Chronological Distribution of Enamel Hypoplasias and Weaning in a Caribbean Slave Population

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ABSTRACT

Dental enamel hypoplasia is a putative marker of childhood morbidity (nutritional or infectious stress) which can be analyzed by age-of-occurrence using a calcification standard. We have recorded age-specific occurrence of (a) minor linear hypoplasias, (b) pits, (c) major growth-arrest lines, and (d) combined hypoplasias in 103 specimens of 17-19th century Caribbean slaves. This population is probably unique in terms of environment, nutritional deficiency and other severe environmental stresses, and (especially) association with historical resources that might allow more specific correlation of stresses with hypoplasia chronology.

Barbados slaves have a clearly defined central age tendency of 3-4 years at formation of hypoplasias. The lateness of the mode, the percent concentration between 3-4 years, and the residual occurrences at 4+ years are relatively pronounced compared to other reported populations (notwithstanding differences in counting techniques). The age of first hypoplasia occurrence per individual is also probably later in slaves than in other populations. The 3-4 year age range encompasses the year following the historically-documented relatively late time that slave children were weaned (at 2-3 years). Other non-industrial populations show a hypoplasia peak at 2-3 years following a presumed weaning at 1-2 years. Thus the weaning hypothesis and other historical factors (such as periodic food shortages and famine conditions) help explain the mode and the residual distribution of hypoplasia. The historical sources also support the general expectation that the post-weaning period was one of high risk.

Following studies of extant populations (Sarnat and Schour, 1941; El-Najjar et al. 1978; Infante and Gillespie, 1974, 1977; Moller et al. 1972; Ainamo and Cutress, 1982; Gruenwald, 1973), dental enamel hypoplasia has recently become popular as a marker of childhood morbidity in excavated remains (Goodman et al. 1980, 1984; Schulz and McHenry, 1975; Cook and Buikstra, 1979; Hillson, 1979; Black, 1979; Clarke, 1980; Corruccini et al. 1982). Although precise causes of this interruption in enamel deposition are multiple (Pindborg, 1982; Fraser and Nikiforuk, 1982; El-Najjar et al. 1978), much of the hypocalcemia leading to hypoplasia formation is generally assumed to reflect metabolic stress, temporarily disturbing amelogenesis, due to malnutrition and/or infection.

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In an earlier analysis of a slave osteological sample from the Caribbean island of Barbados, we assumed that relatively high hypoplasia prevalence would be consistent with this etiology (Corruccini et al. 1982). The historical evidence indicates Caribbean slaves experienced nutritional deprivation and severe developmental disruption (e.g., Dirks, 1978; Sheridan, 1985: 201-219, 234-239; Kiple and Kiple, 1980). Results generally confirmed our expectations (see also Handler and Corruccini, 1983).

We also briefly noted a tendency for more numerous and serious hypoplasias to occur in late- rather than early-calcifying teeth; this tendency was possibly related to a late weaning period in Caribbean slaves. The logical relation between hypoplasia peak and a society's typical weaning age (the post-natal time of maximum environmental/metabolic stress on children in non-industrial societies) is the major theme of many hypoplasia studies (Hillson, 1979; Black, 1979; Clarke, 1980; Goodman et al. 1980).

In this paper we give a more detailed analysis focused on the chronology and classification of enamel hypoplasias in Barbados slaves, with special reference to the weaning hypothesis and the integration of historical and biological data.

Materials and Methods

History

From the middle of the 17th century to 1834, when slavery ended in the British Empire, the wealth of Barbados depended on the cultivation of sugar, mainly grown on plantations serviced by the labor of tens of thousands of slaves of African birth or descent. Archaeological investigations in Barbados during the early 1970s, one phase of a larger historical anthropological project concerned with the social and cultural life of the island's slaves, ultimately focused on a slave cemetery at Newton plantation. Newton was chosen as a site because of the availability of extensive historical documentation and the existence of a slave cemetery permitting excavations in relatively undisturbed contexts. In all major social and demographic characteristics of its slave population, Newton typified medium- to large-scale Barbados sugar plantations of the slave era (Handler and Lange, 1978, pp. 58-102); thus the analysis of Newton's slaves may be generalized to other plantation slave populations on the island.

The skeletal sample reported here includes remains from 104 individuals (103 with any teeth) interred from about 1660 to 1820. Further

laboratory examination revealed three more individuals from among the commingled remains than the 101 previously reported (Corruccini et al. 1982). This is the largest and earliest excavated group of African and African-descended slaves yet reported from the Caribbean and North America.

Official governmental documents, traveller and missionary accounts, statistical reports, private letters, newspapers, plantation journals, etc. constitute an abundance of primary historical sources relating to Barbados during the slave period (Handler, 1971). Much can be gleaned from these sources, although information on slave topics is often highly variable in quality and completeness, particularly for the household and community life of plantation slaves. Caribbean slavery scholars have become accustomed to working with such limitations.

Only a handful of primary historical sources treating Barbados slave life provide any information on lactation and weaning, particularly the age levels of children at time of weaning. The available information largely relates to the preemancipation decades of the 19th century (the later phases of the slave period), and is usually confined to a phrase or two. All of this information is derived from the observations of Whites (usually planters and medical doctors who treated plantation slaves) who were generally knowledgeable about slave life and plantation conditions.

Skeletal biology

Age at death was estimated primarily from dental calcification and eruption schedules until young adulthood, then through use of a tooth-wear scale calibrated against tooth eruption and mesial-distal wear gradient (see Corruccini et al. 1982). In only about five cases did epiphyseal and cranial sutural fusion, and general appearance come into use. Therefore, the descriptive demography of the sample of adult slaves is unavoidably subjective; after age 30 estimates were made to the nearest five years. As the recovered materials were mostly dental, we could not confidently sex individuals more than 50% of the time; accordingly, none of our analysis is sex-specific.

Our diagnosis and classification of hypoplasia is based on modification of the earlier-cited sources along with Dean (1934) and Menezes (1976). The cleaned teeth were examined under a stereomicroscope at 6.4× to 16× for the following three forms of hypoplasia: thin or faint striations representing linear enamel hypoplasia (LEH); horizontal pitting defects (PIT); and horizontally continuous, palpably deep major growth arrest lines (MGA).

In dental patients hypoplasias are often accompanied by mottling or discoloration of enamel deposited after the hypoplasia occurrence (C. Neill, pers. comm.; Dean, 1934; Ainamo and Cutress, 1982; Menezes, 1976). Although we observed no such mottling, much brown mottling occurred independently; the above sources refer to this as "opacity" and classify it not as a hypoplasia but as a different type of enamel defect. In our original brief description (Corruccini et al. 1982) mottling was considered a hypocalcification form and helped account for the 99% prevalence we reported.

Hypoplasias in each of the three categories were counted and measured (to their midpoint) from the cemento-enamel junction using sharpened sliding calipers calibrated to 0.02 mm. Bilateral hypoplasias, or hypoplasias on different teeth representing the same stress event were counted only once. The cervical distance measurements were used to estimate specific age-at-occurrence for the hypoplasia-inducing events following Goodman et al. (1980). The mineralization scale was calibrated, assuming uninterrupted enamel deposition between the given calcification stages. Since a few individuals with multiple hypoplasias could bias the reporting, we also scored age-at-occurrence once per hypoplastic individual, using the mean age, for a separate analysis.

The scale adapted by Goodman et al. (1980) from Swärdstedt (1966) and Massler et al. (1941) is based on U.S. Whites, and might be developmentally accelerated by standards of a malnourished population such as Black slaves. To maintain comparability with other studies we did not attempt to adjust the slave data for possible nutritional or racial differences in calcification.

Results

Of 103 individuals with teeth, 56 are hypoplastic. This 54.5% prevalence is clearly an underestimation. The average number of recovered teeth for individuals without observable hypoplasia is 10.98 and for hypoplastic cases it is 19.45; thus the probability of observing the pathology is highly related to completeness of preservation (postmortem) or tooth retention (antemortem).

Of the 56 individuals with any hypoplasia, 34 show MGA and PIT, the hypothetically more severe manifestations. Published figures and examination (RSC) of Southwestern and Ohio Valley Amerinds and some U.S. colonial Black samples (Clifts Plantation; Catoctin Furnace) indicate the prevalence and severity of MGA and major pitting in the Barbados sam-

ple is perhaps the highest yet observed (also J. L. Angel, D. Cook and G. Armelagos, pers. comms.).

Age-specific occurrence of lines can be graphed to indicate the distribution of individual hypoplasia events. The histogram for individual events in Barbados slaves is given in Figure 1. LEH occurrence peaks at

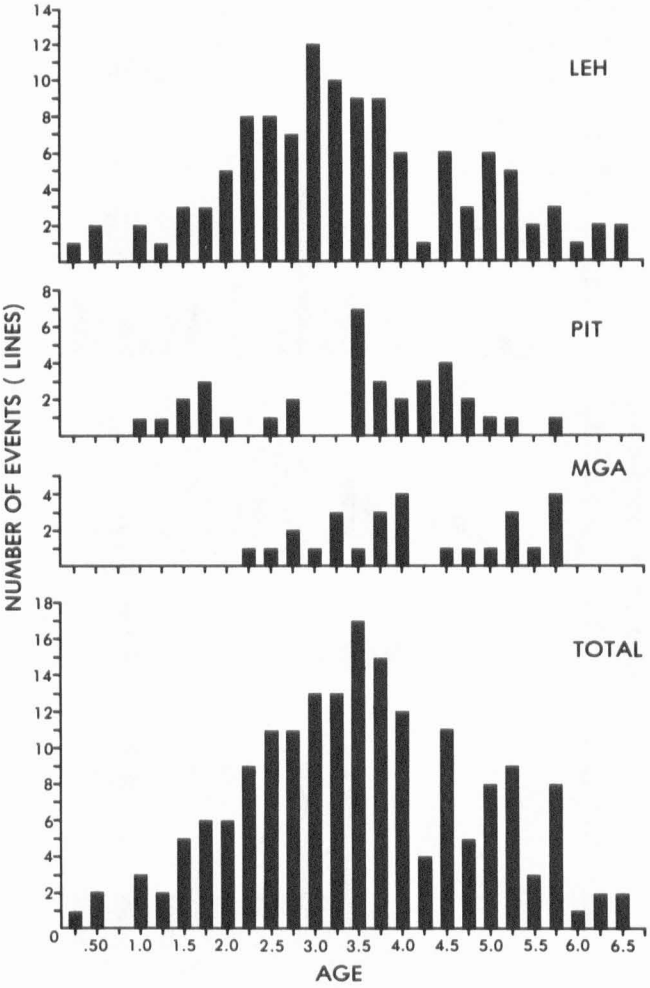

FIG. 1. Age-specific frequency in Barbados slaves of linear enamel hypoplasia (LEH), pitting (PIT), major growth-arrest lines (MGA), and the sum of all three hypoplasia types. All separate enamel defect events were counted; thus there is more than one event per individual in many cases.

ages 3.0-3.25 years, PIT at 3.5 years, and MGA at 4+ years; combined hypoplasias follow a quasi-normal distribution peaking at 3.5-3.75 years. Thus the relatively minor LEH (perhaps representing less severe stress events) peak somewhat earlier than the more pronounced defects. The mode for total hypoplasia occurrence, once adjusted for probable delayed growth, is undeniably in the 3+ to 4 year range.

Considering hypoplasias by individual specimen rather than individual lines (Figure 2), a generally lower modal age shows most hypo-

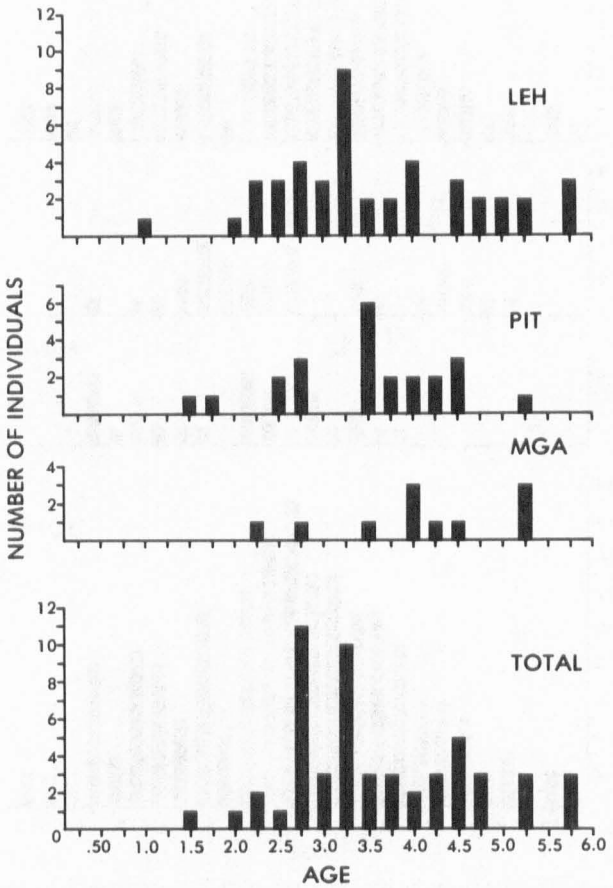

FIG. 2. Age-specific hypoplasia frequency with every individual specimen counted just once. The average age-of-occurrence of hypoplasia was used in individuals with multiple events.

plastic events occurred after the mean age of susceptibility for individuals. By individual specimen, LEH peaks at 3.25 years, PIT at 3.5 years, MGA at 4+ years and summed hypoplasia at 2.75-3.25 years. Thus, for any given individual slave surviving infancy, hypoplasia was most likely to occur around age three. A different perspective is provided by considering age distribution of first lines in the hypoplastic cases; these follow a markedly platykurtic distribution with mode almost evenly spread over the 1.75-3.5 year range. The central tendency, although weakly pronounced, is at 2.75 years for first event.

We find no evidence for differential mortality according to hypoplasia-liability, unlike Goodman et al. (1983). Individuals lacking defects average 27.6 years at death (S.D. = 16.1); those with one defect 31.0 years (S.D. = 13.8); and those with two or more defects, 28.8 years (S.D. = 11.8). The probability for these being samples drawn from the same parent statistical population ranges from .30 to .70. The variance of age-at-death differs, however ($F = 1.76$, just at .05 probability, with more variable ages corresponding to less hypoplasia).

Discussion

The overall central tendency for hypoplasia formation is 3-4 years in postnatally surviving Barbados slaves. This is late in comparison to other populations studied (see Table 1). Chicago Whites peak at 0-1 year, composite prehistoric Amerinds and Missouri Mississippian Amerinds peak at 2-3 years, and Dickson Mound combined pre-agricultural and agricultural Amerinds and medieval Westerhus Swedes peak at 2.5-3 years. The Westerhus distribution (of pre-modal versus post-modal occurrences) alone does not differ significantly from slaves, but compared to all others it is relatively platykurtic. The concentration of defects between 3-4 years, compared to those occurring either earlier or later, is significantly greater in the slaves than in Westerhus ($\chi^2 = 5.71$, $p < .01$).

Except for medieval Swedes, the Barbados slave total hypoplasia distribution appears less leptokurtic than others, with a less pronounced peak. This could suggest chronicity of the condition, but from that interpretation one would expect stronger leptokurtosis in the graphs by individual (Figure 2) than by the event (Figure 1). The opposite occurs, however, and first-time hypoplasia distribution is even more platykurtic. Thus both the peak (or mode) and the residual distribution at post-modal ages are more pronounced in the slaves than in other known samples (but

Table 1

Frequencies of Hypoplasia Occurrence Prior to and Following the Modal Age-of-Occurrence, Compared to Barbados Slaves

Group	Modal Age (years)	Post-Modal Proportion[7]	Barbados Proportion[8]	P[9]
Chicago Whites[1]	1	.27	.96	10^{-10}
Composite Amerinds[2]	3	.09	.61	10^{-10}
Composite Amerinds[3]	2	.31	.86	10^{-10}
Missouri Amerinds[4]	2–3	.50	.86	10^{-10}
Dickson Mounds[5]	3	.50	.61	.02
Medieval Swedes[6]	3	.59	.61	.09

[1]Sarnat and Schour, 1941.

[2]Clarke, 1980.

[3]Radio-opaque tibial lines for comparison to reference 2; Clarke and Gindhart, 1981.

[4]Black, 1979.

[5]Goodman et al. 1984.

[6]Swärdstedt, 1966.

[7]Proportion occurring at ages above the listed mode.

[8]Analogous proportion in slaves relative to other population's mode (not the 3+ year mode shown by slaves).

[9]One-tailed probability from χ^2 test.

see a possible exception in lower class industrial Americans c. 1900; Goodman, 1984).

As we suggested earlier for Barbados (Corruccini et al. 1982; Handler and Corruccini, 1983) and as has been suggested for other populations (especially Clarke, 1980; Goodman et al. 1984; Hillson, 1979), weaning practices are a major explanatory factor for hypoplasia-marked growth disturbances. However, a direct correlation between age of weaning and of hypoplasia concentration has only been presumed, never firmly established in a single population.

The physical evidence shows a modal slave hypoplasia occurrence in the 3-4 year range, a possible direct result of weaning. The historical sources point to a late weaning period in Barbados slaves, thus indicating they conformed to a pattern found in other British West Indian slave societies, a pattern that undoubtedly derived from African practices.

By the early 19th century, over 90% of Barbados' slave population was native-born (Handler and Lange, 1978, pp. 23, 29). In the earlier periods of slavery a much greater percentage of slaves were African-born and African cultural patterns were also followed to a much greater extent. In

18th century African cultures, as today, lactation generally lasted two to three years (Klein and Engerman, 1978). There is suggestive historical evidence for Barbados that during the 18th century weaning also tended to take place between two and three years and generally conformed to the African pattern (Francklyn, 1789, p. 52; Walduck, 1712) as did other areas of the British West Indies (e.g., Collins, 1811, p. 146; Grainger, 1764, p. 17; cf. Klein and Engerman, 1978; Sheridan, 1985, p. 245).

Historical evidence also indicates that during the first few decades of the 19th century (the preemancipation period), the Barbados slave lactation period typically lasted between 18 and 24 months, or even longer (Society for the Improvement, 1811, pp. 128-134; 1811-1816, pp. 119, 139-140; Barbados Council, 1824, pp. 109-110, 114-115), also reflecting a pattern found elsewhere in the British West Indies during this period (Higman, 1984, pp. 353-354; cf. Sheridan, 1985, p. 245).

In general, then, the historical evidence from Barbados and the British West Indies tends to be mutually supportive and indicate that during the 18th century (and earlier) weaning commonly took place from at least two years and extended up to the third year. The evidence also suggests that although the average lactation period somewhat decreased toward the later phases of slavery, it still remained considerably higher than for contemporary Europeans (cf. Klein and Engerman, 1978).

Historical sources not only indicate relatively prolonged lactation, but also that the weaning period itself was particularly stressful and dangerous for slave children. For example, a prominent Barbados planter wrote in his late 18th century tract on slave management how "the dangerous period of a Negro's life is from the time of his being weaned to the age of seven years"; "It is during this period," he stressed, "that these infants are most liable to contract habits and diseases which destroy them" (Gibbes, 1797, pp. 91-92). Other sources also suggest that health risks were high during weaning even though these sources tended to attribute the risk to prolonged lactation rather than weaning per se. In the early 19th century a group of Barbados planters discussed the high incidence of infant child mortality among slaves. This incidence was attributed to, among other reasons, the "great injury . . . often occasioned to them . . . by late suckling" (Society for the Improvement, 1811-1816, p. 119). Two Barbados-born medical doctors, who practiced among plantation slaves, also alluded to the risks of the weaning period. One doctor attributed high infant mortality to the "highly improper food" mothers gave their children as well as "the period of nursing the child being protracted to an age when it is both injurious to mother and infant"; the

mother "suckles her child to a very advanced age," he later added, "and while the practice weakens her . . . the child, whose stomach now requires stronger food, dwindles and becomes scrophulous, and disposed to worms from a continuance of too meagre a diet." The other doctor also assigned the causes for high infant mortality to housing conditions, "improper food, and the late suckling of the children by their mothers" (Society for the Improvement, 1811, pp. 128-134). Moreover, an implicit motive for this opinion was the assumed relationship of prolonged lactation and depressed fertility in slave women. "Not weaning . . . for two or three years," observed a visitor to Barbados around 1780, "occasions even the breeding women to have fewer children than might otherwise be expected" (Francklyn, 1789, p. 52). And in the early 19th century a committee of planters also maintained that "the present very protracted nursing is very injurious to the children & hurtful to the mother, besides having the effects of contributing to lessen the number of births" (Society for the Improvement, 1811-1816, pp. 139-140).

In conclusion, the physical anthropological and historical evidence mutually establish a relatively late weaning in Barbados slaves (thus also supporting the historical evidence for extended lactation in other British Caribbean areas) and suggest that the weaning period was developmentally disruptive and traumatic. Using historical and nutritional data, Kiple (1985, pp. 129-130) has also recently discussed how weaning posed a "serious nutritional threat" and had a debilitating effect on the life of the Caribbean slave child in general; the Barbados data independently support his discussion. The 3-4 year modal range for hypoplasia in Barbados encompasses the year following the relatively late time that slave children were weaned. As far as we are aware, the Barbados case is the first example for a specific population wherein hypoplasia concentration can be shown to occur in the year following an historically documented time of weaning. The weaning hypothesis specifically, and other factors more generally (including periodic food shortages and famines, some of which were caused by hurricanes and heavy storms, epidemic infections and disease, and political events; see, for example, Dirks, 1978; Sheridan, 1976, 1985, pp. 154-173, 200-219) help explain the mode and residual distribution of hypoplasia in Barbados slaves. The historical sources, despite their limitations, also corroborate and support the general expectation that the post-weaning period was one of high risk for Barbados' slaves (as well as for other Caribbean slave populations; cf. Sheridan, 1985, p. 203; Kiple, 1985, pp. 129-130).

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