Chapter 8

Childhood Health and Dental Development

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Dental enamel hypoplasias are defects in crown development that appear as transverse grooves or series of pits that are partially or entirely around the circumference of the tooth. Hypoplastic defects, although they manifest in the teeth, result from metabolic disturbances of malnutrition and disease elsewhere in the body. Enamel hypoplasias thus provide evidence of general stress that may have been brought about by many different kinds of stressors. Like other "general stress indicators" such as life expectancy, infant mortality, or growth-retardation rates, frequencies of hypoplastic defects can be compared among different populations as a gross index of physical well-being and the adequacy of societal resources upon which the physical quality of life may depend. Of particular value, enamel hypoplasias develop in childhood and adolescence, when both the deciduous and permanent teeth are formed.

The evidence of these early stresses remains apparent in adult skeletons in which teeth have been retained. The defects on different teeth and in different locations on teeth represent stresses at different ages during childhood and adolescent growth, similar to the analysis of tree rings for a record of droughts during the lifetime of a tree. These defects have been observed in archaeological collections and living populations representing a very broad range of human experiences, from those of early hominids to industrial nations. Included among these are a number of studies from African American and Afro-Caribbean archaeological sites (Blakey and Armelagos 1985; Blakey et al. 1994; Clarke 1982; Condon and Rose 1992; Corruccini et al. 1985; Goodman and Armelagos 1985; Goodman et al. 1984).

This chapter puts forward an analysis of hypoplasia frequencies in the New York African Burial Ground sample. Comparisons are made of enamel defect frequencies in different age groups and sex/gender groups. We also compare individuals with culturally modified teeth who were probably born in Africa and those with unmodified teeth whose origins are unknown. Finally, we compare the New York sample with skeletal collections from other diasporic archaeological sites. Questions regarding the physical quality of life in childhood are central, as is our assessment of these data for evidence of health differences or transitions among Africa, the Caribbean, and New York, which take place at different points in the life cycles of New York Africans.

Deciduous dental enamel begins to develop during the fifth month in utero, completing development by the end of the first year of postnatal life. Permanent dental enamel begins formation at birth and continues into the sixteenth year of age. General stress indicators are visible in dental enamel because of the process of enamel formation. Ameloblastic (enamel-producing) activity involves cellular production of a protein-rich matrix that mineralizes, forming the crystalline enamel of teeth. If the development of the enamel crown is interrupted by physiological insult, a transverse groove or series of pits (hypoplasia) or discolored enamel (hypocalcification) results in the "rings" of enamel being laid down at that time (Figures 57 and 58).

Hypoplasia results from differential thickness in the enamel, whereas hypocalcification occurs during interruption within the final stages of ameloblastic activity and results in discoloration of the tooth enamel (Blakey et al. 1994:372). Dental enamel is acellular and, therefore, lesions and discolorations due to physiological stress are permanent and are not obliterated through cellular renewal. In addition to general identification of stress incidence during enamel formation, the rate of enamel matrix formation provides a mechanism for estimating the developmental stage at which the growth arrest occurred (Blakey et al. 1994:372; Goodman and Armelagos 1985). Hypoplasia provides

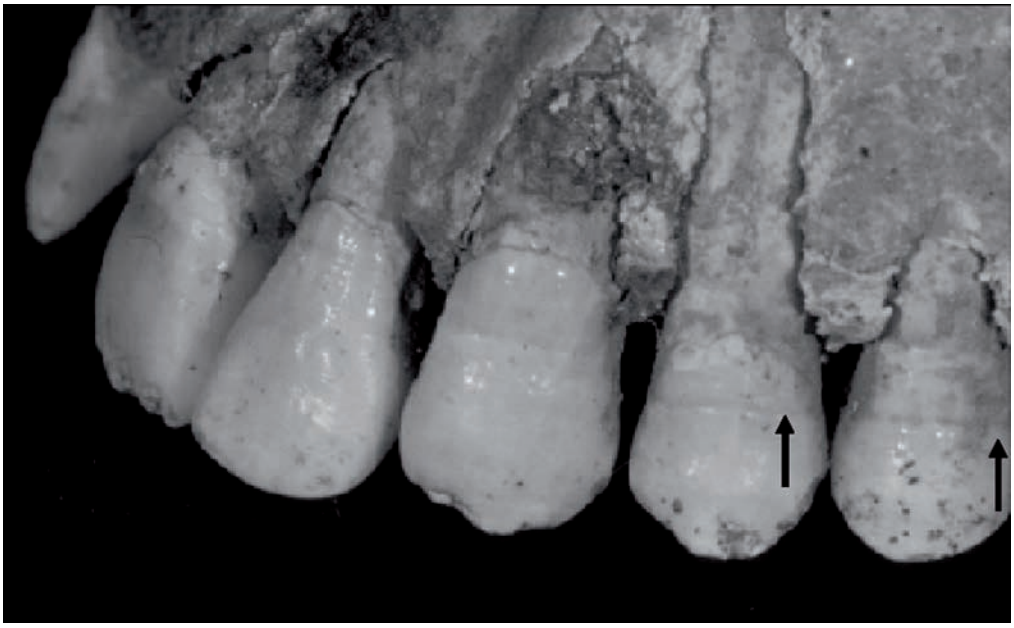

Figure 57. Linear enamel hypoplastic lesions in the anterior maxillary permanent dentition in a female aged 20–25 years (Burial 1).

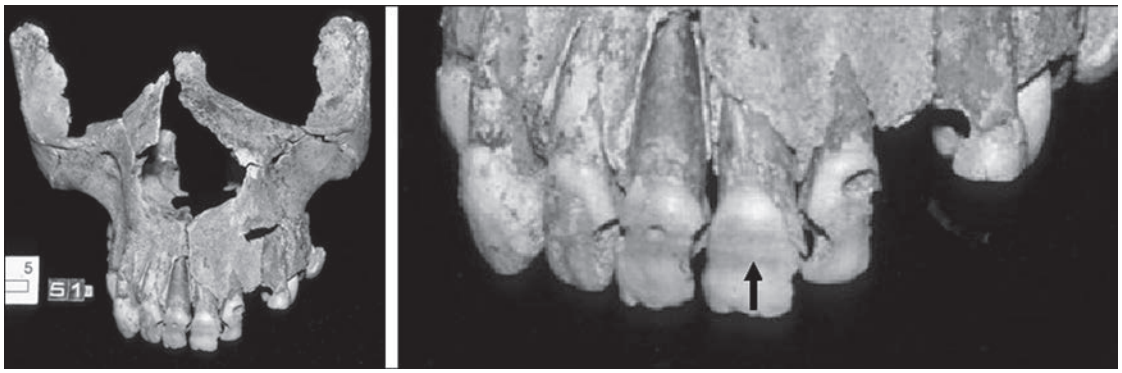

Figure 58. Bands of discoloration caused by hypocalcification in the anterior maxillary permanent dentition in a 24–32-year-old female (Burial 51) (*left*); magnification (*right*).

an estimation of stress severity and/or duration by the size of the malformation. With rare exception, dental enamel hypoplasia is a result of systemic metabolic stress associated with infectious disease, insufficient calcium, protein, or carbohydrates, and low birth weight, characterized together as "general stress" (Blakey et al. 1994; Goodman et al. 1988).

Materials and Methods

A subsample was selected from the New York African Burial Ground sample to study the occurrence and frequency of hypoplasia within adults and children (Table 27). Within this study, the presence of hypoplasia within an individual was defined by the presence of linear or nonlinear hypoplasia in one of the teeth selected for analysis. The absence of hypoplasia was defined by the absence of hypoplasia in all teeth selected for analysis. According to research conducted by Goodman and coworkers, permanent canines and incisors display 95 percent of enamel hypoplasia observed when all available dentition is represented (Goodman et al. 1980). The current study employed this "best tooth" method; we selected individuals with a permanent left or right maxillary central incisor and

Table 27. Summary of Study Samples

Study Description	Dentition	Sample Size	
Hypoplasia and hypocalcification	canines and incisors, permanent	65	99
Hypoplasia and hypocalcification	canines and incisors, deciduous	34	
Hypoplasia controlled for attrition	canines and incisors, permanent	48	
Hypoplasia controlled for attrition	third molars	97	
Canine chronology for hypoplasia	canines, permanent	23	
Hypoplasia and hypocalcification	third molars	111	

a left or right mandibular canine. The presence for permanent teeth was defined according to Codes 1, 2, and 7 within *Standards* (Buikstra and Ubelaker 1994) indicating that teeth are fully developed, in occlusion, and observable. A total of 65 individuals within the New York African Burial Ground were selected for analysis of permanent dentition, which represents the developmental period between birth and 6.5 years of age. A separate selection was conducted for individuals with permanent third molars, left or right, mandibular or maxillary, where presence was defined by Codes 2 and 7 within *Standards* (Buikstra and Ubelaker 1994: 49). One hundred and eleven individuals are included within this third-molar analysis, which represents the developmental period in life from 9 years to approximately 16 years of age.

A subsample was selected from the permanent canine and incisor study and from the third-molar study to control for age- or sex-related differences in dental attrition that might affect hypoplasia frequencies. Individuals with moderate to severe dental wear and individuals for whom dental wear could not be scored (including inability to score because of cultural modifications such as filing and pipe notches), were removed from the canine and incisor sample and from the third-molar sample. Individuals with a dental wear score of 5 or greater, according to Smith (1984), were removed from the permanent incisor and canine sample, resulting in 48 observable individuals. Individuals with a dental wear score of 7 or greater, according to Scott (1979), were removed from the third-molar sample, resulting in 97 observable individuals.

Deciduous dentition was studied by selecting individuals older than 1 year with one left or right central maxillary incisor, one left or right mandibular canine, and one second molar (Figure 59). The presence of deciduous teeth was defined by Codes 1, 2 and 7 within the *Standards* where the teeth were fully developed and observable. Thirty-four subadults were selected to assess hypoplasia in deciduous dentition. Developmental stages spanning approximately 5 months in utero to 16 or 17 years of life are represented by the dentition selected for analysis within this study. Statistical analysis for each study employed SPSS software version 11.5.

Twenty-three individuals were assessed for the chronology of physiological stress episodes that resulted in hypoplastic lesions. Chronology was determined for defects in the left permanent mandibular canines; however, right mandibular canines were used when the left was absent or unobservable. Measurements for the beginnings and endings of hypoplastic lesions had been recorded by members of the New York African Burial Ground Project in the late 1990s (Figure 60). The distance from the dental cervix to the onset of the incisal (beginning) aspect of the lesion was recorded, followed by the measurement of the cervical (latest developing) aspect of the lesion. A midpoint for this episode was calculated, and this measurement was used in conjunction with the total crown height measurement to estimate the age at which each stress episode occurred.

Total crown height was divided by the number of years the mandibular canine develops (6 years), and this figure served as an index representing an increment of growth in 1 year. The midpoint measurement was divided by the yearly incremental growth index, which provided the number of years prior to the end of enamel development (6.5 years of age) at which the incident occurred. Next, this figure was subtracted from 6.5 to arrive at the age of occurrence for each episode. For analysis within this study, the midpoint of the canine, representing the developmental period of 3.5 years, was calculated for each tooth. Episodes were coded as occurring before 3.5 years and after 3.5 years (Table 28). Three and a half years is also the age at which central incisal crown development

Figure 59. Deciduous mandibular dentition with a single non-linear hypoplastic pit in the right canine of a subadult aged 3–5 years (Burial 7). This individual also appears to have been anemic.

Figure 60. Permanent mandibular canine and lateral incisor with linear hypoplasia in a male aged 35–45 years (Burial 9).

Table 28. NYABG Canine Chronology Formula and Example Calculation: CH/6 = YGI 6.5 – (MID/YGI) = Age of Occurrence

Crown Height (CH) (mm)	Total Years of Development	Yearly Growth Increment (YGI)	Crown Midpoint at 3.5 Years	Hypoplastic Lesion Midpoint (mm)	Formula	Age of Occurrence (Years)
12.71	6	12.71/6 = 2.12	6.36	3.93	3.93/2.12 = 1.85	6.5–1.85 = 4.65

Table 29. Frequency of Hypoplasias in Males and Females at NYABG (n = 59)

Males (n = 35)		Females (n = 24)	
Present	Absent	Present	Absent
74.3% (n = 26)	25.7% (n = 9)	62.5% (n = 15)	37.5% (n = 9)

Note: Six of the 65 individuals with adult dentition were too young to determine sex. Therefore, these individuals are not represented in the total number of males and females.

ends, providing a comparison of frequencies represented between the incisor and canine and between the correspondent ages of crown development within the canine.

Results

Among the 65 individuals with permanent dentition, 70.8 percent were hypoplastic. Frequencies for hypoplasia in permanent dentition were higher in the New York African Burial Ground sample than those observed in the enslaved populations of Catoctin Furnace, Maryland (Kelley and Angel 1987), or Newton Plantation in Barbados (Corruccini et al. 1985). The New York frequencies were lower than the total frequencies observed in the largely free and freed nineteenth-century Philadelphia First African Baptist Church (FABC) cemetery sample (Blakey et al. 1994) or enslaved African Americans buried in nineteenth-century Charleston, South Carolina, 38CH778 (Rathbun 1987). The difference in hypoplasia frequencies may reflect the time trajectories and geographic locations represented within these populations. A greater number of people within the New York African Burial Ground and Barbados sites more likely were born in Africa than would have been the case for the nineteenth-century African Americans in Philadelphia and the South. The latter group spent their lives within the conditions of slavery or as free people living under conditions of economic and social inequality.

The difference in hypoplasia frequencies for men and women in the New York African Burial Ground (62.5 percent of the women [n = 15] and 74.3 percent of the men [n = 26]) was not statistically significant, indicating that male and female children experienced similar frequencies of stress episodes from birth to the age of 6.5 years. However, the New York African Burial Ground sample does fall into the general pattern established by previous studies (mentioned earlier and here) indicating that the men have consistently higher percentages of hypoplasia than females (Khudabux 1991; Owsley et al. 1987; Rathbun 1987). Blakey and coworkers (1994) reported hypoplasia in 86 percent of the women and 92 percent of the men among 54 individuals from the FABC sample. Angel and coworkers reported that 71 percent of men and 43 percent of the women at Catoctin Furnace, Maryland, had hypoplasias. The Blakey et al. (1994) study of the Catoctin site indicates that women had higher frequencies of slight linear enamel hypoplasias; however, men had a greater frequency of moderate to severe hypoplasias (68 percent of males [n = 17] and 37.9 percent of females [n = 11]). Among the populations compared within this study, Rathbun (1987) reported the highest frequencies in men and women at the Charleston, South Carolina, site (71 percent in women and 100 percent for men). Tables 29 and 30 provide comparative frequencies and other data for the studies just discussed, and frequency data for the New York African Burial Ground sample are presented in Table 31.

Among the 99 New York African Burial Ground individuals within the canine and incisor studies (permanent and deciduous), 37.4 percent (n = 37) died before the age of 15 years, 86.5 percent of whom

Table 30. Comparison of Frequencies Reported in Skeletal Populations

Site/ Location	Region	Rural/ Urban	Historical Period	Hypoplasia Frequency/ Secondary Dentition (%)	Hypoplasia in Females (%)	Hypoplasia in Males (%)	Hypoplasia in Subadults/ Deciduous Dentition (%)
NYABG, New York	Northeast, North America	urban	1694–1794	70.8 (n = 46)	62.5 (n = 15)	4.3 (n = 26)	85.3 (n = 34)
Newton Plantation, Barbados	Barbados, West Indies	rural	1660–1820	54.5 (n = 56) [a]			
FABC, Pennsylvania	Northeast, North America	urban	1821–1843	89 (n = 54) [b]	86 (n = 29) [b]	92 (n = 25) [b]	92.5 (n = 30) [c]
Catoctin Furnace, Maryland	Southeast, North America	urban	1790–1820	46 (n = 7) [d]	43 [e]	71 [e]	
					slight 79.3 (n = 23) [f]	slight 68 (n = 17) [f]	
					moderate to severe 37.9 (n = 11) [f]	moderate to severe 68 (n = 17) [f]	
Charleston, South Carolina (38CH778)	Southeast, North America	rural	1840–1870	85 (n = 23) [g]	71 (n = 10) [g]	100 (n = 13) [g]	

[a] Newton Plantation site frequencies from Corruccini et al. (1985).

[b] First African Baptist Church (FABC) adult frequencies reported from Blakey et al. (1994).

[c] First African Baptist Church (FABC) frequencies in children cited from Rankin-Hill (1997).

[d] Catoctin site frequencies reported from Kelley and Angel (1987) for overall frequencies.

[e] Frequencies by sex for Catoctin Furnace are from Angel et al. (1987) and Blakey et al. (1994).

[f] Frequencies reported by Blakey et al. (1994), representing frequencies of slight hypoplasia or moderate to severe hypoplasia within the Catoctin Furnace site.

[g] South Carolina 38CH778 site frequencies for males and females from Rathbun (1987). Combined secondary dentition frequency calculated from male and female frequencies reported by Rathbun.

Table 31. NYABG Frequency of Hypoplasia by Age Group and Sex (n = 99)

Age Group	Frequency Within Age Group		
		Men (n = 35)	Women (n = 24)
1–14 (n = 37)	86.5% (n = 32)		
15–24 (n = 17)	76.5% (n = 13)	83.3% (n = 5 of 6)	75.0% (n = 6 of 8)
25–55+ (n = 45)	66.7% (n = 30)	72.4% (n = 21 of 29)	56.3% (n = 9 of 16)

Note: Three children within the 1–14 age category had permanent dentition.

(n = 32) had hypoplasias. Young adults who died between the ages of 15 and 24 years of age represent 17.2 percent of the population, 76.5 percent of whom had hypoplasias. A total of 45.5 percent of the people who died after the age of 25 years (n = 45), 66.7 percent (n = 30) of whom had hypoplasias. The frequency of childhood growth disruption is lowest in the oldest age-at-death groups.

Most of this sample experienced generalized stress in their childhood years. Individuals with permanent dentition (n = 65) representing the period of childhood between birth and 6.5 years of age had hypoplasia in 70.8 percent (n = 46) of the cases, overall. Notably, this frequency is about 20 percent lower than that for the Philadelphia FABC remains. Among children with deciduous dentition, 85.3 percent of the children (29 of 34) had hypoplasia, representing disrupted development between the fifth month in utero through the end of the first year of life. In contrast with the permanent dentition findings, this frequency is more than 20 percent higher than for the FABC.

If the FABC can serve as an operational reference point, one can ask why it is that the childhoods of those who died as adults in New York were relatively less stressed, and those who died as children in New York were relatively more stressed, in comparison with the Philadelphians who died in the 1830s and 1840s. The interpretation of this issue bears on the specific histories of in-migration in the two cities that will be addressed later in this chapter.

The foregoing data suggest that the individuals who experienced early stress episodes resulting in enamel hypoplasia were more likely to have died in childhood and that enslaved children in colonial New York experienced high levels of stress. The lower frequency of individuals with hypoplasia among those who were older than age 25 at death may reflect the forced migration of enslaved men and women arriving in colonial New York. These individuals seem more likely to have experienced childhood stress episodes in Africa than in New York, and their lower defect frequencies might reflect childhood experiences elsewhere. The brisk importation, low fertility, and high child mortality of eighteenth-century New York meant that an African who lived there as an adult was more likely to have been born in Africa (or possibly the Caribbean) than to have been born and survived to adulthood in New York. Although some children were imported, those who died as children in New York seem more likely to have been born there than those who died there as adults. Hypoplasia frequencies in the dead children, therefore, seem most likely to reflect the conditions of New York. The data on lead and strontium content in teeth (see Chapter 6) support those assumptions about the nativity of young children.

Those who died between 15 and 24 years of age had intermediate frequencies of defects in the teeth that developed during early childhood, as shown in Figure 61. We also examined frequencies of hypoplasia in third molars that develop between 9 and approximately 16 years of age. The late childhood and adolescence stress represented by hypoplastic third molars was present in 44.4 percent (n = 12) of those who died between 15 and 24 years and was present in only 10.7 percent (n = 9) of those who died at 25 years of age and older, in whom we could observe third molars (Figure 62). These differences were statistically significant (Pearson chi-square with Yates Continuity Correction = 13.035, df = 1, $p < .0005$). Interestingly, the 15–24-year-olds would have died quite close to the time when these late stresses were occurring. The analysis of 111 individuals with third molars was conducted apart from our usual analysis of incisors and canines. The third molars are less sensitive to hypoplasia than are the anterior teeth and cannot be directly compared with them; however, these hypoplastic lesions may represent more severe episodes of stress (Goodman and Armelagos 1985).

Based on historical documentation of importation ages, we suspect that many of the 15–24-year-olds

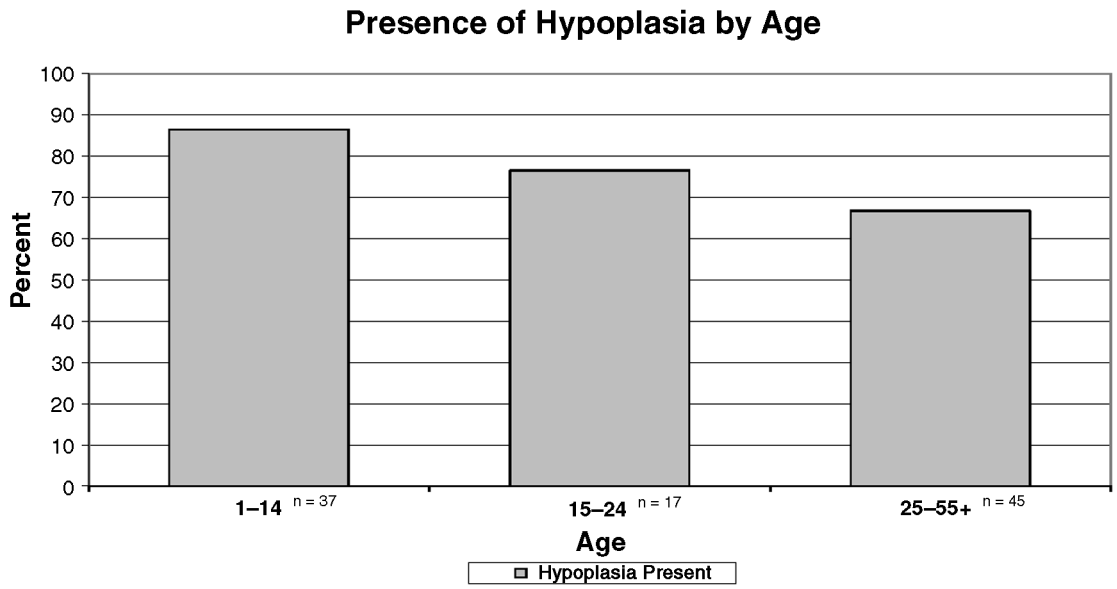

Figure 61. NYABG presence of hypoplasia by age (n = 99).

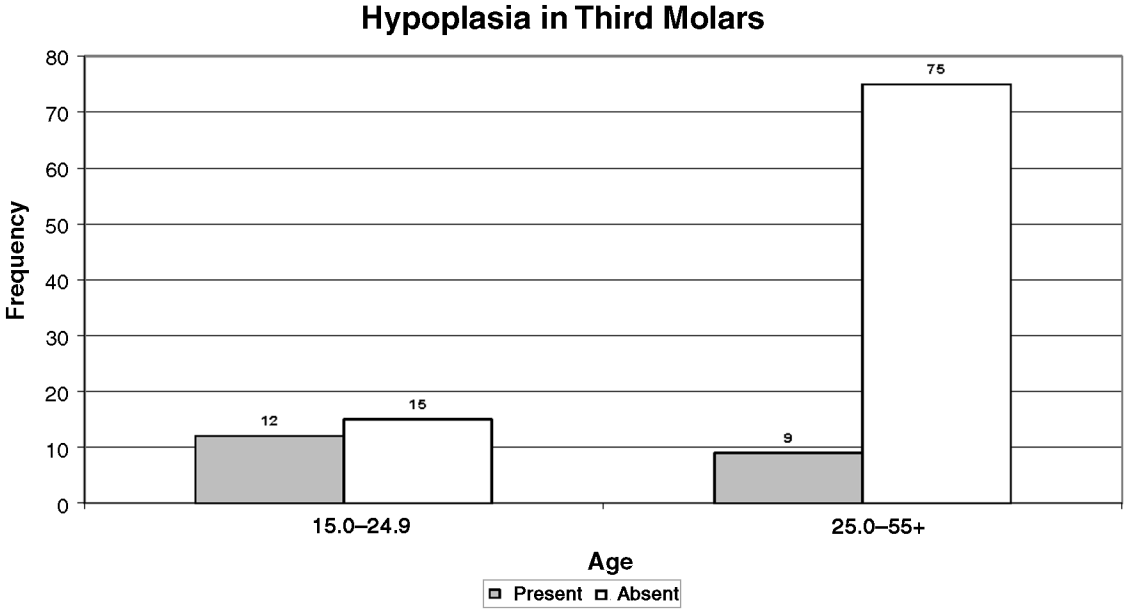

Figure 62. NYABG hypoplasia in third molars (n = 111).

were likely, because of age, to have been new arrivals through the trade in human captives, with the Middle Passage constituting another plausible stressor for them. Fifteen years of age was also the beginning of adulthood in most eighteenth-century censuses in New York; 10 years of age was the criterion of adulthood less frequently used. Studies of active periosteal lesions in this group showed more new infection in the 15–24-year age range than among the older individuals who exhibited a preponderance of sclerotic and healed lesions. Mortality was also very high among the 15–24-year-old males and females, as is detailed in other chapters. Changing conditions of life either through forced migration or/and adult status may be involved in these effects.

The skewing of subadult nativity toward New York, and the skewing of adult nativity toward central and West Africa may help explain low frequencies of

hypoplasia in adults and high frequencies in subadults, when compared to nineteenth-century Philadelphians. The FABC, conversely, shows relatively low frequencies in subadults and high frequencies in adults. This may also be related to different places and conditions of childhood for those who died as children and those who died as adults in Philadelphia, as African births probably were not a major factor in mid-nineteenth-century Pennsylvania. Among the FABC sample, subadult nativity should be skewed toward Philadelphia, as similarly those who died as children in New York were also often born there. Philadelphia in the mid-nineteenth century can be characterized as having a free, disenfranchised, predominantly impoverished, unskilled wage-laboring black community. There was mobility toward greater economic stability among some blacks in the early part of that century, but this was halted during a peak period of Irish immigration into the city at about the time the FABC cemetery was in use (Du Bois 1899; Rankin-Hill 1997). These conditions were stressful, yet hypoplastic stress effects in these dead Philadelphian children were less frequent than in the enslaved children of colonial New York City.

The FABC adults, however, contained a large number of persons who were born and raised in bondage both in late-eighteenth-century slaveholding Pennsylvania and on the eighteenth- and nineteenth-century Southern plantations from which they were given manumission, bought their freedom, or escaped to Philadelphia (Rankin-Hill 1997).

For these FABC adults, their hypoplastic indicators of childhood stress were higher relative to those who died as New York Africans but whose childhoods were frequently spent in Africa. This interpretation of the data is assisted by the facts that the same researchers (and methodological training) were involved in both studies, both archaeological samples are sizable, both primary and secondary dentition were observed, and both sites are in the urban Northeast, thus greatly improving the reliability of comparisons.

Because much of this interpretation relies on the relation of hypoplasia frequency to age, one should examine the extent to which age-related occlusal wear might play a role in reducing our ability to observe hypoplasias, thus reducing the count of defects in older individuals. Subsets of the permanent dentition samples were created to control for the possible effect of dental attrition on hypoplasia frequencies between age and sex groups because of the loss of observable data through tooth wear. The incisor and canine study, as well as the third-molar study, displayed the previously reported pattern of hypoplasia frequencies when attrition was controlled. The highest frequencies were found in individuals aged 15–24 years, and lower frequencies were found in individuals who lived to be 25 years of age and older. These differences were statistically significant in the third-molar analysis only (Pearson chi-square with Yates Continuity Correction = 10.678, df = 1, $p < .002$). Men had higher frequencies of hypoplasia than women within both age groups in the canine and incisor study. These gender differences were not statistically significant. Tables 32 and 33 provide a summary of hypoplasia frequencies within each study. These findings show that the observed decrease in hypoplasia frequencies for older age groups and the differential frequencies between men and women were not a result of lost data because of tooth attrition.

Maxillary central incisors are intrinsically the most sensitive to developing hypoplasias, among all teeth, followed by the mandibular canine (Goodman and Armelagos 1985a, 1985b). Within this study, we compared hypoplasia frequencies in the permanent maxillary central incisors and the mandibular canines in the New York African Burial Ground. Among the 65 individuals, 26 (40 percent) evinced hypoplasia in the maxillary central incisor versus 41 (63.1 percent) in the mandibular canines. Utilizing the overlap in developmental periods represented by these teeth (birth to 3.5 years in the central incisor and 0.5–6.5 years in the mandibular canine) while taking into analytical consideration the intrinsic sensitivity of incisors to hypoplasia in comparison with canines, we sought to assess the periods most stressful in early childhood between birth and 6.5 years for the New York African Burial Ground population.

Hypoplasia chronologies were calculated for the mandibular canines of 23 individuals (Table 34). Among the 37 hypoplasias recorded for these individuals, 73 percent occurred between the ages of 3.51 and 6.5 (n = 27). Analyzed by individual (n = 23) and age, hypoplasias developed between the ages of 3.51 and 6.5 years of age in 95.7 percent of the cases (n = 22). The maxillary incisor frequency may be compared with the mandibular canine chronology frequencies by individual for an analysis of hypoplasia within the most sensitive teeth, by age range—between birth and 3.5 years (evinced by the most sensitive tooth, the maxillary central incisor) and between 3.51 and 6.5 years of age (evinced by the canine, the most sensitive tooth for this developmental period). The

Table 32. NYABG Frequency of Hypoplasias in Canines and Incisors (Controlling for Attrition), by Age and Sex (n = 48)

Age Group	Frequency within Age Group		
		Men (n = 24)	Women (n = 21)
15–24 (n = 16)	81.3% (n=13)	100% (n = 5)	75.0% (n = 6)
25–55+ (n = 32)	71.9% (n=23)	65.2% (n = 15)	34.8% (n = 8)

Note: Three individuals with adult dentition in the 15–24 age group were too young to determine sex. Thus, these individuals are not represented in the total number of males and females.

Table 33. NYABG Frequencies of Hypoplasias in Third Molars by Age Group, Controlling for Attrition (n = 97)

Age Group	Frequency within Age Group
15–24 (n = 26)	46.2% (n = 12)
25–55+ (n = 71)	12.7% (n = 9)

Table 34. NYABG Frequency of Hypoplasia by Age Intervals in Mandibular Canines, by Age Intervals (n = 37 Hypoplasias)

Age (in years)	Frequency
0.5–1	
1.01–2	
2.01–3	16.2% (n = 6)
3.01–4	18.9% (n = 7)
4.01–5	46.0% (n = 7)
5.01–6	18.9% (n = 7)
6.01–6.5	

difference between these two hypoplasia frequencies—40 percent (maxillary central incisors) and 95.7 percent (mandibular canines, between ages 3.51 and 6.5)—is, we believe, substantial when utilizing these data to understand stress episode frequency and quality of life in early childhood (Table 35).

Another factor that must be considered in the interpretation of the canine chronology data is the variability of susceptibility within tooth types. Goodman and Armelagos (1985b:485), studying the Dickson Mounds population, found mandibular canines to be most sensitive to enamel disruption between ages 3.5 and 4 years of age. Among the 23 New York African Burial Ground individuals in this canine chronology study, only 13.5 percent (n = 5) of the stress episodes occurred during this peak period of enamel susceptibility. However, 59.5 percent (n = 22) of the hypoplasias occurred between 4.1 years and 6.5 years of age. These patterns are not consistent with Goodman and Armelagos (1985b). Thus, our finding that 95.7 percent of individuals developed hypoplasias in the mandibular canine between 3.51 and 6.5 years of age is likely a reflection of real age-related differences in stress frequencies, and not simply an artifact of enamel sensitivity.

The individuals within the age category of 1–14 years were more likely to have been born in New York than individuals who were older at the time of death.

Table 35. NYABG Comparison of Hypoplasia in Incisors and Canines

Tooth	Developmental Period/ Age (in years)	Frequency
Maxillary central incisor	0–3.5	40% (n = 26 of 65)
Mandibular canines	0.5–6.5	63.1% (n = 41 of 65)
Mandibular canine chronology	0–3.5 3.51–6.5	26.2% (n = 6 of 23) 95.7% (n = 22 of 23)

Note: Five individuals within the mandibular canine chronology study had multiple hypoplasias and are represented in both the 0–3.5 and the 3.5–6.5 developmental period/age category frequencies.

Their early deaths and high levels of stress indicators, such as hypoplasia, support an interpretation that these children were born into the arduous conditions of enslavement and therefore experienced greater levels of diseases and illnesses, possibly a consequence of being forced to work at young ages. The peak frequencies of hypoplasia between the ages of 3 and 4 years in secondary dentitions observed by Corruccini et al. (1985) among enslaved Barbadians were attributed to weaning at ages 2–3. Blakey et al. (1994) tested the weaning hypothesis within African American enslaved groups to argue that enslaved children experienced physiological stress from multiple sources and that weaning did not account for the peak in hypoplasia frequencies. Furthermore, Blakey's study suggests the need for historical and cultural contexts to be considered within a biocultural interpretation. The high frequencies of hypoplasia during the fifth year demonstrate that this stage was a vulnerable and stressful age for children who survived early infancy and who died as adults. This window on childhood appears to be most pertinent for those who were born in Africa, although childhoods in the Caribbean, New York, and other locations were doubtlessly mixed into our adult sample. How much more stressful the fifth year of age was compared to earlier ages, however, has not been confirmed using enamel defects because of variation in hypoplastic sensitivity across different parts of the crown. Moreover, these data represent the experiences of survivors, whereas the high death toll of infants clearly represents vulnerability and stress among those who did not survive to exhibit developmental defects in secondary teeth. Those deaths (see Chapter 7) clearly resulted from conditions in New York City, albeit precipitated partly by the poor health of captured mothers whose own experiences of childhood stress were relatively less frequent.

The project has used a political-economic framework for explaining biological variations in the New York African Burial Ground sample. For example, Susan Goode-Null's (2002) study of childhood health and development in the New York African Burial Ground sample found that the enslaved people brought into New York between the years of 1664 and 1741 were largely from the Caribbean. Following McManus's *A History of Negro Slavery in New York* (1966), Goode-Null explained that between 1741 and 1770, because of the cessation of slave trading between the British and Spanish colonies and the fear that a slave revolt aborted in 1741 might repeat the events of the 1712 slave revolt in New York, enslaved Africans were imported directly from Africa, rather than via the Caribbean and were largely young women aged 13–40 years and children preferably of 9–10 years of age, rather than adult males. Adult enslaved men from the Caribbean were considered the strategists behind the successful and aborted revolts (Goode-Null 2002:28; see also Chapter 13 in this volume and Medford [2009] for further reference to these factors).

These historical data suggest at least two additional interpretations. One explanation assumes that many children experiencing stress episodes during the ages of 3.5–6.5 years and who lived to adulthood, were born within the colony of New York. Goode-Null's study reported that enslaved children in New York were frequently sold by the age of 6 years (Goode-Null 2002:37–38; Medford 2009). Advertisements indicated that domestic skills promoted the marketability of enslaved children. Therefore, it is likely that children approaching the age of 6 years may have experienced trauma related to separation from their parents, differential nutrition provisions provided by nonparental custodians or slaveholders, or stresses and increased exposure to disease from induction into domestic or other labor duties. Children under the age of 15 were highly stressed, and approaching the age of 6 may have been a significant stage within the

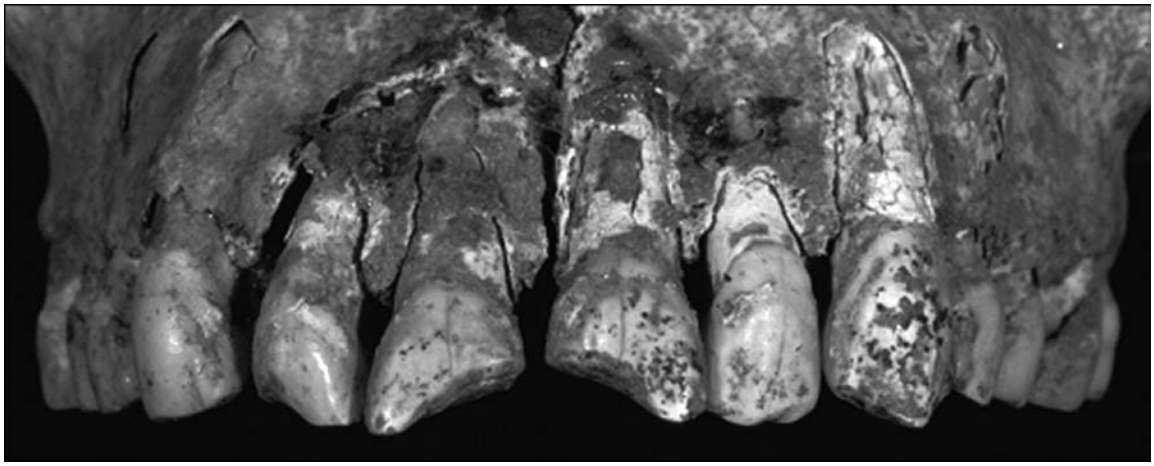

Figure 63. Dental modification.

life histories of children born within the legal status of "slave" in colonial New York. Furthermore, legal definitions of "adult" were applied to children over the age of 10 years in the 1731 and 1737–1738 censuses, and at 16 years in the census data prior to 1731 and after 1737–1738, including the 1810 census (see Chapter 13) (Goode-Null 2002). This legal status as "adult" would most likely have affected the character of labor expected of young enslaved Africans under the age of 15 and within the age group of 15–24. These data further suggest that a child approaching the age of 9 or 10 may have been prepared for an occupational position through entry into labor training and work. Substantial third-molar defect frequencies, especially for those who died between 15 and 24 years of age, characterize stresses of older children and adolescents whether or not they were born in New York.

A second interpretation assumes the inclusion of children imported from Africa to New York, again around the age of 9 or 10, as enslaved laborers. These children may have experienced high levels of physiological stress during their earlier childhood, related to shifts in political power and socioeconomic upheaval within the Atlantic slave trade networks that may have factored into their enslavement. Also, children under the age of 15 years could likely have experienced the Middle Passage prior to their arrival in New York. Any of a host of other possible inadequacies of the large, stratified agrarian societies from which they derived may have contributed to moderately high hypoplastic frequencies in the childhoods of those who died as adults in New York. Consistent with other findings of this study, most of the stresses shown by adult teeth were likely produced by factors within their native African environments with a minority of the adult teeth developing during childhoods in New York. The high frequency of third-molar hypoplasias in those who died between 15 and 24 years of age also suggests effects deriving from arrivals in New York between 9 and 16 years of age in at least 44 percent of the individuals. Those who lived to old age showed far less stress during 9–16 years of age than those who died shortly after arrival in New York.

Our observation that those who lived the longest also had the lowest evidence of childhood stressors may suggest that higher chances of survival to adulthood are associated with having lower stress in childhood, irrespective of where the childhood took place. An attrition of hypoplastic individuals that is associated with age has been postulated elsewhere (Blakey and Armelagos 1985). These are not mutually exclusive propositions; those born in Africa may have had fewer childhood stressors and survived to older ages at death in New York than those who were born in New York City.

One approach to this question has been to compare hypoplasia frequencies for individuals with culturally modified teeth to those without such modifications (Figure 63 and Table 36). Handler's historical study (1994) and our chemical research (see Chapter 6) strongly suggest that modified teeth most frequently indicate African birth. Individuals without cultural modification (probably both African and non-African born) had higher frequencies of hypoplasia than individuals with modified teeth (modified, 66.7 percent [n = 6]; unmodified, 71.4 percent [n = 40]).

Table 36. NYABG Hypoplasia in Culturally Modified and Unmodified Permanent Teeth

Culturally Modified (n = 9)	Unmodified (n = 56)
66.7% (n = 6)	71.4% (n = 40)

The mean ages at death for individuals with modified and unmodified teeth were comparable, although slightly older for individuals with modified teeth (34 years of age for individuals with modified teeth and 31 years for individuals with unmodified teeth). Although consistent with the association between African birth and lower defect frequencies, these differences were not statistically significant at the $p < .05$ level. Chemical and mtDNA analyses will provide greater insight into these interpretations. Indeed, chemical sourcing data would add greatly to the conclusiveness of these tests by providing an independent method of identifying place of birth in at least 200 New York African Burial Ground individuals; this should be done in a future study.

The highest levels of hypoplasia were found within the individuals with deciduous dentition and may therefore represent effects of prenatal stress experienced by the mother during pregnancy. Furthermore, the decreasing frequencies of hypoplasia exhibited by individuals who lived longer suggest a relationship between stress episodes indicated by hypoplasias and a decreased life span.

Dental Enamel Hypocalcification

A study of dental enamel hypocalcification was conducted to assess frequencies within a subsample of 99 individuals. This subject had permanent dentition, including a left or right maxillary central incisor and a left or right mandibular canine, and also included children with deciduous left or right maxillary incisors, left or right mandibular canines, and a second molar.

Within this study of the New York African Burial Ground sample, 67.6 percent (n = 23) of the 34 children with deciduous dentition had hypocalcification (Table 37). Among the 65 individuals with permanent dentition, 18.5 percent (n = 12) had hypocalcification. Women had a higher frequency of hypocalcification than did men (72.7 percent of the 24 females versus 27.3 percent of the 35 males).

Within this subsample, 60.5 percent (n = 23) of the 38 children under the age of 15 years had hypocalcification (see Table 37), whereas only 10 percent (n = 2) of the 20 young adults aged 15–24.9 years and 28.6 percent (n = 10) of the adults aged 25 and older had hypocalcification (see Table 37). These differences were statistically significant (Pearson chi-square = 19.84, df = 2, $p < .0005$) and mainly reflects the change from predominantly primary to secondary teeth by age 15. The difference between hypocalcification frequencies found in individuals with deciduous dentition (67.6 percent, n = 23) and permanent dentition (18.5 percent, n = 12) should not be considered in the same manner in which this age-related pattern in hypoplasia has been considered. Deciduous dentition is more likely to become hypocalcified than to exhibit hypoplasia, and deciduous dentition typically displays

Table 37. NYABG Comparison of Hypocalcification and Hypoplasia Frequencies by Age Group (n = 99)

Age Group (in years)	Percentages within Age Groups	
	Hypocalcification	Hypoplasia
1–14	62.2 (n = 23)	86.5 (n = 32)
15–24	10 (n = 2)	80.0 (n = 16)
25–55+	28.6 (n = 10)	66.7 (n = 30)

Note: Three subadults in the 1.0–14.9 age range had permanent teeth. These individuals are only represented once within the combined studies of permanent and deciduous dentition.

higher frequencies of hypocalcification in comparison to permanent dentition (Blakey et al. 1997). Thus, the observed low frequency of hypocalcification in permanent dentition follows the expected pattern caused by suspected intrinsic differences between deciduous and permanent dentition that may have nothing to do with stressor prevalence. Comparisons of hypocalcification across primary and secondary dentition are therefore inappropriate.

However, comparison of the two defect types within deciduous dentitions is of interest. Deciduous dentition forms in utero and continues into the first year of life and therefore represents early childhood development and a measure of prenatal health and the health status of the mother. Hypocalcification and hypoplasia frequencies were both highest in children dying prior to the age of 15 years, demonstrating high physiological stress and vulnerability during the prenatal and early childhood years. The higher levels of hypoplasia (86.5 percent) versus hypocalcification (65.7 percent) within deciduous dentition (n = 34) is unexpected, however, given the tendency of deciduous teeth to preferentially exhibit hypocalcification. Hypoplasia frequency in this case is extraordinarily high compared to other deciduous dental studies using similar methods (Blakey and Armelagos 1985; Blakey et al. 1994, 1997; Rankin-Hill 1997). Both defect frequencies indicate the extremely high levels of stress experienced in utero and during the first year of life among the New York African Burial Ground children who died before the age of 15.

Conclusions

Historical data on the ages of children who were in various stressful contexts have been applied to explain developmental defect frequencies that occurred at different ages in the childhood and adolescent periods of the life cycle. Children likely born in colonial New York within the condition of slavery were more vulnerable to health risks and early death due to nutritional deficiencies and illness than is evident for the childhoods of those who were likely to have been born in Africa. The findings of this study suggest disparity between early childhood health and nutrition for individuals more likely to have been born in colonial New York and individuals likely to have been born as free people in the agricultural villages of the war-torn states of central and West Africa (see Medford 2009). The fact that higher frequencies of enamel defects were found in children under the age of 15 and among individuals without dental modification, than among individuals who were most likely to have been born in Africa (older individuals and those with modified teeth), supports this hypothesis. The chronology of physiological insults resulting in hypoplasia further supports the vulnerability of childhood and adolescence for enslaved Africans in New York.

The third-molar data reflect the trajectory of life experience for individuals, most of whom were likely to have been born in Africa and enslaved in the Americas. Significantly higher hypoplasia frequencies found in the third molars representing the developmental period of 9–16 years correspond with historical data indicating high levels of importation of older children, adolescents, and young adults in the eighteenth century. These findings indicate that the quality of life for Africans was greatly compromised upon entry into the New York environment of enslavement through the processes of either birth or forced migration.