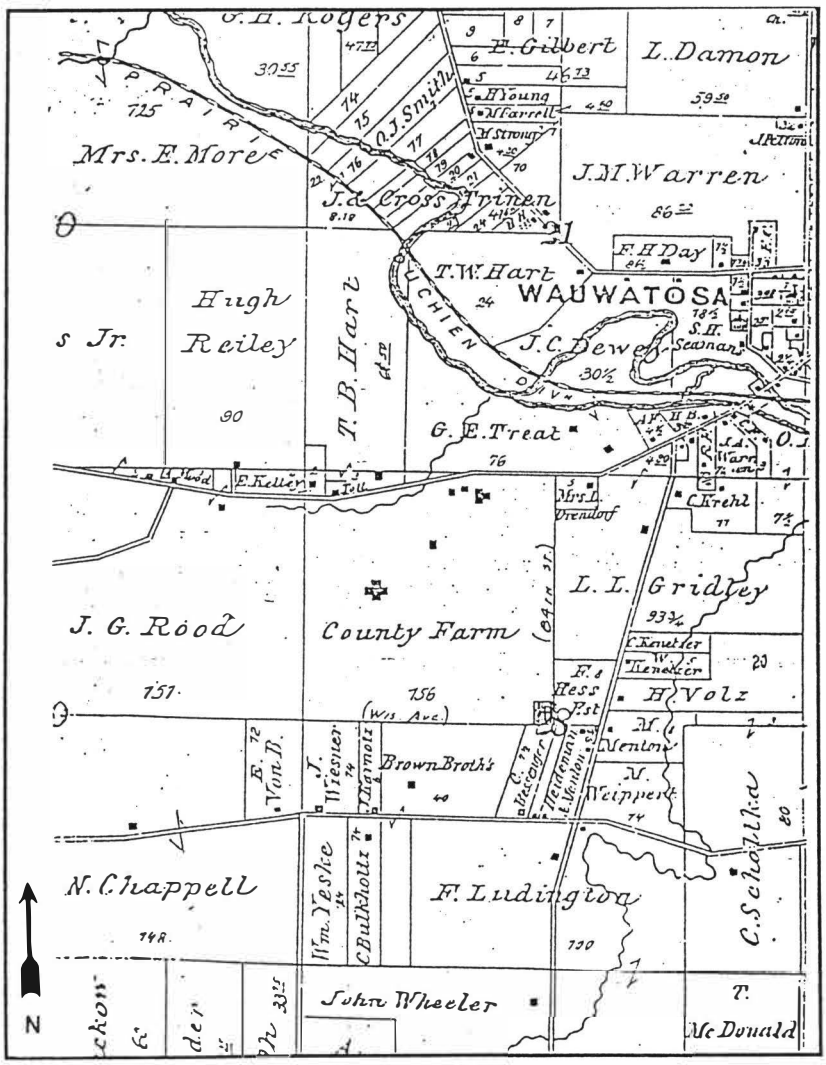


Figure 3. Plat Map From 1876 With the Original Cemetery Marked by a Cross in the Southwest Corner of the County Farm (from Richards and Kastell 1993:29).

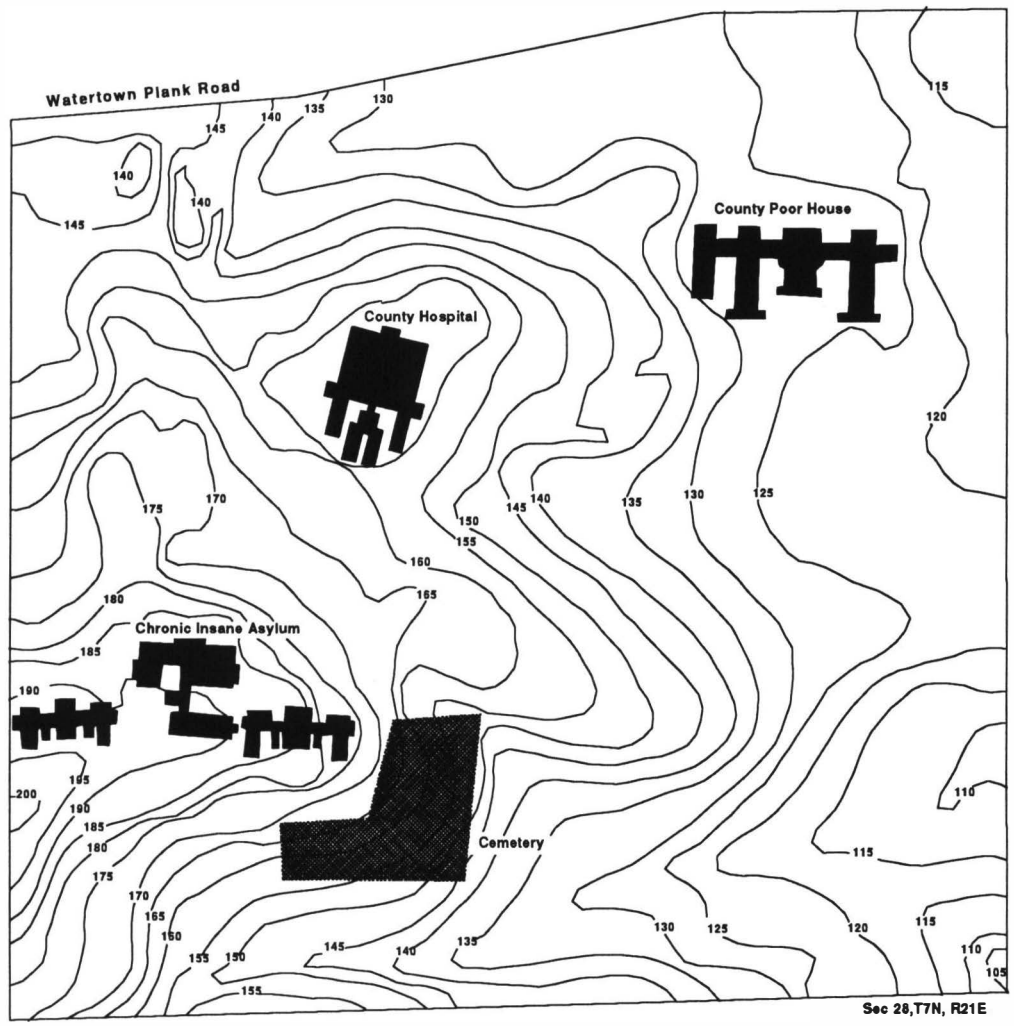


Figure 4. Topographic Map of the Second Pauper Cemetery, From the 1920s (after Richards and Kastell 1993:36).

remainder of the cemetery was excavated by Great Lakes Archeological Research Center, Inc. between 1991 and 1992. The location of the first cemetery and the integrity of the rest of the second cemetery were confirmed with remote sensing and some test excavations (Overstreet and Sverdrup

1992a, 1992b). Figure 5 illustrates the location of all three cemeteries in relation to each other and modern roads.

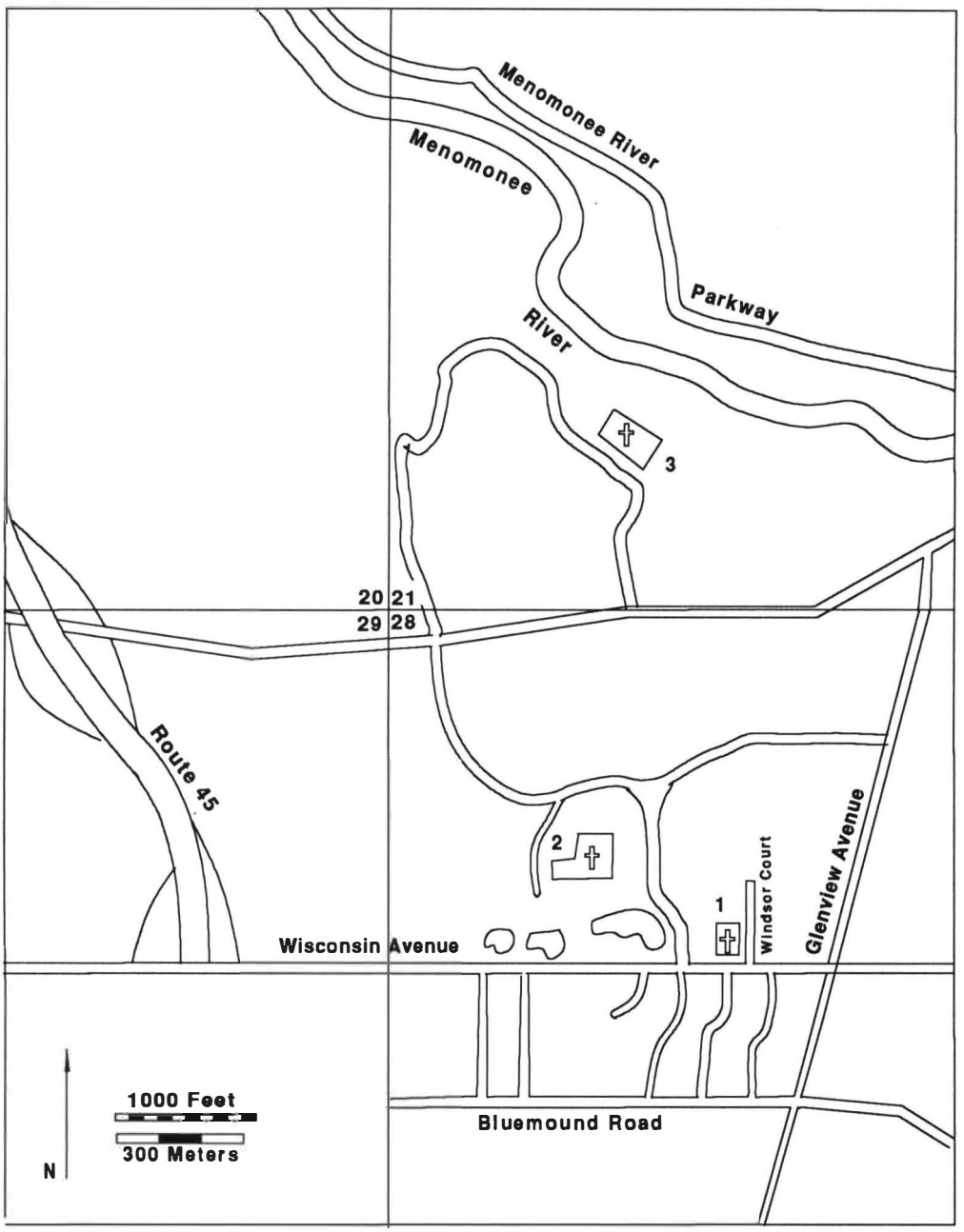


Figure 5. Location of All Three Cemeteries in Relation to Modern Roads (after Richards and Kastell 1993:37).

CHAPTER III

ENAMEL ETIOLOGY AND HISTOLOGY

Dental Anatomy

A number of sources were consulted for compiling this summary of dental anatomy and histology (Bhussry and Sharawy 1991; Hillson 1993; Sharawy and Yeagar 1991; Steele and Bramblett 1988; Suga 1992).

Tooth structure can be broken down into three main categories: enamel, dentin, and cementum. Each is composed of varying amounts of inorganic material and therefore has different degrees of hardness. The underlying structure of the tooth is provided by the dentin. The enamel caps the dentin and is usually the only the portion of the tooth visible in the mouth during life. The tooth articulates with the surrounding alveolar bone through the root. Even though the majority of the root is dentin, cementum makes up the exterior component. The cementum is a thin coating that provides a platform of attachment for anchoring the periodontal ligaments.

The hardest substance of the tooth, and consequently the body, is the enamel. It is composed of less than 5% organic matter, does not have cells or

specific organic structures, and is not considered to be living tissue (Hillson 1993; Suga 1992). The high mineral content of enamel results in it being the best preserved of hard tissues in most archeological settings. The dentin consists of 30% organic material, and is the first portion of the tooth to develop. The cementum consists of around 50% organic materials, and its main purpose is connective. Cementum is the weakest tissue of the tooth and usually does not survive in archaeological samples.

There are a number of landmarks on a tooth that describe adjoining surfaces between the types of tooth tissue (Figure 6). These landmarks are used as reference points in making dental observations. The cemento-enamel junction is at the base of the crown, and is sometimes known as the neck or

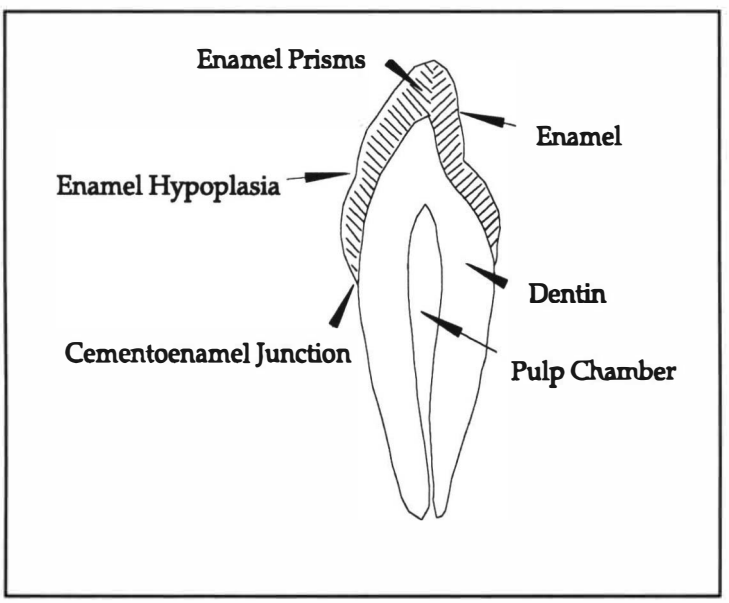


Figure 6. Tooth Anatomy, Representation of the Longitudinal Section of a Canine (after Rose, et al. 1985:283).

cervix. The dento-enamel junction describes the border between the enamel cap and the dentin. The pulp cavity is the opening inside the dentin that serves as a canal for the nerves and arteries. The crown is composed of enamel and dentin and is the area of the tooth above the root.

Dentition can be divided into a number of different categories (Figure 7). A separation can be made between the anterior dentition and the posterior dentition, or cheek teeth. The anterior dentition is made up of the incisors and canines while the posterior dentition is composed of the premolars and molars. Distinctions can also be drawn between primary (deciduous) and secondary (permanent) dentition.

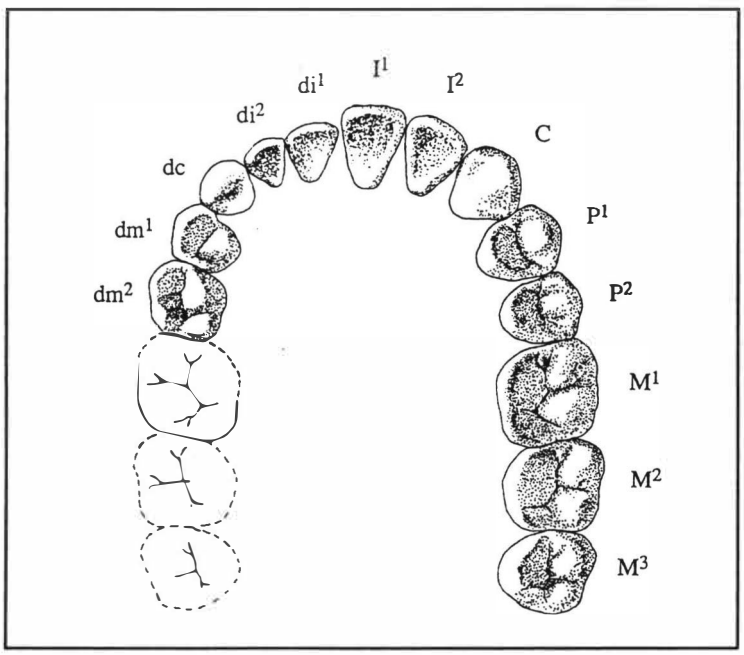


Figure 7. Dental Arcade, Occlusal View of Maxillary Deciduous and Permanent Dentition (from Schwartz 1995:171).

Normal Enamel Development

Enamel is formed through the dual processes of organic matrix formation, or production, and mineralization. The structure of the enamel is laid down in the matrix stage and mineralized in the second stage. Both processes are closely linked but variations in either stage cause divergent results, so each will be treated individually.

The ameloblast is the cell that is crucial for enamel formation. It adapts its function in enamel creation to whichever chemical process is required. In matrix production, the ameloblasts set down the organic netting of the enamel and secrete crystalline apatite into this structure. The matrix is partially mineralized, but the organic ratio is still relatively high when compared to the finished enamel. At this stage, the matrix contains amelogenin, the protein from the matrix stage. Once the ameloblast converts over to mineralization mode, also termed the maturation stage, the amelogenin is replaced with apatite for complete mineralization. During mineralization, the matrix looses more than 90% of the previously formed organic components, mainly protein and water. Maturation of the enamel, or final mineralization, begins before the completion of matrix formation and prior to the matrix reaching full thickness.

Ameloblasts develop the enamel starting from the dentin surface and

move outward toward the exterior of the crown. Each sequence of ameloblasts starts and finishes on a slightly staggered schedule. The cells at the coronal area of the cusps begin and end formation first. This sequentially staggered starting process continues down the sides of the tooth until, finally, the cells near the neck of the tooth, or the cemento-enamel junction, begin matrix formation. The resulting picture of enamel development is one of layers being built on the dentin foundation, beginning at the crown and finishing closest to the root of the tooth. Enamel thickness varies according to location on tooth crown.

The ameloblasts are formed after the odontoblasts begin production of the dentin. The odontoblasts perform a similar function to the ameloblasts but are responsible for the development of dentin. These two types of cells begin their respective protein synthesis from the same starting point. The odontoblasts begin first by depositing the organic matrix of dentin, or the predentin. As the odontoblasts move away from the starting line, they stack layers of dentin upon each other to form the underlying shape of the tooth. The dentin lies beneath the enamel at the tooth crown and is the chief component in the tooth root. The ameloblasts only begin formation of the enamel matrix after the first layer of the dentin has been developed.

The other main component of the tooth root is the cementum. Formed by cementoblasts, cementum is the layer coating the root of the tooth. It

serves as an attachment for the periodontal ligament, which holds the tooth in place in the surrounding bony structures of the alveolar of the maxilla or mandible.

Enamel Development Defects

Suga's (1992) experiments with rat incisors have shown that environmental (chemical) disruptions to enamel development during matrix formation interrupt the secretory activity of the ameloblasts. This results in an observable hypoplastic line in the enamel which is sometimes accompanied by hypomineralization. If the causative agent of the disturbance is removed, the ameloblasts usually recover their normal function and resume developing enamel matrix at an appropriate depth. If the ameloblasts do not recover completely from the disruption of matrix formation, or if the causative agent is not removed, enamel matrix formation does not return to fulfill enamel potential. Hypomineralization occurs when the ameloblasts do not regain their full function before entering the maturation/mineralization stage or when they are interrupted in this stage. These conclusions are challenged by Suckling's research (1989, 1986), which maintains that hypomineralization/hypocalcification can be a result of disruptions to the matrix formation also.

Interruptions in matrix and mineralization of enamel can result in

varying degrees of external and internal hypoplasias in addition to various versions of hypomineralization. External hypoplasia is caused by a systemic disruption of the ameloblasts during matrix formation and is not usually accompanied by internal hypomineralization (Suckling 1989; Suga 1992). External hypoplasias are evidenced by "...malformation of histological structure (the shape and arrangement of the enamel rods) and the surface contour and thickness of the enamel..." (Suga 1992: 288). This type of enamel malformation is most popular with biological anthropology studies because is can be observed macroscopically as lines or pits along the enamel surface.

Enamel hypoplasias can be a result of hereditary factors, localized trauma, or systemic metabolic stress (Sharawy and Yeager 1991; Suckling 1989). Localized trauma results in defects that only impact adjacent teeth or even a single tooth (Shafer, et al. 1983; Skinner and Hung 1989; Ravn 1975). Systemic metabolic stress usually affects multiple teeth which are undergoing development at that time (Sharawy and Yeager 1991). Environmentally induced hypoplasia can be correlated to the stage of enamel development at the time of incidence, as evidenced in Suga's (1992) studies of calcium or magnesium deprived diets of rats.

Biological anthropology research utilizes hard tissue development chronologies that provide correlations for age at time of enamel defect onset. Hypoplasia studies in human communities suggest changes in dental health

in populations moving from hunting/gathering subsistence to an agricultural based society (Goodman et al. 1984c; Goodman and Rose 1990; Sullivan 1986; Ubelaker 1984). The utility of dental studies to reflect nutritional or disease stress on a population lies in the high mineralization of dental tissue, which is not subject to secondary change (Suga 1992). In contrast, Harris lines, or growth arrest lines in the skeletal tissue, which may mark similar events, are subject to reabsorption. Problems in matrix formation, resulting in enamel hypoplasias, are observable on a non-invasive scale, which are useful for living populations and skeletal populations with destructive restrictions.

CHAPTER IV

LITERATURE REVIEW

Enamel Hypoplasia Research

Kreshover (1960) describes the first hundred years of tooth defect studies as a shift from academic curiosity in isolated etiology theories to a recognition of and emphasis on the wide array of chemical, physical, and biological factors attributed to defects of the enamel. He quotes G.V. Black's interpretation which recognized, as early as 1924, that enamel defects are not caused by a short list of diseases but are rather the result of anything that extensively interferes with nutrition. Kreshover expands this definition to include local or systematic disturbances, depending on severity, which are generally nonspecific.

Enamel hypoplasia studies have a rich history in twentieth century dentistry and biological anthropology. In the 1940s, Schour produced a series of papers on tooth crown development and enamel formation. With his colleague Sarnat (1942, 1941), he mapped out tooth formation, the historical uses of enamel studies, and the attributes of chronologic enamel aplasia (or hypoplasia) to disease periods during growth, which were used to

trace occurrence onset and duration. Sarnat and Schour also suggested that the pathways to enamel hypoplasia were too diverse to be attributed to a single cause. Previous researchers had been studying enamel hypoplasia in relation to particular diseases (e.g. Hutchinson's studies of congenital syphilis), while Sarnat and Schour gathered all the data into more general categories of disease or environmental disturbances (e.g. fluorosis). They looked for correlations with specific disease histories in the patients studied, but found that in 50% of the cases absolutely no etiological factor could be identified.

Schour and Massler (1945) conducted a number of experiments to isolate nutritional impact on multiple oral structures; finding the enamel structures to be sensitive to changes in calcium, vitamins A, C, and D, and fluorine levels. They also made note of enamel's propensity to a "kymographic biological record of the nutritional status" (Schour and Massler 1945: 714, 1027). Enamel (as well as dentin) was found to be uninfluenced by nutritional processes after its formation and is therefore a permanent record of past nutritional status. Kreshover (1944) led the way in experiments to determine the range of factors that would disrupt enamel formation enough to cause hypoplasia. As part of his dissertation research, Kreshover infected rats and guinea pigs with tubercle bacilli in order to extrapolate associated changes in human enamel from disease pathways.

While Kreshover (1960) summarized the non-human studies, clinical researchers, such as Via and Churchill (1959), were continuing human studies focusing on the developmental factors contributing to enamel defects in the deciduous dentition. Their research attempted to draw correlations between enamel hypoplasia occurrence, cerebral disorders, and abnormal factors of development and birth (e.g. maternal diabetes, prematurity). Kreshover interprets some of these correlations as byproducts of nutritional deprivation which may injure tissue development. The unyielding nature of enamel formation would result in the deficiencies being permanently recorded in tooth structure in a way that similar defects in bone would not, because of the resorptive and remodeling nature of skeletal tissue.

Goodman and Rose (1990) summarize many of these and other historical developments in enamel hypoplasia studies. They also note that prior to 1960, two assumptions, deriving mainly from Sarnat and Schour's work (1942, 1941), had a permeating effect on the research of this generation. The "uniformitarian" approach assumed that (a) "...teeth are universally susceptible to enamel defects," and (b) the "...chronological pattern of enamel defects was due to a universal pattern of susceptibility," (Goodman and Rose 1990: 79). The first assumption led to textbooks proclaiming the idea that all teeth growing at the time of insult would exhibit equal reduced lines of enamel. The second assumption influenced researchers to treat all

populations as having the same potential for enamel hypoplasia as the research populations. This led to the homogenizing of the Chicago results (Sarnat and Schour 1942) in which enamel hypoplasias mainly occurred during the first year of life. This study was extrapolated to the population at large, and enamel hypoplasia was considered to be mainly prevalent prior to age one. Goodman and Rose (1990) note that these research blinders hindered the discovery of disruption patterns and prevented studies in population variations of stress.

In the 1960s, public health surveys were undertaken to track oral health in third world countries. Sweeney and Guzman (1966) surveyed children in highland Guatemala to provide baseline data for protein supplementation programs. Their dental survey concerned itself with the occurrence of enamel hypoplasia as a separate entity from the relationship to decaying or caried teeth. Their observations on three different villages also produced a glaring example of discrepancies between incidences of enamel hypoplasia between villages. While the authors and other researchers commonly attributed the hypoplastic line on deciduous teeth as being associated with the trauma of birth, Sweeney and Guzman proposed other types of etiology. They drew a comparison between the dental defects and Harris lines (stages of arrested growth in bone). Sweeney and Guzman attempted to explore the multiple influences possible (e.g. bottle feeding,

maternal infection, social or genetic predispositions, infections or fevers) in so large an affected portion of the population (close to 50% in the highest impacted village). Sweeney and colleagues (1969) pursued this research but could not find significant correlations to the presence of hypoplastic lines and infections, etc. around the time of birth. It should also be noted that these surveys are merely presence/absence studies and the linear enamel hypoplasia location is not quantified. Goodman and Rose (1990) credit Sweeney with being one of the first to draw correlations between linear enamel hypoplasia development and factors affecting growth such as nutritional status and infectious disease.

Along similar lines, Massler and colleagues (Johnson, et al. 1965), attempted to trace deciduous enamel defects to genetic health problems and gestational developing problems. Enamel hypoplasia rates in 'mongoloid' (trisomy 21) children were found to occur in higher percentages than comparative samples of non-mongoloid children. This study represents another attempt to use dental defect rates as a method of measuring particular disease or maturational abnormalities.

While the next generation saw diversification in studies of enamel defects by dentists, it also brought the application of dental methodologies to archaeological populations. Swärdstedt (1966) made use of Massler's work on tooth formation to develop a simplified chart of enamel mineralization in six

month growth increments. With disclaimers about crown height variability between populations, Swärstedt proceeded to take measurements from the apical border of the cemento-enamel junction to the middle of the enamel hypoplasia. Swärstedt became the first researcher to quantify the age at occurrence of hypoplastic defects. Additionally, he made status comparisons between groups within a single population and found the lower "slave" social class to suffer a higher incident of enamel hypoplasia defects.

Further institutionalization of enamel hypoplasia studies is evidenced by the section afforded it in Pindborg's (1970) Pathology of Dental Hard Tissues. Pindborg lamented the lack of scientific rigor in population studies noting enamel hypoplasia. He chastised researchers for not separating primary and secondary dentition data, not listing diagnostic criteria, and lumping hypoplasia and enamel opacities into a single category when their etiology is different, all of which make cross-comparisons difficult. Pindborg provided Massler et al.'s (1941) schematic for tooth development and Sarnat and Schour's (1942) results from the Chicago study; reinforcing the idea that most defects occur before one year of age. Also of note is that hypoplasia etiology at this point was still unknown. Pindborg discussed a number of theories, including the interruption in matrix formation, but this was not yet portrayed as the pathway of hypoplastic defects.

Contemporary population studies continued in all areas of the globe to

document the "...commonness of linear enamel hypoplasia in children in malnourished communities all over the world" (Jelliffe and Jelliffe 1971:839). In a letter to the editor of the *American Journal of Clinic Nutrition*, Jelliffe and Jelliffe, in agreement with Sweeney and colleagues as to nutritional agency, cited multiple observations that linear enamel defects are common in Asia, Fiji, rural Jamaica, Haiti, and Panama. Infante (1974) picked up the call for investigations of the nutritional influences of enamel defects in hopes of preventing more serious dental problems. He cited an example from the United States (Apache tribe) in hopes of turning the spotlight on underprivileged groups in developed nations. Enwonwu (1973) demonstrates high hypoplasia frequency in children of low socioeconomic class in Nigeria and makes connections between poor maternal nutrition and deciduous dentition defects. Research in Uganda (Moller, et al. 1972) focused on public health concerns of fluoridated water and the accompanying drop in dental problems of all varieties. While enamel hypoplasia rates were not high in this population (no more than 11.4% in permanent teeth), it is of note that developmental defects had become such a standard observation that even in populations with low rates it was still considered necessary to gather data on the trait.

Participating in the same public health program as Sweeney and Guzman (1966), Infante and Gillespie's (1974) research in rural Guatemala

attempted to interpret the distribution of the defect. The results remained elusive, with no sex differences in expression, no likelihood of siblings developing lesions (familial factors), and no reduction in occurrence if the mother and child were receiving protein supplements. The main recommendation of the authors was for further exploration of weanling diarrhea as a potential factor because children born in the later part of the year, when weanling diarrhea rates are at their lowest, have lower rates of enamel hypoplasia manifestation.

Use of a contemporarily documented skeletal collection by El-Najjar and colleagues (1978) noted frequency differences in hypoplasias rates by racial group in the Hammon-Todd series from Cleveland. Goodman (1988) revisited this material to look at hypoplasias in light of Sarnat and Scour's (1942, 1941) Chicago studies, pointing out the non-universality of their results.

By the 1980s, archaeological samples were the focus of increased interest. Cook directing a number of studies on cemeteries from the lower Illinois River valley. One particular paper documented the only prehistoric recorded case of *amelogenesis imperfecta*, or hereditary enamel hypoplasia (Cook 1980). Found in a six year old, this individual from the Schild Cemetery (A.D. 1100) exhibited reduced enamel on both the deciduous and permanent dentition. In similar populations Cook and Buikstra (1979) used

enamel defects as "...an indirect measure of the pressures associated with increased reliance on agriculture during the Woodland period," (p. 658). Attempting to compare nutritional health, the authors used a number of Middle and Late Woodland skeletal samples to test hypotheses of stress between the groups. This sample population was further used by Cook (1984) to test a wider array of observations using skeletal health as a window to the alterations in food production. Enamel hypoplasia had become one of a wider array of skeletal indicators to evaluate population changes and their corresponding stresses.

Concurrently, in the central Illinois River valley, studies were being conducted on the dental material of the Dickson Mounds collection. Rose and colleagues began looking at dentition in the Mississippian populations in order to use adult samples to measure childhood stress and determine the different patterns of stress in archaeological populations. Focus on enamel hypoplasia and pathological, or Wilson bands, proved that the prehistoric samples showed later stressful episodes than Sarnat and Schour's (1941) Chicago sample (Rose 1977; Rose, et al. 1978).

Goodman, Armelagos, and Rose (1980) analyzed dentition from the Dickson populations spanning A.D. 950 through A.D. 1300. Following Swärstedt's (1966) research example, they took measurements of enamel hypoplasias from the cemento-enamel junction in order to reveal age at

occurrence of defect. Raw measurement numbers were converted to age at onset in six month increments, using Swärdstedt's revisions of Massler, et al. (1941). While this research found that enamel disruptions increased over time, this study is more important for the recommendations it made for standardization in methodologies. The authors called for repeatable measurements and standard graphs for reference tooth development; also noted were recommendations for teeth that are most "susceptible" to defect development (mandibular canines and maxillary central incisors) which would be timesaving for limiting future studies.

Along with permanent dentition studies at Dickson Mounds, Blakey began taking inventory of the deciduous teeth in order to examine stress both pre- and neo-natal in order to determine the amounts of maternal stress being transferred to the infant (Blakey and Armelagos 1985; Blakey 1981). The Dickson Mounds data proved to be fertile ground for testing numerous models of enamel hypoplasia development and distribution rates (Goodman and Armelagos 1985a, 1985b; Goodman, et al. 1984a, 1984b) while also being presented as a standard component of pathological analysis (Goodman, et al. 1984c). All of which lead to the recognition in the anthropological community of dental defects as unique indicators of population health.

Tooth Development Debates

Converting enamel hypoplasia measurements into meaningful age representations is an important but problematic part of dental studies. Measurements need to be converted to an age-at-occurrence in order to make individual teeth comparable and to discuss growth disruptions at the personal level. Goodman, et al. (1980) rely on the chart of tooth development provided by Swärstedt (1966), modified from Massler, et al. (1941) (Appendix D).

Garn , et al. (1959), however, brought attention to the tooth developmental charts being used by Schour and researchers. Garn and colleagues claim that the Schour and Massler charts are adapted from earlier studies, which had extremely small sample sizes and that the variability in tooth formation is actually much larger than represented. Also noted is the need for sample groups that are statistically large enough to have adequate representations of all age categories, since even their study (Garn, et al. 1959) is under represented in some age groups (i.e. less than 100 individuals).

For this study, the Swärdstedt (1966) chart, instead of more recent enamel formation studies, is used because the Massler and co-workers' (1941) study relied on direct enamel observation. More recent work relies mainly on radiographic standards which reflect calcification and not matrix

development. Since the maturation stage occurs at an unknown length of time after matrix development, the early chart is still the most useful for identifying initial crown formation.

The location of the hypoplasia is measured in relation to the cemento-enamel junction and converted to an age based on the developmental chart. Tooth growth from this chart appears to be based on a developmental curve, with unequal amounts of enamel produced in different half year periods.

Goodman and colleagues (1980) are not clear as to how ages are translated for the increments without direct millimeter notations. Later articles on the Dickson Mound collection refer to dividing the teeth into equal increments for each half year period represented. Goodman and Armelagos (1985b: 482) note that the maxillary central incisor is divided into, "nine zones of equal width corresponding to nine half-year developmental periods." Other teeth are divided similarly based on their time of growth. Goodman and various colleagues (1989, 1985a, 1984a) do not specify if the increments are equal widths, but cite the 1980 study as containing the referential chart. The 1980 article makes no specific note of how the enamel surface was divided other than referring to Swärdstedt's chart. Goodman and Rose (1991) note that while the 1980 study proposed a weighted interpretation of enamel development, later work (Goodman 1988; Goodman, et al. 1987) has adopted the constant velocity approach (or "null hypothesis")

and divides enamel into equal increments.

Swärdstedt's chart assumes that enamel formation happens at a variable velocity. This non-linear approach to tooth growth accounts for different rates of enamel formation at different ages. A linear, or constant velocity approach, averages the different rates of enamel formation to produce a smoothed developmental pattern.

Other researchers, referencing the Goodman, et al. (1980) reprint of Swärdstedt's chart, make no further notations of linear versus non-linear development of tooth formation and, consequently, enamel hypoplasia measurements (Hutchinson and Larsen 1988; Lanphear 1990; Larsen and Hutchinson 1992; Mack and Coppa 1992; Moggi-Cecchi, et al. 1994; Rathbun 1987; Rose, et al. 1985; Van Gerven, et al. 1990). Yamamota (1992) and Schulz and McHenry (1975) use Massler, et al.'s (1941) chronology, making no reference to equal or unequal age divisions. Wood's (1996) research uses the constant velocity approach explained in Goodman and Rose (1990).

Berti and Mahaney (1992) have examined linear enamel hypoplasia chronologies for confidence intervals of enamelization schedules. Critiquing the chart method of converting ages, the authors tested constructed chronologies of sample and tooth-specific methods (in which population specific adjustments are made for tooth growth and height), finding them confusing and of questionable value. Berti and Mahaney's remaining

recommendation is to publish measurements for potential reevaluation.

CHAPTER V

METHODOLOGY

Research Problem

The purpose of this study is to analyze linear enamel hypoplasia rates in an historic skeletal sample from a cemetery in Milwaukee, Wisconsin, and to compare these results with those from 15 other populations from all over the world, ranging in time from 2500 BC to the present. For comparison purposes, enamel hypoplasia rates will be classified by total number of incidences and by age at occurrence.

The Milwaukee County Institutional Grounds cemetery (1882-1925) was excavated between September 1991 and November 1992 in a salvage operation by Great Lakes Archaeological Research Center, Inc. (Richards and Kastell 1993). The recovered cemetery population consists of 1649 individuals, of which 588 are subadults. While all of the MCIG skeletal and dental materials are in good to excellent condition, this study focuses on the adults in the collection.

Sample Population

At the outset of this project, the collection could be divided into two main subsamples: individuals with corresponding burial records and unidentified individuals. The first subsample chosen for inclusion in this study were the individuals whose archaeological lot numbers had been matched with burial records (Richards and Kastell 1993). The second portion of the study subsample contained a like number of individuals drawn from the larger, unidentified portion of the cemetery. Unfortunately, further research indicated that only a few individuals had actually been matched correctly to burial records and that most of the population remains unidentified (Sullivan, personal communication). The entire sample numbers 140 individuals (979 observable teeth), none of which, at this date, have accompanying records.

The sample was derived from individuals with two or more accompanying anterior teeth. The presence or absence of twelve teeth were recorded: mandibulary and maxillary incisors and canines (left and right). Anterior dentition was the focus of this study because previous literature has noted that anterior teeth are more sensitive to stressors than posterior teeth (El-Najjar et al. 1978; Goodman and Armelagos 1985a, 1985b; Goodman et al. 1987; Patterson 1984; Sullivan 1986). Among anterior teeth, maxillary central

incisors and mandibular canines are reported as the most frequent indicators of hypoplasias (Cutress and Suckling 1982; El-Najjar, et al. 1978; Goodman 1988; Goodman and Armelagos 1985a; Sullivan 1986). All incisors and canines were included in this study in an effort to test frequencies of specific anterior tooth sensitivity to decreased enamel thickness. Additionally, the labial surface of teeth has been noted to be more sensitive to developing LEH than the lingual surface. Sullivan (1986) notes that in tooth development disruption there is a threshold effect that allows LEH to occur on the labial/buccal surfaces while not always appearing lingually. For the purpose of this study, if a defect occurred on the labial surface it was examined and scored for LEH.

Tooth Selection

Studies of the Dickson Mound skeletal population in the central Illinois River valley by Goodman and Armelagos (1985a, 1985b) have found that the presence of hypoplasia is most common on the middle third of the tooth crown and of low frequency in the earliest period of development (Goodman 1988). The MCIG collection has some attrition in many of the teeth, but those with wear extending beyond the incisal/occlusal third were not included in this study (Ensor and Irish 1995; Wood 1996). This may result in bias when determining the chronological frequencies of hypoplasia,

but since previous studies have attested to minimal incidence of hypoplasias on the first third of crown development (supported by the author's observations), teeth exhibiting small amounts of wear were included in this study.

This method is different than the one that Knick (1981) used while conducting a study on the Schild skeletal population (Mississippian, circa A.D. 1065) in the lower Illinois valley. He used only individuals twenty to thirty years old in order to have minimal enamel wear. The demographic nature of almshouse burial populations does not permit the construction of large sample sizes in the young adult range, making exclusion of the remainder of the population problematic if the researcher is interested in assembling a collection large enough to produce statistically valid results.

Teeth not included in this sample were ones that (a) had a great deal of enamel chipping, (b) exhibited a prohibitive amount of tarter or calculus build-up, (c) exhibited excessive staining, (d) were missing more than one-third of the crown due to attrition, or (e) were covered by metal capping (e.g. gold). In order to prevent damage which might impair future dental research on this collection, the author elected not to clean teeth of calculus build-up, even though this would have provided a larger sample of teeth from the individuals studied.

Classification System

Hypoplasias observed on teeth were classified as either linear band/line, pit, or mottled areas. Bands/lines are linear depressions in the tooth crown resulting from a decreased layer of enamel during formation. A pit is a single, circular decrease in the enamel which is not linear or symmetrical across the tooth. Mottled areas are groups of pits that also are not linear or symmetrical. A series of pits in a linear plane, for the purposes of this study, were classified as a band/line since they were symmetrical across the lingual plane (Wood 1996). The definition of a hypoplasia defect from the Commission of Oral Health, Research and Epidemiology (1982) has been used in this study. Hypoplastic defects are defined as surface defects of the enamel with associated reduced enamel thickness that can be:

> (a) shallow or deep pits or rows of pits arranged in a linear fashion across the tooth surface or generally distributed over the whole or part of the enamel surface;
> (b) the defective enamel may occur as small or large, wide or narrow grooves;
> (c) in some instances there may be partial or complete absence of enamel over small or considerable areas of dentine (Commission of Oral Health, Research and Epidemiology 1982: 160).

Bands/lines are the only observations that are used in the remainder of this study because of their tendency to represent discrete and pervasive growth disruptions (Ensor and Irish 1995; Goodman, et al. 1987).

Linear bands and lines have often been classified as separate observations in the literature (Corruccini, et al. 1985; Moggi-Cecchi, et al. 1994; Sarnat and Schour 1941), but the distinctions are not clearly differentiated or well quantified. Bands, grooves, or major growth arrests are wide marks which are considered broad enough to be measured with calipers. However, if the width of the hypoplasia cannot be easily measured it has been referred to as a line, linear enamel hypoplasia, or narrow mark. The aid of a binocular microscope to confirm identification of hypoplasias has proved useful (Larsen and Hutchinson 1992), but it has also blurred the distinction between "measurable" and "non-measurable" defects as defined in previous studies. Since the distinction between band and line seems to be based more on the sharpness of the eyesight of a particular researcher than any non-arbitrary criteria, in this study the two terms have been collapsed into one category, that of band/line. The present study is concerned with the relationship between and frequencies of insults, rather than duration of the causal stress, the data for width of hypoplasia was not collected.

Recordation

Observations of the labial surface were made by gross visual examination but confirmed by the use of a binocular microscope (5x). Only hypoplastic disturbances that could be felt by running a dental probe along

the surface of the tooth from the crown to cemento-enamel junction (horizontally) were scored. This follows the Dental Commission's recommendations that tooth surfaces should be, "inspected visually, and defective areas tactilely explored with a probe to determine abnormalities of surface contour" (Commission on Oral Health, Research and Epidemiology 1982: 160). Enamel hypoplasias were measured under the microscope with a Helios thin-tipped caliper calibrated to 0.05 mm. The measurements were taken from the cemento-enamel junction (CEJ) to the center of the decreased thickness in the enamel. These measurements of enamel hypoplasia allowed for multiple incidents of insult on a single tooth to be recorded. The binocular microscope was of significant help in confirming the presence/absence of an enamel hypoplasia.

The method proported by Massler and colleagues (1941), updated by Swärdstedt (1966), and modified by Goodman, et al. (1980) was used for converting recorded measurements to age at time of incident (which is useful for comparisons to other studies). The particular tooth is divided into equal intervals that correspond to half-year increments of development (0.5 to 1.0 years, 1.0 to 1.5 years, through 6.0 to 6.5 years). The assumptions inherent in this approach are that crown dimensions and timing of crown formation is standard both temporally and regionally. While those assumptions are not accurate, the primary focus at present is to relatively measure hypoplasia

occurrence within this population and between the other samples.

Because of the long period of permanent tooth formation, observations from incisors and canines cover a developmental time frame of birth to 6.5 years. The frequency of hypoplastic events per tooth, the age of occurrence they represent, and the number of incidents per individual were calculated. The frequencies were then used for comparison between tooth types and other skeletal samples.

Data Analysis

The data were collected and entered into a multi-relational database using Lotus Approach 3.01 (1994). Chi-square analyses (Pearson's and Yates corrected) were conducted using SPSS (1996).

Analytical Methods

This study will examine the MCIG data in light of both Swärstedt's (1966) and Goodman et al.'s (1987) modifications to chronological divisions of hypoplasia measurements in order to reveal any differences in interpreting age-at-occurrence data. For the ages calculated using the non-linear development chart, a cubic spline (Mathcad 1995) has been calculated to fill in the missing millimeter measurement in relation to half-year increments. The final measurement conversions are listed in Appendix E. Goodman and

Table 1

Regression Equations for Estimations of Age at LEH Formation

Tooth	Formulae
Maxillary Teeth	
I1	Age = - (0.454 × Ht) + 4.5
I2	Age = - (0.402 × Ht) + 4.5
C	Age = - (0.625 × Ht) + 6.0
Mandibular Teeth	
I1	Age = - (0.460 × Ht) + 4.0
I2	Age = - (0.417 × Ht) + 4.0
C	Age = - (0.588 × Ht) + 6.5

Age = age in years; Ht = distance of the LEH in mm from CEJ.

Regression equations are based on mean crown heights of Swärdstedt (1966) and the crown formation standard of Massler, et al. (1941) (after Goodman and Rose 1990:98).

Rose (1990) have published the regression equations for converting height of enamel defect on the crown to an age at occurrence. The equations used for the age conversions in the linear development system are listed in Table 1. The raw data for individual tooth measurements are curated with the collection.

A variety of methodologies for counting enamel hypoplasias have been used in order to determine systematic stress episodes. Goodman, et al. (1980) introduced a scoring method that graded an age zone as positive for

enamel hypoplasia if at least two teeth exhibited reduced enamel in that zone (labeled as matching hypoplasias) (e.g. Goodman, et al. 1984a; Powell 1988; Rathbun 1987). This method of scoring age zones rather than individual hypoplastic events has not been applied by all archaeological researchers. Corruccini and co-workers (1985) compare hypoplasia data as individual lines versus individual specimens, in which age range is scored only once if multiple hypoplasia lines occur. Lanphear (1990) uses a more simplistic rating system which involves scoring each hypoplastic event individually . The variety of approaches to tallying hypoplasia, in hopes of illuminating stress episodes, make cross-population comparisons difficult.

Results will be tallied according to Lanphear's (1990) method of recording single hypoplastic events. This methods leaves out the arbitrary nature of "matching" hypoplastic lines between teeth, the results of which can vary depending on how the age ranges on a tooth are constructed.

Comparison Populations

A number of enamel hypoplasia studies have been selected for comparison with the MCIG cemetery data. Data has been gathered for comparison of individual frequencies of LEH occurrence and of frequency peaks by tooth type in six month age categories.

CHAPTER VI

RESULTS

MCIG Cemetery

Since other studies have found no statistically significant difference between the likelihood of males and females developing enamel hypoplasia (Goodman, et al. 1987; Lanphear 1990), and because of the under representation of females in this sample (n = 7 or 6% of the sex determined sample), the male and female data are pooled for analysis. Of 140 individuals, 1671 teeth were examined, 989 teeth were observable, and 288 exhibited at least one hypoplasia event.

In the sample studied, the average number of observable teeth per individual, out of a possible twelve, is 7.06. The 58 individuals that did not have any enamel hypoplasia accounted for 415 of the observable teeth (42.0%), averaging 7.16 teeth per individual. The 82 individuals that had at least one enamel hypoplasia accounted for 574 of the observable teeth (58.0%) and an average of 7.00 teeth per individual were present. Fifty-six individuals had more that one hypoplasia observed on the available teeth.

Presence or Absence of LEH

Of 140 individuals included in this study, 82 individuals (58.6%) had at least one hypoplasia. When changing the analysis to the level of the tooth, 288 teeth in a sample of 990 observable teeth (29.1%) had at least one incidence of LEH. When all anterior teeth are examined (Table 2), the mandibular left canine exhibits the highest rate of LEH of 42.4%, which contrasts with a low of 13.7%, for the maxillary right lateral incisor.

Other comparisons of the tooth rates of LEH can be made using individuals in the MCIG sample. The average frequency of LEH for the maxilla is 25.8% (123 teeth with at least one incidence of LEH out of 476 observable teeth) and the mandible is 32.1% (165 teeth with LEH, 514 total teeth). When the teeth are separated by side, the left (mandible and maxilla combined) has 29.6 % of teeth with LEH (144 out of 486 total teeth) while the right side is at 28.6 % (144 teeth with LEH, 504 total teeth). When the data is broken down by tooth type the central incisor has an LEH frequency of 25.8% (77 teeth with LEH, 298 total teeth), lateral incisor at 23.9% (77 teeth with LEH, 322 total teeth), and the canine at 43.6% (134 teeth with LEH, 307 total teeth).

Table 2

Frequency of LEH by Tooth in the MCIG Sample: Anterior Teeth

Tooth		Total number of teeth	Number with at least one hypoplasia	
Maxillary				
Left	Central Incisor	75	22	(29.3%)
	Lateral Incisor	73	14	(19.2%)
	Canine	84	28	(33.3%)
Right	Central Incisor	79	19	(24.1%)
	Lateral Incisor	73	10	(13.7%)
	Canine	92	30	(32.6%)
Mandibular				
Left	Central Incisor	77	17	(22.1%)
	Lateral Incisor	85	24	(28.2%)
	Canine	92	39	(42.4%)
Right	Central Incisor	67	19	(28.4%)
	Lateral Incisor	91	29	(31.9%)
	Canine	102	37	(36.3%)
Totals		990	288	

Age at Occurrence of LEH

When age at occurrence is calculated for individual hypoplastic events two methods can be used to translate the data: a linear or a non-linear growth chart. Using the non-linear growth chart, LEH on the mandibular canine peaks between 3 and 3.5 years of age (Tables 3 and 4, Figures 8 and 9). LEH on the maxillary central incisors peaks between 2 and 3 years of age. When the linear growth chart is used, the peaks become further separated. The mandibular canine peaks between 4 and 5 years of age while the maxillary central incisor peaks between 2 and 2.5 years of age (Tables 5 and 6, Figures 10 and 11). The linear and non-linear data at age of LEH incident, for maxillary central incisors and mandibular canines, are graphed together in Figure 12.

Of note is that the mandibular canine and maxillary central incisor peaks are separated by one and a half years, which is consistent with previous research. Differing LEH peak, by tooth type, has been documented in other collections (Goodman 1988; Goodman and Armelagos 1985a; Lanphear 1990), and has been attributed to the intertooth difference in hypoplasia formation. The LEH results of peaks by tooth type, based on the non-linear growth conversions, are closest to the results Lanphear (1990) found in collection from the Monroe County Poorhouse.

Table 3

Frequency of LEH per Developmental Age in the MCIG Sample: Maxillary Teeth, Non-linear Growth Conversions

	Maxillary Left			Maxillary Right		
Developmental age (years)	Canine	Lateral Incisor	Central Incisor	Central Incisor	Lateral Incisor	Canine
0-0.5	0	---	0	0	---	0
0.5-1	0	---	0	0	---	0
1-1.5	1	1	0	0	1	3
1.5-2	2	1	2	0	1	1
2-2.5	2	2	9	9	1	5
2.5-3	5	4	13	9	2	5
3-3.5	7	4	3	4	5	6
3.5-4	5	3	2	1	0	12
4-4.5	11	1	0	0	1	10
4.5-5	4	---	---	---	---	3
5-5.5	2	---	---	---	---	1
5.5-6	1	---	---	---	---	1
Totals	40	16	29	23	11	47

When the LEH data for all teeth are summarized, the age at occurrence rates peak between 2 and 3 years of age (Figure 13). On a non-linear growth chart, the LEH data peak between 2 and 2.5 years of age. On a linear growth chart,

Table 4

Frequency of LEH per Developmental Age in the MCIG Sample: Mandibular Teeth, Non-linear Growth Conversions

	Mandibular Left			Mandibular Right		
Developmental age (years)	Canine	Lateral Incisor	Central Incisor	Central Incisor	Lateral Incisor	Canine
0-0.5	---	1	0	0	0	---
0.5-1	0	1	1	1	1	1
1-1.5	4	3	0	1	4	3
1.5-2	4	4	1	3	5	4
2-2.5	10	10	13	10	17	8
2.5-3	2	11	4	7	12	7
3-3.5	14	4	1	4	4	15
3.5-4	8	0	0	0	0	14
4-4.5	13	---	---	---	---	11
4.5-5	6	---	---	---	---	5
5-5.5	4	---	---	---	---	0
5.5-6	0	---	---	---	---	1
Totals	65	34	21	26	43	69

the peak is between 2.5 and 3 years of age. The use of differing age at occurrence conversions produces slightly varying overall results.

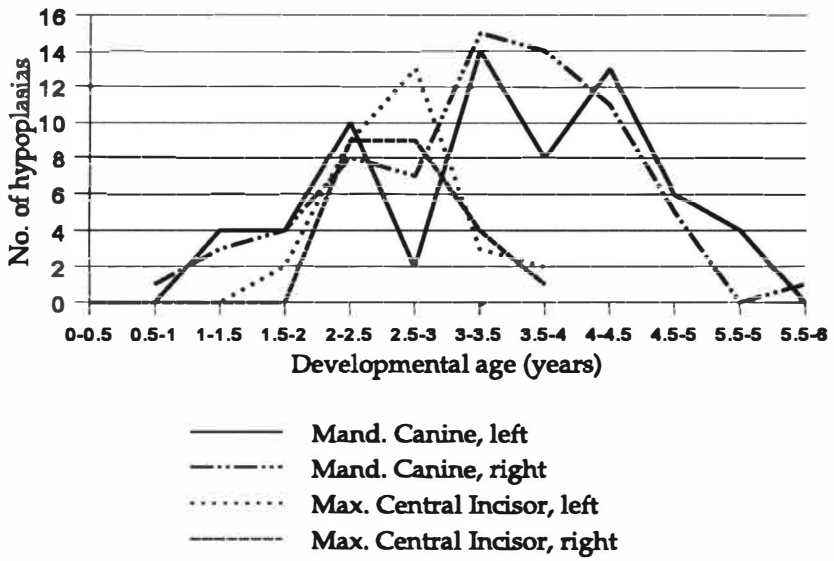


Figure 8. Frequency of LEH in the MCIG Sample Based on Non-Linear Growth Chart.

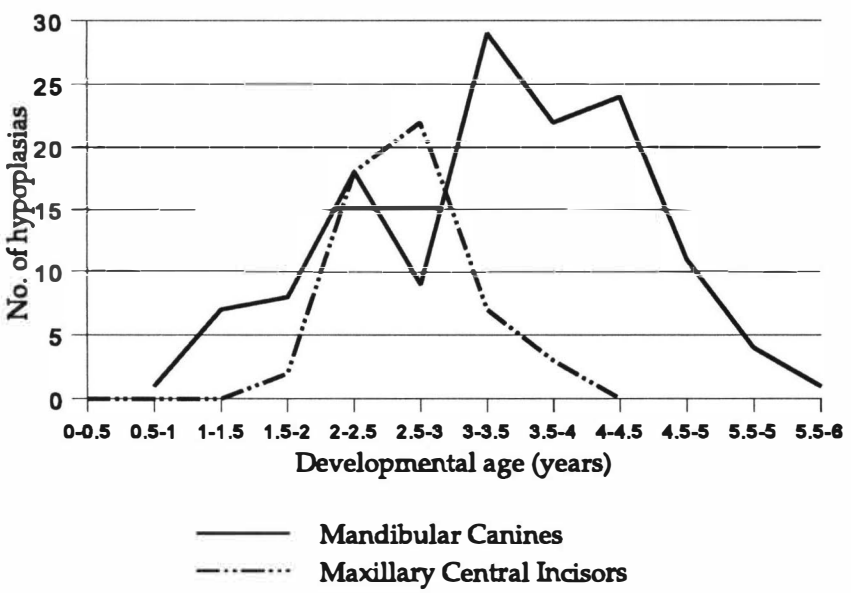


Figure 9. Frequency of LEH in the MCIG Sample Based on Non-Linear Growth Chart, Right and Left Combined.

Table 5

Frequency of LEH per Developmental Age in the MCIG Sample: Maxillary Teeth, Linear Growth Conversions

	Maxillary Left			Maxillary Right		
Developmental age (years)	Canine	Lateral Incisor	Central Incisor	Central Incisor	Lateral Incisor	Canine
0-0.5	0	---	0	0	---	0
0.5-1	0	---	0	0	---	1
1-1.5	2	1	2	0	2	2
1.5-2	3	2	6	4	0	6
2-2.5	2	2	9	10	3	2
2.5-3	5	5	7	4	4	7
3-3.5	6	2	3	4	1	7
3.5-4	4	4	2	1	1	7
4-4.5	11	0	0	0	0	10
4.5-5	4	---	---	---	---	3
5-5.5	2	---	---	---	---	1
5.5-6	1	---	---	---	---	1
Totals	40	16	29	23	11	47

Table 6

Frequency of LEH per Developmental Age in the MCIG Sample: Mandibular Teeth, Linear Growth Conversions

	Mandibular Left			Mandibular Right		
Developmental age (years)	Canine	Lateral Incisor	Central Incisor	Central Incisor	Lateral Incisor	Canine
0-0.5	---	0	1	0	0	---
0.5-1	0	1	1	1	0	0
1-1.5	2	2	0	1	3	1
1.5-2	3	4	1	2	2	4
2-2.5	3	8	10	7	18	3
2.5-3	8	15	8	11	15	6
3-3.5	4	4	0	4	5	8
3.5-4	10	0	0	0	0	14
4-4.5	12	---	---	---	---	16
4.5-5	15	---	---	---	---	12
5-5.5	8	---	---	---	---	4
5.5-6	0	---	---	---	---	1
Totals	65	34	21	26	43	69

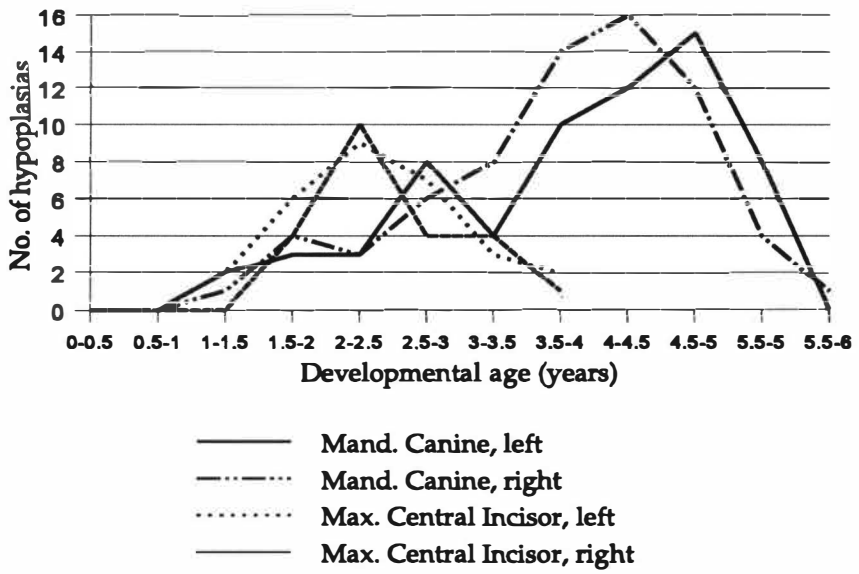


Figure 10. Frequency of LEH in the MCIG Sample Based on Linear Growth Chart.

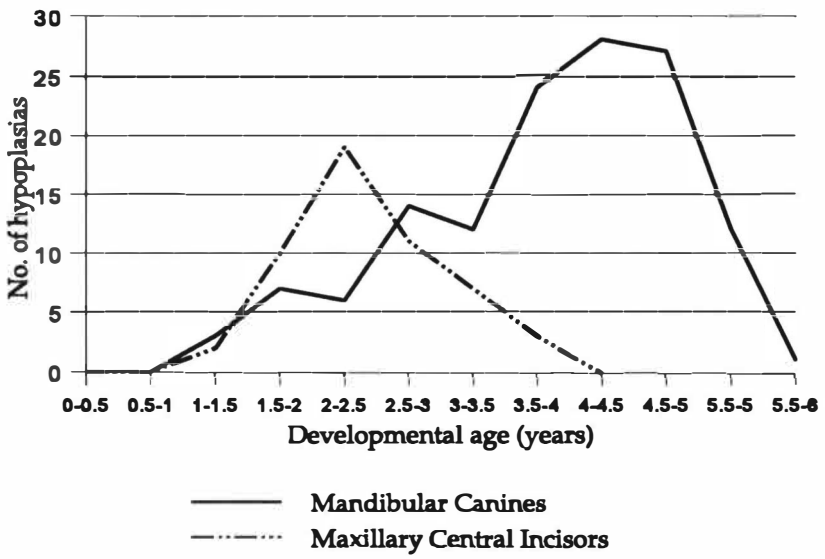


Figure 11. Frequency of LEH in the MCIG Sample, Based on Linear Growth Chart, Right and Left Combined.

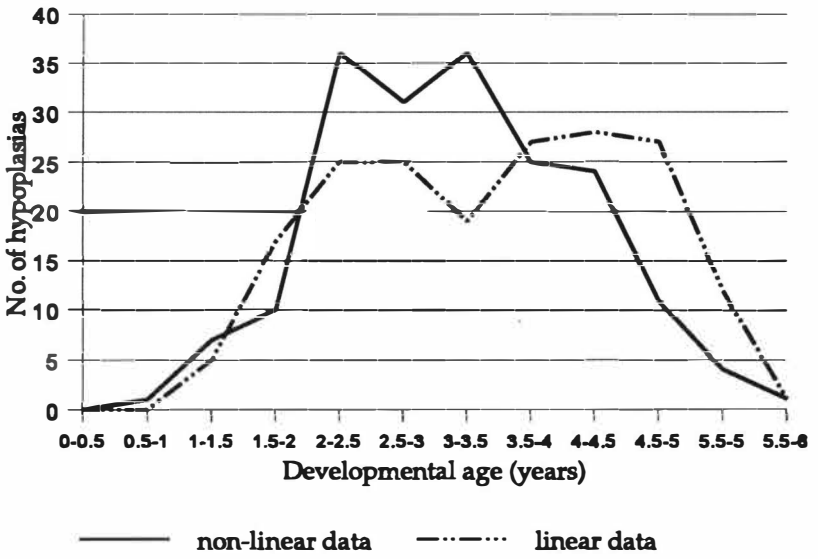


Figure 12. Frequency of LEH in the MCIG Sample, Based on Linear and Non-Linear Growth Charts, Mandibular Canines and Maxillary Central Incisors.

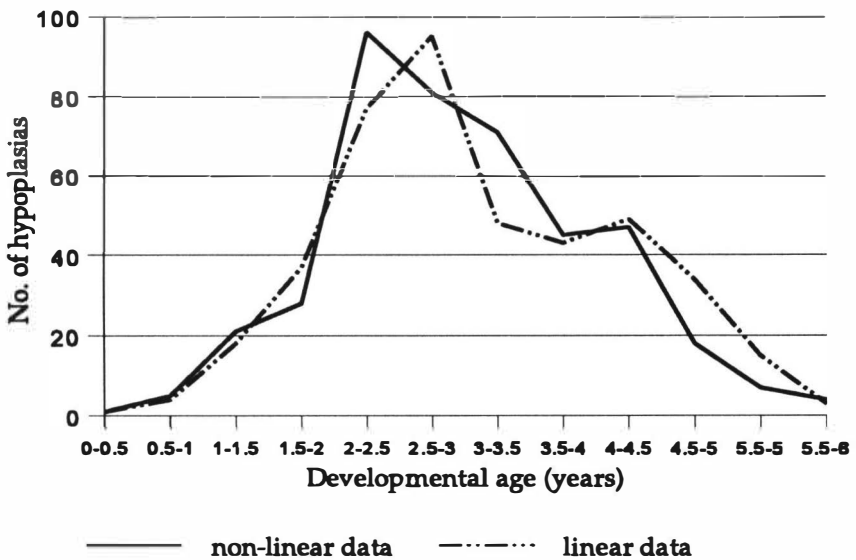


Figure 13. Frequency of LEH in the MCIG Sample, Based on Linear and Non-Linear Growth Charts, All Anterior Teeth.

Comparison Populations

Presence or Absence of LEH

Of the eight populations chosen for comparison with MCIG for presence/absence of LEH, the frequency of individuals with LEH ranges from 26.0% to 82.2% (Table 7). The Monroe County Poorhouse sample, the only other historic industrial population but of an earlier era, has a much higher rate of LEH at 73.0% than MCIG (Lanphear 1990). Based on the frequency, MCIG seems most comparable with two slave populations also exhibiting LEH in the 50% range; eighteenth century slaves from Virginia and Maryland at 56.0% (Kelley and Angel 1987) and the Newton Plantation at 54.5% (Corriccini et al. 1985).

Chi-square tests (Table 8) confirm that these two samples are not significantly different (when $p = 0.05$) from the MCIG population. Additionally, the population of slaves from Charleston, South Carolina approaches the significance level, with the Pearson's and Yates corrected chi-square test falling on either side of the 0.05 cutoff.

Age at Occurrence of LEH

The results of simple comparisons of peak age of LEH occurrence between the MCIG population and other studies reveals a similarity between

Table 7

Frequency of LEH in Comparison Populations and the MCIG Sample

Population/Site	Dates	Subsistence	Individuals			Sources
			No.	LEH	%	
Virginia and Maryland	19th century	Agric. Slaves	34	9	26.0%	(Kelley and Angel 1987)
Rural Mexico	20th century	Industrialists	300	140	46.7%	(Goodman et al. 1987)
Newton Plantation	AD 1660-1820	Agric. Slaves	103	56	54.5%	(Corruccini et al. 1985)
Virginia and Maryland	18th century	Slaves	25	14	56.0%	(Kelley and Angel 1987)
Milwaukee Co.	1882-1925	Industrialists	140	82	58.6%	(this study)
Monroe Co. Poorhouse	AD 1826-1863	Industrialists	160	117	73.0%	(Lanphear 1990)
38CH778, Charleston SC	AD 1840-1870	Agric. Slaves	31	24	77.4%	(Rathbun 1987)
Québec City	AD 1746-1747	Agriculturalist	44	35	79.5%	(Wood 1996)
1st African Baptist Church	AD 1823-1841	Agriculturalist	73	60	82.2%	(Blakey 1987)

LEH = Number of individuals with linear enamel hypoplasia.

(after Lanphear 1990:41)

Table 8

Chi-Squared Evalutation of Comparison Populations With Milwaukee County

Population/Site	Pearson's Chi-Squared	Yates Corrected Chi-Squared	Significance (Pearson's/Yates)	Significant to 0.05 level	Key
1st African Baptist Church	12.046	11.007	p = 0.001/0.001	significant	1
Virginia and Maryland, 19th c.	11.300	10.050	p = 0.001/0.002	significant	2
Monroe Co. Poorhouse	7.082	6.445	p = 0.008/0.011	significant	3
Québec City	6.361	5.487	p = 0.012/0.019	significant	4
Rural Mexico	5.412	4.946	p = 0.020/0.026	significant	5
38CH778, Charleston SC	3.826	3.068	p = 0.050/0.080	not significant*	6
Newton Plantation	0.427	0.273	p = 0.513/0.601	not significant	7
Virginia and Maryland, 18th c.	0.058	0.000	p = 0.810/0.984	not significant	2

* = significant by Pearson's but not Yates corrected chi squared.

1. Blakey 1987; 2. Kelley and Angel 1987; 3. Lanphear 1990; 4. Wood 1996; 5. Goodman, et al. 1987; 6. Rathbun 1987; 7. Corruccini, et al. 1987.

emerging industrial populations (Table 9). The maxillary central incisor and mandibular canine data from MCIG, regardless of growth velocity used, closely follow the results of studies on the Monroe County Poorhouse and the Hammon-Todd collection. The twentieth century samples, Chicago, Jordan, and Mexico, exhibit peak age at occurrence ranges between birth and three years of age. The North American industrial era samples overlap both the twentieth century and the agricultural populations, with peaks between 2 and 5 years of age. The samples from agriculturally based populations exhibit a peak age of occurrence between 2 and 6 years, while the peaks for hunting and gathering groups are between 3 and 6 years.

Suggestions that LEH represents the post weaning period, with all the accompanying exposures to disease and varying nutrition, are consistent with an observed decline in mean age at onset of weaning from prehistoric to modern populations. Specifically, this shift is evident in the changes in economic practices. The Milwaukee County sample, similar to other North American pre-industrial populations, falls between the agricultural and fully industrial groups.

Table 9

Peak Age at Stress of LEH Occurance in Comparison Populations and the MCIG Sample

Population/site	Period	Subsistance	Peak age at stress	Sources
Chicago	20th century	Industrialists	0-1.0	(Sarnat and Schour 1941)
Jordan	20th century	Agric. and nomadic	1.0-3.0	(Alcorn and Goodman 1985)
Rural Mexico	20th century	Agriculturalists	1.0-3.0	(Goodman, et al. 1987)
Milwaukee Co. (Linear growth)	AD 1882-1925	Industrialists	2-2.5 (I), 4-5 (C)	(this study)
Milwaukee Co. (Non-linear growth)	AD 1882-1925	Industrialists	2-3 (I), 3-3.5 (C)	(this study)
Monroe Co. Poorhouse	AD 1826-1863	Industrialists	2.5-3.0 (I), 3.5-4.0 (C)	(Lanphear 1990)
Hammon-Todd	AD 1852-1933	Industrialists	2.5-3.0 (I), 3.5-4.0 (C)	(Goodman 1988)
Dickson Mounds	Mid. Miss.	Agriculturalists	2.5-3.0	(Goodman et al. 1984a)

Table 9--Continued

Population/site	Period	Subsistance	Peak age at stress	Sources
Dickson Mounds	Pre-Miss.	H/G and trans.	3.0-3.5	(Goodman et al. 1984a)
Swedish	Medieval	Agriculturalist	2.0-4.0	(Swärtstedt 1966)
California	2500 BC - AD 1800	H/G and agriculturalists	4.0-6.0	(Schulz and McHenry 1975)

H/G = hunter/gathers, trans. = transitional hunter/gathers to agriculturalists, I = incisor data reported, C = canine data reported.

(after Lanphear 1990:40)

CHAPTER VII

CONCLUSIONS

The nature of this almshouse population precludes it from being fully representative of the poor in Milwaukee during the early eighteenth and nineteenth centuries. Avella (1987) and Conzen (1976) have documented women and children who were served by the community's social welfare programs, however, women are not being interred in the county cemetery at the same rate as men in the community. Some of the disproportion could be attributed to the male/female ratios in a developing community, such as a traditional imbalance toward males in migration. Since the sex ratio of this sample is biased in favor of males, any conclusions from this population do not reflect heavily on the status of childhood nutrition of women in this community or their community geographic origin.

The number of observable teeth for individuals with LEH and without LEH are essentially the same, so differential recovery of total numbers of teeth was not a factor in the analysis. Of all the anterior teeth surveyed, the mandibular canine provides the highest frequency of teeth with LEH per individual, followed by the maxillary canines. Since these teeth take the

longest amount of time to develop, it is not surprising that they incur LEH more often than teeth with shorter developmental spans. The next most sensitive teeth to LEH seem to be maxillary central incisors and mandibular lateral incisors.

Considering peak age at stress in the MCIG sample, the differing peaks by tooth type between the maxillary central incisors and the mandibular canines are consistent with those reported for other samples (Goodman 1988, Lanphear 1990). Explanations for intertooth differences in hypoplasia formation have not been adequately explored in the literature and it remains to be explained why canines are so much more sensitive at a later developmental age than incisors to developing LEH.

The largest problem in LEH studies seems to be the meaningful representation of LEH measurements as age ranges at time of incidence. Data collection of LEH measurements seems to be standard, but conversion of this data is fraught with inconsistencies. Some researches score gross numbers of LEH, while others use somewhat arbitrary threshold systems to determine when a group of LEH can be considered to represent a single incidence of major growth arrest. Some of these techniques become difficult to apply to populations of any advanced age ranges, where dental health is not always paramount, thus precluding individuals with attrition. A common standard translation of numbers of LEH incidence and conversion

to age at incidence ranges would be useful. The MCIG data illustrates how using various conversion methods for age ranges can separate the peaks of LEH incident from 2 to 3.5 years to as large as 2 to 5 years.

The MCIG sample does not provide any information contrary to earlier studies concerning the progression of peak frequencies by age range. MCIG falls comfortably within the industrial population ranges. When comparing individuals in a population with LEH, MCIG seems to be in the mid-range of LEH frequencies at 58.6% and is similar to three agricultural slave populations: Charleston (Rathbun 1987), Newton Plantation (Corruccini, et al. 1985), and eighteenth century Maryland and Virginia sites (Kelley and Angel 1987).

The combination of peak age at stress and frequency of LEH data suggests that while members of the MCIG population were affected by developmental disturbances during childhood, they were not affected as often as the residents of the Monroe County Poorhouse in Rochester, New York, early in the nineteenth century. While the peak age of the stress could be extrapolated to indicate similar weaning ages for the MCIG and the Monroe County Poorhouse, with the concurrent potential for disease exposure and nutritional inadequacies, frequencies of LEH in the populations are not comparable. The result of comparisons between the MCIG sample and other populations, for frequency of LEH, place MCIG in the mid-range of

low socio-economic populations in the experience of childhood stress.

According to Lanphear (1988), in 1850 the Monroe County Poorhouse population mainly consisted of immigrants from Ireland, Germany and England. In contrast, the waves of immigrants in the later part of the eighteenth and early nineteenth centuries were most from southern Europe (Nugent 1992). The difference in populations may be expressed by the nature of migrations in the last two centuries. Lanphear (1988) notes that the majority of individual in the early-1800s in Rochester were passing through New York looking for land on the western frontier. The Irish immigrants in this population were motivated initially by general agricultural depression in the homeland and later by experiences in the potato famine of the 1840s. This type of migration is centered around land and agriculture.

The individuals in the MCIG population were from a later migration, with many Germans but also individuals from eastern and southern Europe. The job market in Milwaukee was characterized by an industrial base that processed and shipped food (plant and animal) out Lake Michigan to the eastern United States and Europe (Olson 1987). By the late nineteenth century, the migrations were focused on jobs in the growing industrial markets of Milwaukee and Chicago. The difference in the populations of Monroe County Poorhouse and MCIG, between land and labor based immigrations, may be reflected in the frequencies of LEH. The earlier

communities were assembled from donor populations, mainly Irish, which had experienced hard agricultural times in Europe in the early to mid-1800s. The later migrations of the late 1800s and early 1900s to Milwaukee were comprised of labor seeking individuals from other communities in Europe (Nugent 1992). MCIG's LEH rates are lower than the agricultural based community at Rochester, which had assembled populations who had experienced heavier stress in childhood.

As additional information becomes available for identification of individuals within the MCIG population, it might be useful to make LEH comparisons by ethnic group (including country of origin), and decades of LEH impact. It would be interesting to determine childhood stress levels by migrational groups both from Europe and eastern North America.

Further research on this collection might attempt to survey LEH, not at the gross incidence level, but using Goodman and coworkers' (1984a) technique of "matching" hypoplasia incidences to see how the results vary from collection of LEH totals. The main problem with matching LEH is that it is not the raw measurements which are matched but measurements as reflected in the converted age ranges. Research using these techniques need to clarify how the conversions are executed, and how they affect which LEH measurements are rejected as not exhibiting a major growth arrest. Surveys on the sensitivity of labial versus lingual surfaces is another aspect of LEH

studies that is not covered in this research but has provided some questions as to the legitimacy of LEH scoring.

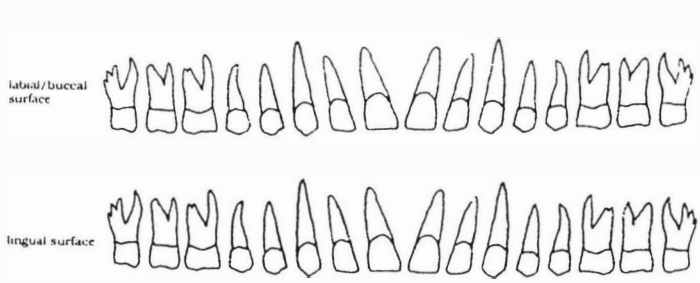
Appendix A

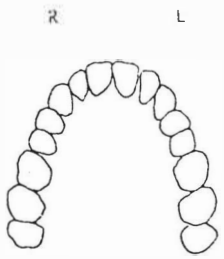
Form Used for Recording LEH

Milwaukee County Institutional Grounds Cemetery
Adult Dentition

date _____ ENAMEL HYPOPLASIAS lot# _____
observer _____ area _____

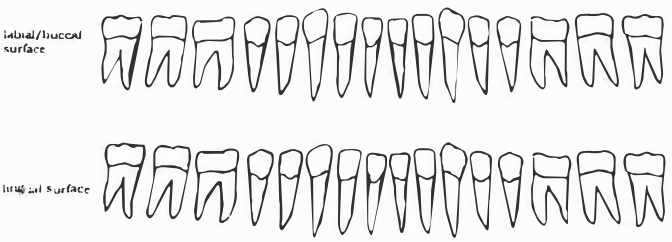
maxillary/palatal dental pathology

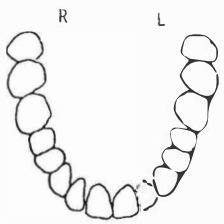




KEY
p = tooth present
/ = tooth not present
p̸ = tooth present but not observable for enamel hypoplasias (due to build-up of plaque, wear, or fragmentation)

mandibular/jaw dental pathology





comments:

IV: Stages of enamel hypoplasias

I. bands
II. lines
III. pits
IV. mottled

SUMMARY:

no observable teeth ____
no observable enamel hypoplasias ____
evidence of enamel hypoplasias ____

Appendix B

Inventory of Individuals in the MCIG LEH Study

Appendix B

Burial number	Sex	Age	No. of teeth	No. of teeth with LEH
3036	Male	50+	2	2
3038	Male	35-50	8	0
3039	Male	35-50	2	0
3041	Female	50+	6	0
3047	Female	35-50	3	1
3055	Male	35-50	12	2
3056	Male	35-50	4	0
3060	Male	35-50	9	6
3061	Male	50+	3	0
3062	Male	50+	5	5
3063	Male	35-50	9	3
3065	Male	35-50	2	0
3066	Male	35-50	3	2
3067	Male	50+	8	6
3068	Male	50+	11	0
3070	Male	20-35	9	0
3071	Male	35-50	5	5
3073	Male	50+	7	2
5001	Male	50+	2	2
5027	Male	20-35	9	5
5048	Male	?	7	0

Burial number	Sex	Age	No. of teeth	No. of teeth with LEH
5074	Male	35-50	10	0
5079	Male	50+	7	7
5089	?	35-50	2	0
5090	Male	50+	2	2
5097	Male	35-50	10	0
5101	?	20-35	11	0
5103	Male	50+	10	1
5105	Male	20-35	12	8
5107	Male	35-50	9	4
5108	Male	35-50	6	0
5116	Male	35-50	6	1
5120	Male	35-50	10	5
5121	?	35-50	11	1
5122	Male	35-50	9	0
5125	Male	35-50	4	4
5128	Female?	50+	2	2
5132	Male	35-50	6	5
5133	Male	35-50	4	1
5139	?	35-50	4	3
5141	Male	35-50	8	0
5144	Male	35-50	9	1
5146	Male	35-50	3	1
5148	?	35-50	8	4

Burial number	Sex	Age	No. of teeth	No. of teeth with LEH
5150	Male	35-50	6	4
5151	Male	35-50	7	5
5152	Male	35-50	10	5
5153	Male	35-50	8	8
5154	Male	20-35	10	0
5155	Male	35-50	12	0
5185	Male	35-50	8	7
5206	Male	35-50	7	4
5210	Male	35-50	9	7
5213	Male	35-50	4	3
5236	Male	35-50	7	1
5243	Male	50+	11	0
9210	?	35-50	7	0
9211	?	35-50	3	3
9212	Male	35-50	12	0
9215	Male	35-50	5	0
9216	Male	35-50	10	0
9217	Male	20-35	11	9
9218	Male	35-50	8	0
9219	Male	35-50	8	0
9222	?	35-50	2	1
9223	Male	35-50	6	0
9224	Male	20-35	10	1

Burial number	Sex	Age	No. of teeth	No. of teeth with LEH
9225	Male	20-35	7	0
9227	Female?	20-35	8	2
9229	Male	35-50	9	0
9230	Male	35-50	5	2
9232	Male	35-50	11	0
9233	Male	35-50	7	0
9236	Female?	35-50	4	0
9237	Male	30-40	8	4
9238	Male	?	12	0
9239	Male	35-50	10	2
9240	Male	20-30	10	0
9241	Male	20-35	4	2
9244	Male	35-50?	11	0
9246	Male	?	2	2
9247	Female	18-25	12	0
9248	Male	35-50	10	0
9251	Female?	?	3	0
9252	Male	25-35	6	2
9253	?	?	11	0
9254	Male	20-35	8	2
9257	Male	35-45	2	0
9258	Male	20-35	11	0
9259	Male	35-50	4	2

Burial number	Sex	Age	No. of teeth	No. of teeth with LEH
9260	Male	35-50	10	8
9261	Male	20-35	5	2
9262	Male	30-40	4	3
9263	Male	30-40	4	0
9265	Male	35-50	7	3
9267	Male	35-50	2	1
9268	Male	35-50	5	1
9270	Male	20-35	5	4
9273	Male	35-50	4	1
9275	Male	35-50	5	0
9276	Male	20-35	8	6
9277	Male	30-40	9	2
9278	Male	?	5	0
9279	?	?	12	7
9281	Male	35-50	4	3
9282	Male	35-50	7	7
9283	Male	35-50	7	1
9284	Male	35-50	11	3
9285	Male	35-50	11	0
9286	Male	35-50	11	2
9289	?	35-50	9	7
9290	?	35-50	8	2
9291	?	20-35	6	2

Burial number	Sex	Age	No. of teeth	No. of teeth with LEH
9292	Male	35-50	5	0
9293	Female	35-50	6	0
9294	Male	35-50	5	5
9296	Male?	35-50	5	0
9297	Male?	?	6	0
9302	Male	35-50	6	6
9304	Male	20-35	7	0
9305	Male	20-35	11	5
9308	Male	35-50	10	4
9311	Male	20-35	7	0
9312	Male?	20-35?	8	1
9313	Female?	Adult	5	0
9318	Male	20-35	9	3
9332	Male	20-35	3	0
9344	Male	35-50	2	0
9346	?	35-50	7	0
9349	Male	35-50	7	6
9350	Male	20-35	10	6
9353	Male	35-50	8	5
9356	Male	35-50	6	3
9357	?	35-50	10	0
9358	Male	35-50	8	7
9362	Male	35-50	6	2

Burial number	Sex	Age	No. of teeth	No. of teeth with LEH
9363	Male	20-35	10	0
9365	Male	35-50	8	1
9367	Male	35-50	3	0
9369	Male	20-35	7	0
Totals			989	288

Age and sex data compiled with the help of Norman Sullivan.

Appendix C

Inventory of Individuals From MCIG Cemetery Whose Dentition was Examined but Discarded From This LEH Study

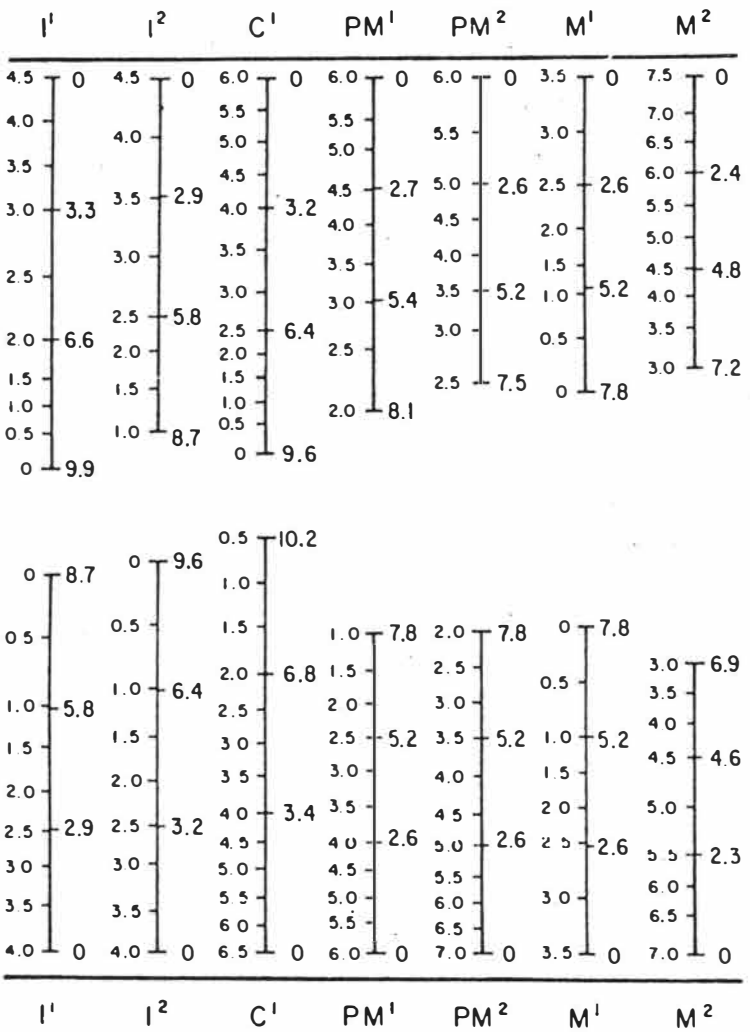
Appendix C

The listed individuals were discarded from the study because they met any one of the following criteria: (a) no anterior teeth present, (b) anterior teeth present but did not retain any enamel, (c) anterior tooth wear greater than one-third of crown surface, (d) crown surface obscured by calculus build-up or excessive staining, or (e) teeth were present but not observable due to capping.

3037	5207	9354
3054	5212	9355
3064	5215	9359
3069	5229	9368
3074	5230	9371
5028	5234	
5047	5235	
5075	5237	
5092	5242	
5094	9220	
5098	9221	
5102	9226	
5104	9235	
5109	9243	
5110	9266	
5111	9271	
5113	9274	
5114	9280	
5118	9295	
5119	9298	
5123	9300	
5124	9303	
5129	9310	
5130	9314	
5135	9330	
5136	9342	
5138	9345	
5140	9347	
5143	9348	
5147	9351	
5149	9352	

Appendix D

Mineralization Diagram for Enamel Formation


I¹
I²
C¹
PM¹
PM²
M¹
M²
3.3
6.6
9.9
2.9
5.8
8.7
3.2
6.4
9.6
2.7
5.4
8.1
2.6
5.2
7.5
2.6
5.2
7.8
2.4
4.8
7.2
8.7
5.8
2.9
9.6
6.4
3.2
10.2
6.8
3.4
7.8
5.2
2.6
7.8
5.2
2.6
7.8
5.2
2.6
6.9
4.6
2.3

Appendix E

LEH Measurement Conversions Using Non-Linear Growth Chart

Appendix E

Developmental age (years)	Maxillary		
	Canine	Lateral Incisor	Central Incisor
0-0.5	8.80-9.60	---	8.96-9.90
0.5-1	8.17-8.80	---	8.30-8.96
1-1.5	7.53-8.17	7.71-8.70	7.55-8.30
1.5-2	7.01-7.53	6.77--7.71	6.60-7.55
2-2.5	6.40-7.01	5.80-6.77	4.95-6.60
2.5-3	5.40-6.40	4.31-5.80	3.30-4.95
3-3.5	4.36-5.40	2.90-4.31	2.23-3.30
3.5-4	3.20-4.36	1.45-2.90	1.10-2.23
4-4.5	2.43-3.20	0-1.45	0-1.10
4.5-5	1.58-2.43	---	---
5-5.5	0.85-1.58	---	---
5.5-6	0-0.850	---	---

Appendix E--Continued

Developmental age (years)	Mandible		
	Canine	Lateral Incisor	Central Incisor
0-0.5	---	7.77-9.40	7.38-8.70
0.5-1	9.00-10.20	6.40-7.77	5.80-7.38
1-1.5	7.83-9.00	5.24-6.40	4.95-5.80
1.5-2	6.80-7.83	4.31-5.24	4.00-4.95
2-2.5	5.80-6.80	3.20-4.31	2.90-4.00
2.5-3	4.99-5.80	2.28-3.20	2.35-2.90
3-3.5	4.19-4.99	1.05-2.28	1.02-2.35
3.5-4	3.40-4.19	0-1.05	0-1.02
4-4.5	2.63-3.40	---	---
4.5-5	1.96-2.63	---	---
5-5.5	1.40-1.96	---	---
5.5-6	0.59-1.40	---	---
6-6.5	0-0.59	---	---

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