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Congenital Syphilis in the Past: Slaves at Newton Plantation, Barbados, West Indies

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ABSTRACT Hutchinson's incisors and Moon's molars are specific lesions of congenital syphilis. The extensive but fragmentary clinical literature on these conditions describes reduced dimensions and thin enamel in the permanent incisors and first molars, crowding and infolding of the first molar cusps, notching of the upper incisors, and apical hypoplasias of the permanent canines. A Barbados slave cemetery (ca. 1660–1820 AD) includes three individuals with these features, suggesting a frequency at birth of congenital syphilis in the population approaching 10%. These three cases show triple the frequency of all hypoplasias and more than seven times the frequency of pitting hypoplasia present in the remainder of the series. The recognizable congenital syphilis cases account for much of the remarkably high frequency of hypoplasias in the series as a whole. We infer that syphilis contributed substantially to morbidity, infant mortality, and infertility in this population. Presence or absence of congenital syphilis may account for much of the variability in health and mortality seen among nineteenth century African-American populations.

Skeletal remains from historic populations offer fascinating possibilities for relating osteological and documentary evidence for the health costs of specific diseases. Previous studies of remains from the Newton Plantation Cemetery, Barbados, have been especially interesting in the articulation between linear enamel hypoplasia (LEH) as a nonspecific health indicator and historical records of diet, living conditions, and mortality in Caribbean slave populations (Corruccini et al., 1985, and elsewhere). The remarkable severity of LEH in this collection led two of us (K.P.J. and D.C.C.) to question whether weaning stress and nonspecific morbidity were sufficient to account for these lesions, as was previously supposed. To what extent was a specific infectious disease, congenital syphilis, responsible for producing these high levels of LEH? In this paper, we first review the extensive, but disparate and quite fragmentary clinical literature on the dental effects of congenital syphilis, in order to familiarize paleopathologists with the unusual morphology that results. We search for these morphological patterns in the Newton Plantation teeth. We then reassess the health of the Newton Plantation population in light of our findings.

Congenital syphilis has been diagnosed very seldom in the voluminous literature on the paleopathology of the treponemal diseases. In their exhaustive review, Baker and Armelagos (1988) have discovered only two cases: one from preColumbian Virginia (Ortner and Putschar, 1981) and one from Peru (Goff, 1967). Both diagnoses are based on bone lesions in children. In the first case, the distinctive dental features of congenital

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syphilis are absent; in the second, they are not discussed. Reports of the absence of these dental features in ancient populations are uncommon (but see Leigh, 1934; Cook, 1990). Among more recent populations of interest to paleopathologists, Hutchinson's incisors are reported in a single nineteenth century African American specimen (Rose, 1985). We know from historical sources that physicians recognized congenital syphilis from 1500 forward (Quétel, 1990; Dennie, 1962). Why have paleopathologists not found its distinctive features?

The dental features of congenital syphilis were first described by Hutchinson (1858) and Moon (1877). Hutchinson's incisors constitute one element of Hutchinson's triad (permanent incisor malformations, nerve deafness, and interstitial keratitis) that was the basis for diagnosis of congenital syphilis before immunological tests were developed early in this century. Hutchinson described the incisors in congenital syphilis as follows.

> The central upper incisors are the test-teeth. You may neglect all the others, for, although malformations are often observed in them also, as for instance, a rounded peg-like form in the lower incisors, yet there is nothing that is trustworthy, and much that is liable to mislead. Look at once at the two upper central incisors; and if they be broad, well-made teeth, you may throw away suspicion as far as dental indications are concerned . . . The teeth are short and narrow. Instead of becoming wider as they descend from the gum, they are narrower in their free edges than at their crown, their angles having been, as it were, rounded off. In the centre of their free edge is a deep vertical notch, made by the breaking away or non-development of the middle lobe of the tooth-crown. This notch, taken together with the narrowness and shortness of the tooth, is the main peculiarity; but you will observe also that the colour of these teeth is not good. Instead of looking like ivory with a thin coating of pearl, they present a semi-translucent appearance, not unlike that of bad size, as we see it displayed in the oilmen's shops (Hutchinson, 1861:515).

> As before observed their softness from deficiency of enamel renders them liable to premature wearing down. The teeth of a syphilitic patient not twenty, will often be ground down as much as those of a very old person . . . (Hutchinson, 1858:450).

Several metaphors have been advanced for the peculiar shape of the upper central incisors in congenital syphilis. Hutchinson (1885:Plate VI) described them as "screwdriver" shaped, referring, we believe, to the bulbous, notched blade of a London or cabinet pattern forged screwdriver (Salaman, 1989), "broader at its neck than at its free edge" (Fournier, 1884:22). The term *pumpkin seed* (Dennie, 1962:75) is perhaps more meaningful to modern readers. Fournier also noted very early (1884) that the distinctive morphology of Hutchinson's incisors is rapidly worn away and is generally not obvious after age 30 years. Estimates of the prevalence of Hutchinson's incisors in samples of children and young adults with congenital syphilis range widely, averaging ~33% (see Table 2). Anderson (1939) has described an open-bite malocclusion as a consequence of the incisor malformations.

Moon's molars or mulberry molars or Pflüger molars (Pflüger, 1924) are first permanent molars with similar deformities. There is considerable variation among sources in describing these molars and considerable confusion among the secondary citations of these descriptions; hence the plethora of eponyms and outright errors (for example, see Kieser, 1985). Moon's initial description is skimpy (Table 1), but his illustrations are excellent (Moon, 1877). He speculates that the dental features of congenital syphilis may be largely a consequence of mercury therapy. Other descriptions of the molars in congenital syphilis appear in Table 1. Estimates of frequency of mulberry molars in samples of children or young adults with congenital syphilis range widely, averaging ~27% (Table 2).

Permanent canines are mentioned in several discussions of the dental effects of congenital syphilis, but the changes are less distinctive than in the upper central incisors and first molars (Hutchinson, 1858; Fournier, 1884). Bradlaw (1953:147) provides a useful description: "The canine may show a circumferential groove near the point of the crown which is similarly lost from attrition, leaving a shallow notch." Stoll (1921) describes the syphilitic canine as follows.

> The hypoplastic tip of the canine sometimes suggests the tip of a kernal of corn, both in shape and in its yellow color, while at other times its terraced appearance resembles a Burmese pa-

TABLE 1. Descriptions of first permanent molars in congenital syphilis

The first permanent molars are exceedingly prone to be smaller and more dome-shaped than usual (Moon, 1877:241)
Here the malformation consists of a true atrophy of the cusps of the tooth. . . . The body of the tooth for two-thirds or three-fourths of its height, is in a normal condition; but its upper segment, on the contrary, is lessened in all its diameters—atrophied, eaten, as it were; separated by a circular furrow, as though it were set in. At first sight one would say it was a smaller tooth growing out of a larger one, or better still "a stump of dentine emerging from a normal crown." . . . The masticating surface of the tooth, instead of being neatly divided in a series of tubercles or cusps separating the undulated depressions, presents an irregular appearance, bristling with roughened elevations, granular or acuminate, filled with sinuousities, more or less deep, some of which penetrate to the dentine. Furthermore, this surface, in place of the pearl-like color which distinguishes the normal tooth, has a dirty yellow or brown tint. . . . Under the influence of mastication the grinding surface, abnormally constituted and partially deprived of enamel, wears away, and there remains a tooth doubly remarkable. First, because it is notably shortened; second, because it ends in an absolutely flat surface—a true *plateau,* with a yellowish center and a peripheral border of white enamel (Fournier , 1884:19–20)
[The base of the crown is of normal breadth, while the crown at the chewing surface is narrowed, such that the cusps are underdeveloped. This gives the impression that the crown of the tooth is not developed to its full size. I have given this kind of tooth the name "budform." In both the cross section and the longitudinal section the tooth morphology differs from that of a normal tooth. Whereas the cross section of normal molars is oblique-angled, forming a square, the cross section of molars showing syphilitic changes approximates a circle. While the normal molar has its smallest diameter at the neck, and its greatest width at the cusps, in this form of tooth these are reversed: the base of the tooth has the larger diameter.] (Pflüger, 1924:606)
The true mulberry molar of syphilis is a first permanent molar characterized by enamel cusps showing crests of sound enamel on a base of hypoplastic deposits. These cusps are generally crowded together on a crown surface of dwarfed dimensions . . . The so-called "extra tubercle" on the mulberry molar of syphilis is formed by the buckling or telescoping of normal enamel layers over dwarfed and hypoplastic formations. While it may be a common feature of the syphilitic molar, this structure can be found in any severe hypoplasia of the first year layers. This tubercle of Carabelli should not be confounded with the true supernumerary cusp of the first molar which is an anomaly of no special significance (Karnosh, 1926:41)
The most characteristic features of the abnormality seen clinically were (1) the undersized, malformed appearance of the teeth, (2) the contracted appearance of the mamillons (sic), marginal ridges and cusps of the incisors, cuspids, and molars and (3) a peculiar open bite malocclusion (Anderson, 1939:57)
. . . The Hans Pflüger rosebud molars, the raspberry or multiple cusped molar of Henri Moon and the Mayan molar of Dennie, in which the process of enamelization stops before the dentine is entirely covered (Dennie, 1962:75–76)
The first molar typical of congenital syphilis . . . the so-called dome-shaped or bud-formed molar, is considerably smaller than the normal first molar and smaller than the adjoining second molar. Its most important characteristic, however, is reduction in size of the crown towards (sic) the masticatory surface, and particularly in the mesiodistal direction. In the labiopalatinal or labiolingual direction, the narrowing often occurs in nonsyphilitic first molars. The breadth of the bud-formed molars at the middle of the crown is on an average 85 per cent of the normal but at the masticatory surface only 62 per cent . . . (Putkonen, 1962:51–52)
The shape of the permanent first molars is altered in about 30 percent of patients with congenital syphilis. The occlusal surfaces are much narrower than normal so that they appear pinched. The teeth also show hypoplasia of the enamel and are called *mulberry molars. Pflüger molar* is identical to the mulberry molar except that hypoplasia is not present (Bhaskar, 1965:109)
The crowns of the first molars . . . are irregular, the enamel of the occlusal surface and occlusal third of the tooth appearing to be arranged in an agglomerate mass of globules rather than well formed cusps. The crown is narrower at the occlusal surface than at the cervical margin (Shafer et al., 1974:51)

goda. When the hypoplastic part has crumbled away, a puckered appearance results as though the edge had been drawn up with a shirring string (Stoll, 1921:920).

Histology of the teeth in congenital syphilis has been inadequately described and does not aid in evaluating skeletal remains. Karnosh (1926) largely described his sections in gross terms as a severe chronologic hypoplasia in the first months of life. Burket (1937) and Bauer (1943) described changes in the ameloblasts and pulp but not in hard tissues. De Wilde (1943) reported nonspecific changes in dentin. Sarnat and Shaw (1942) found both enamel and dentin to be normal in histology, with all deformities attributable to shape changes at the dentinoenamel junction. Bradlaw (1953) suggested that degeneration of the central ameloblasts produces the notches in Hutchinson's incisors, and reported areas of amorphous enamel along the dentinoenamel junction. The bulk of this literature describes changes that are nonspecific and that overlap with other causes of enamel hypoplasia (Kreshover, 1960).

TABLE 2. Frequency estimates of dental signs of congenital syphilis from clinical sources

Source	Age range	N	Hutchinson's (%)	Moon's (%)
Cannon, 1927	3 wk–44 yr	202	10	16
Quinlan, 1927	5 wk–16 yr	50	22	8
Jeans and Cooke, 1930	Child > 2 yr	707	11	—
Stathers and Skidmore, 1932	4 yr–40 yr	105	15	—
Stokes, 1934	—		29	3
Brauer and Blackstone, 1941	9 mo–17 yr	38	63	37
Johnston et al., 1941	1 yr–15 yr	34	53	38
Sarnat and Shaw, 1942	Child > 7 yr	57	30	—
Fiumara and Lessell, 1970[1]	Mean 30 yr	271	63	65
Putkonen, 1962	—	254	45	22
Putkonen, 1963	Treated	36	28	17
Putkonen and Paatero, 1971	—	42	31	—

[1] Fiumara and Lessell (1970) report the highest values, but there are several internal inconsistencies in their tables rendering these figures suspect.

Hutchinson and other early writers expressed some reservations regarding whether the dental signs of congenital syphilis might be produced by mercury treatment of syphilis rather than the disease itself. Since 1945, mercury and other heavy metals have been replaced by antibiotic therapies. Modern accounts attribute the dental signs to a specific feature of untreated congenital syphilis: the severe stomatitis, called *snuffles* in older sources, that develops during the first year of life. There is evidence that dental lesions do not develop until the third or fourth month after birth (Bernfeld, 1971; Putkonen, 1963). Hypoplasia of the deciduous teeth in children with congenital syphilis was noticed as early as 1877 (Coles, 1877), but these changes appear to be nonspecific (Burket, 1937; De Wilde, 1943).

MATERIALS AND METHODS

The Newton Plantation collection consists of dentitions of about 104 individuals excavated in 1971–1973 by Handler and Lange (1978), who describe the living conditions and mortuary practices of slaves on Barbados during the late seventeenth through early nineteenth centuries. Age determination, demography, and dental health have been described elsewhere (Corruccini and Handler, 1980; Corruccini et al., 1982, 1985, 1987a,b, 1989; Handler et al., 1982, 1986; Handler and Corruccini, 1983, 1986).

We (K.P.J., D.C.C.) examined the dental remains for gross evidence of congenital syphilis. Each permanent incisor and first molar was coded as normal, affected, worn, lost antemortem, or absent. Frequency data are developed from the count of teeth judged normal and affected; that is, teeth too worn to score are eliminated. First molars or incisors from 84 individuals could be scored. Descriptions of individuals meeting the criteria developed from our search of the literature on congenital syphilis appear below.

RESULTS

Three specimens from Newton Plantation show evidence of congenital syphilis in the form of Hutchinson's incisors and/or Moon's molars. Two specimens, while lacking these distinctive lesions, have anomalies in shape and size that suggest a similar pathological process. An additional 12 cases (6A, 20, 24, 26A, 33, 38A, 60, 67, 69, 79, 81, 85) show antemortem loss of first molars, which *may* have been the result of caries in hypoplastic defects of congenital syphilis, or, of course, of other causes.

Description

NP 53: Moon's molars.

This incomplete dentition was recovered in three field units in the most disturbed and commingled area of the cemetery: *NP 50* and *NP 57,* isolated maxilla fragments; and *NP 53,* the disturbed partial skeleton of an adolescent female (Handler and Lange, 1978). We have combined these units because they represent a single individual. Size, age, and wear facets correspond in all details (Fig. 1). The mandibular dentition is

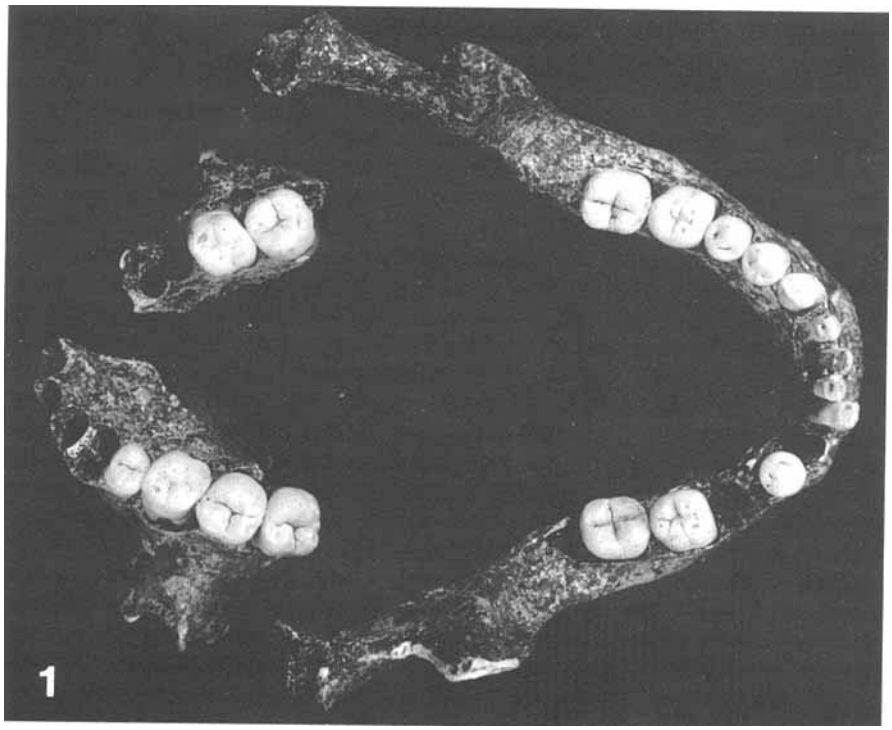

Fig. 1. *NP 53*, occlusal view.

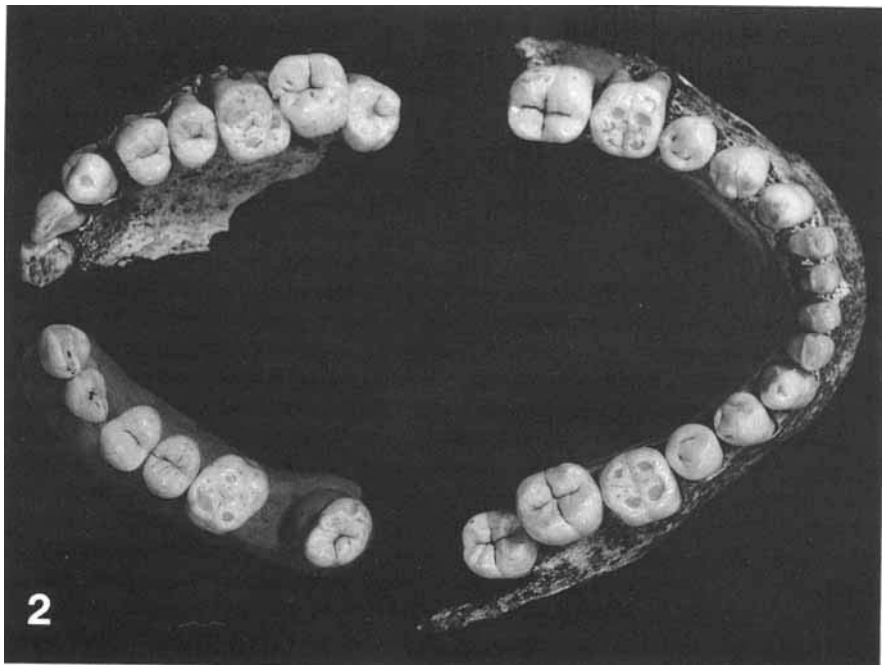

Fig. 2. *NP 54*, occlusal view.

complete apart from the right canine and second premolar. There is agenesis or failure of eruption of the mandibular third molars. The maxillary dentition is represented by the right second premolar through third molar and the left first and second molar. Enamel has exfoliated postmortem from the interproximal surfaces of the incisors, the buccal surface of the canine, and the lingual and interproximal surfaces of the lower left first molar.

The lower incisors are narrow and straight sided. A diastema separates the left lateral incisor and the canine. Occlusal edges are worn flat. There is pitting hypoplasia of the occlusal one-thirds of the right incisors and linear enamel hypoplasia on all four, resulting in a constriction of the middle one-third of the crowns. Distinctive features of Hutchinson's incisors are absent, but wear is more advanced than one would expect from the remainder of the dentition.

The upper first molars are reduced in all crown dimensions with respect to the adjacent teeth. The cusps of the trigone (protocone, paracone, and metacone) are small and clustered toward the center of the occlusal surface. The enamel on the cusp tips is infolded. Dentin is exposed on the paracones. The hypocones are more normal in size and shape. On the left, the hypocone is blunt and rounded. On the right, a cluster of small pits depresses the lingual surface of the hypocone. Both maxillary first molars show multiple episodes of linear enamel hypoplasia. Deep, ill-defined LEH in the middle thirds of the crowns produces a waisted appearance.

Cusps of the mandibular first molars are small and clustered centrally. Dentin is exposed on the tips of the mesial cusps. Cuspal enamel is thin and infolded. There are zones of pitting hypoplasia on the buccal aspect of the protoconid and hypoconid. There is no visible LEH on the lower first molars.

NP 54: Hutchinson's incisors, Moon's molars, and unusual canines.

The dentition of this young adult female (Fig. 2) is complete except for the left mandibular third molar and the right maxillary canine and second molar. Some enamel has exfoliated postmortem on the four mandibular incisors, the two maxillary central incisors, the maxillary right lateral incisor, and the maxillary left second molar, resulting in the loss of the mesial and distal margins of these teeth. A portion of enamel has also been lost on the buccal aspect of the first left maxillary molar.

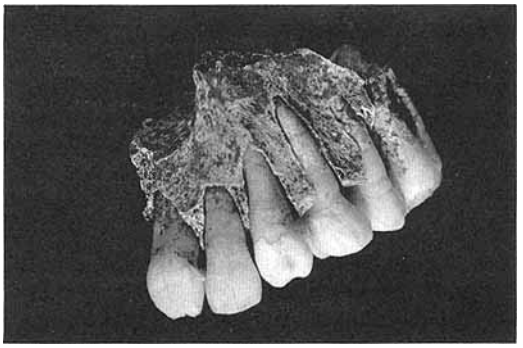

Fig. 3. *NP 54*, buccal view of maxillary I1 through M1. Note occlusal notch in central incisor and canine.

The maxillary central incisors of this individual are bulbous and small, creating gaps or diastemas of at least 2 mm between the teeth. The central incisors constrict and narrow toward the occlusal edge. A faint crescentic notch on the occlusal surface (Fig. 3) is partially obliterated by wear.

The maxillary lateral incisors are more normal in their appearance in this individual. They are less bulbous in labial/lingual profile than are the central incisors. A faint tuberculum dentale is accentuated by an LEH episode in the middle one-third of the tooth. The lower incisors are narrow in mesial/distal diameter and widely spaced, but the exfoliated enamel makes it difficult to determine the true width of the teeth or the true size of the gaps between the teeth. There is overbite and overjet, but open-bite malocclusion is absent.

The upper first molars are smaller than the second molars. The cusps of the trigone are reduced in size and are clustered toward the center of the occlusal surface of the first molar. The metacone seems to exhibit a somewhat greater reduction in size than the paracone and protocone. The hypocone is blunt, rounded, and larger than the cusps of the trigone. The trigone cusps exhibit wear

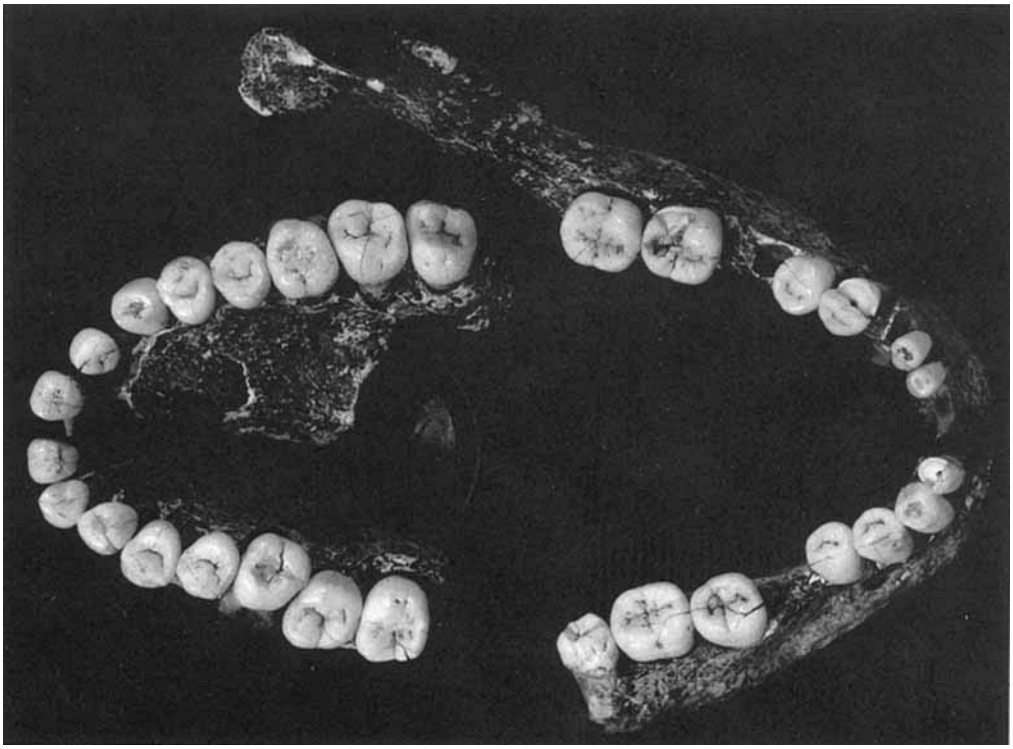

Fig. 4. *NP 75*, occlusal view.

to the dentin because the enamel is very thin, while wear is not evident on the hypocone. Hypoplastic pitting is evident near the occlusal surface of the first molars and may be a result of the thin enamel in this area. An LEH episode is visible on the buccal surface of the right first maxillary molar.

Mandibular first molars are smaller than the second molars. All the cusps on the first molars are reduced in size and are congregated centrally on the occlusal surface of the tooth. The thin enamel is infolded at each cusp, and attrition has exposed dentin prematurely. Hypoplastic pitting is evident throughout the occlusal surface. There is no visible LEH on the mandibular first molars.

Maxillary and mandibular canines are as distinctive in morphology as the upper central incisors. Canines are bulbous column-like pegs with an occlusal notch (Fig. 3) and an elevated ring of enamel on the occlusal surface. Canines are visibly smaller and simpler than usual and exhibit no identifiable mesial canine ridge, distal accessory ridge, or tuberculum dentale. Canines exhibit LEH episodes on all surfaces.

NP 75: Hutchinson's incisors and Moon's molars.

The dentition of this young adult female (Fig. 4) is complete except for the mandibular right central incisor and left canine. There is extensive postmortem fracturing of the enamel and dentin, resulting in the loss of portions of the crowns of the maxillary right canine and the mandibular right I2, left P3, and left M2. The mandibular first molars were lost during life, and both mandibular M2s are carious. A supernumerary molar is present distal to the mandibular right M3. There is pipewear on the right canines and anterior premolars.

Maxillary incisors are bulbous, small, and widely spaced. The central incisors are peg-like, conforming to the pumpkin-seed shape

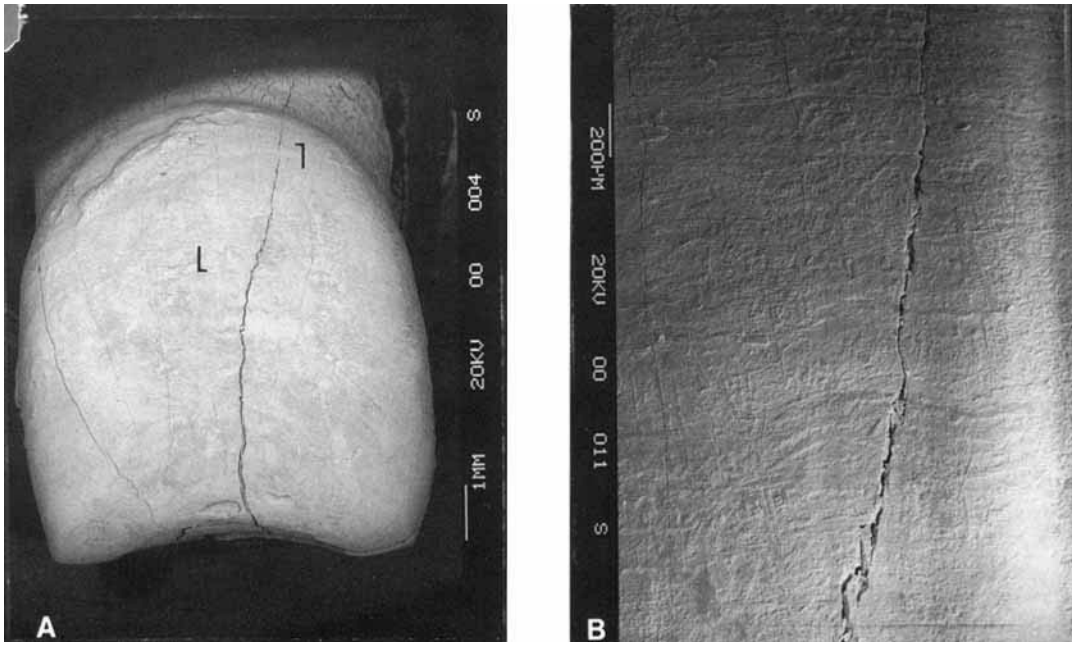

Fig. 5. SEM images of left central maxillary incisor, *NP 75*. **A:** Labial view showing crescentic notch in occlusal aspect and irregular surface. Irregular mass near CEJ is calculus. Scale is 1 mm. **B:** Detail of highly irregular perikymata (outlined section of A). Scale = 200 µm.

described by Dennie (1962). The occlusal surfaces are quite constricted. Shallow occlusal notches are paralleled by weakly defined perikymata on the labial surface of the central incisors. The perikymata are tightly curved, following the contour of the notch (Fig. 5). Occlusal enamel is very thin, and dentin is exposed (Fig. 6). The maxillary lateral incisors are more normal in appearance, but they are peg-like and have large lingual tubercles. The lower incisors are narrow, with a constricted occlusal surface and faint notches. There is an elevated ring of enamel on their occlusal surfaces.

The maxillary first molars are small in comparison to the adjacent teeth and are unusual in shape. The cusps of the trigone are constricted in size, crenulate, and more centrally placed on the occlusal surface than normal. The metacone is especially reduced. Cusp tips are infolded and the central foveas are depressed. The hypocones are relatively normal. They are large and bulbous in comparison to the cusps of the trigone. The result is a marked reduction in the buccal one-

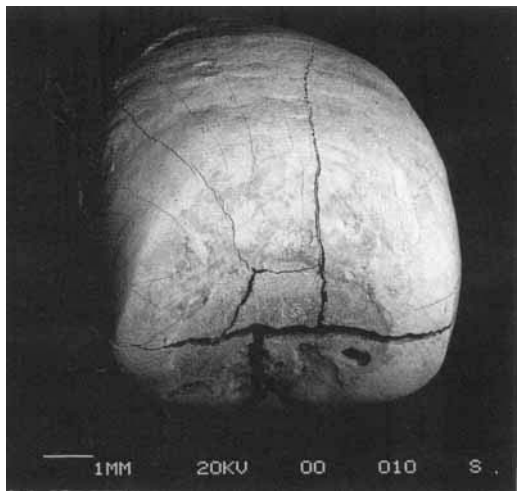

Fig. 6. Oblique view of labial and occlusal surfaces, left central maxillary incisor, *NP 75*. Note extreme curvature of perikymata, thin enamel, and cupped worn surface of exposed dentin. Scale = 1 mm.

half of the crown. On the right, this contributes to a malocclusion that displaces

the M1 lingually. The crown height also is reduced in comparison with the adjacent teeth. There is a small carious lesion in the occlusal surface of this tooth.

The mandibular right M2 is markedly smaller than the left and has a deep central fovea and rounded buccal cusps. Pitting hypoplasia appears on the occlusal one-thirds of the maxillary P3s, and linear enamel hypoplasia is present in the cervical one-half of the canines. Other hypoplastic lesions are faint.

NP 43: Borderline features.

The occlusal surface of the mandibular first molar is small in comparison with the second molar but is otherwise normal in morphology. The cusps of the first molar are not inset from the buccal and lingual surfaces of the crown, as is typical of Moon's molars.

NP 82: Borderline features.

Incisor and first molar crowns are compressed, with small occlusal surfaces as compared with the midcrown diameters, but are otherwise normal in morphology.

Morphology and occlusion

In our examples, the maxillary first molars are quite distinctive in form. The cusps of the trigone are markedly reduced and crowded, whereas the hypocone is comparatively normal. The first molar is ordinarily trapezoidal, with the paracone, or mesiobuccal cusp, accounting for the most prominent and most acute angle. In our examples of Moon's molars, this pattern is inverted, and the hypocone occupies the most prominent and acute angle (see Figs. 1, 2, 4, and 6). We speculate that this change in shape reflects the relatively late calcification of the hypocone and the late union of the hypocone with the coalesced cusps of the trigone revealed in Kraus' (1963) painstaking studies of the formation of the maxillary deciduous second molar.

This unusual morphology produces a distinctive malocclusion of the maxillary first molars that is visible in all three of our cases. The first molar is displaced lingually along the angled contact facet between M1 and M2. In addition, the M1 is tipped, so that the occlusal surface faces somewhat lingually. In one case (*NP 54*) the second molar is displaced buccally as well. Malocclusion in the lower jaw is less pronounced, but, in both cases in which mandibular first molars are present, these teeth are displaced buccally, and the second premolars are rotated. Neither the relatively normal size of the hypocone nor the resultant malocclusion has been previously described in the literature we have consulted. However, an illustration in an early paper (Johnston et al., 1941) shows a similar malocclusion.

Frequency at birth

The demography of the Newton Plantation collection has been presented in earlier studies (Corruccini et al., 1982, 1989; Handler and Lange, 1978). Although it is difficult to make demographic or epidemiologic inferences from three cases of congenital syphilis, it may be surprising that we have no cases in children under age 5 years. However, permanent incisors or first molars were recovered for only one child under age 5 years and 16 children between ages 5 and 18 years; hence this deficiency is an artifact of mortuary practices, preservation, or recovery methods.

All three cases are in young adults or adolescents. This is not unexpected, because severely hypoplastic teeth such as those showing congenital syphilis would be lost due to caries or worn beyond recognition in older individuals.

Age-specific frequency in adults is likewise difficult to determine. Several discussions of the dental signs of congenital syphilis stress that wear and caries-related loss erase the evidence of this disease in adults older than age 30 years or so. Eighty-four individuals had either incisors or first molars that were sufficiently unworn to permit evaluation for signs of congenital syphilis. Our three cases constitute 3.8% of these individuals. This figure represents a minimum frequency for this population, both because other affected teeth may have been lost due to wear or caries and because there may be unrecognized multiple entries for unaffected individuals like that represented by *NP 50, NP 53,* and *NP 57*. This problem is

a minor but unavoidable one that hampers any frequency calculations in paleoepidemiology. We recognized that these three disturbed units were in fact one individual only because we saw the similarity in the pathological changes in the teeth. One-half of burials at Newton Plantation show significant disturbance (Handler and Lange, 1978); hence the true frequency might double in the most extreme, and rather unlikely, case.

When frequency is evaluated on a per-tooth basis, results range from 5% for right lower M1 to 9% for left upper M1, with values for the various permanent incisors falling within this narrow range. Per tooth sample sizes are small because of poor recovery, antemortem loss, and wear. The range is 32–51 specimens per tooth. If the two borderline cases are included, these minimum frequency figures will rise accordingly.

The clinical literature shows that the dental features of congenital syphilis are present in about one in three cases (Table 2); hence we can expect approximately nine of the 84 scoreable individuals at Newton Plantation, or approximately 10%, to have suffered from this disease.

Enamel hypoplasia frequencies

Perhaps the most interesting previous work with the Newton Plantation dentitions is the evidence for ill-health provided by enamel hypoplasias (Corruccini et al., 1982, 1985; Handler and Corruccini, 1986). These studies focus on weaning and nutrition as proximal causes for disturbances in enamel development. Our identification of congenital syphilis in these remains raises a question: To what extent can the remarkable frequency and severity of enamel hypoplasias in the Newton Plantation series be attributed *specifically* to congenital syphilis? To answer this question, we reanalyzed the data presented in the papers cited above.

We are able to identify three cases of congenital syphilis through dental lesions at Newton Plantation. These three cases account for an astonishingly disproportionate share of the enamel hypoplasias in this series (Table 3). Our three cases have an average of 7.0 episodes of disturbed enamel development per person, whereas individuals without the dental signs of congenital syphilis have 2.18 episodes per person. This difference is not evenly distributed across types of developmental disturbance recognized by Corruccini and coworkers (1985). Individuals with dental signs of congenital syphilis are 2.5 times more likely to exhibit low-to-severe levels of linear enamel hypoplasia than those without, twice as likely to exhibit extremely severe hypoplasia (MGA), and 7.5 *times* more likely to exhibit pitting hypoplasia. When we consider that the cases of congenital syphilis we are able to recognize through dental features are only the tip of the iceberg, representing perhaps one-third of those with congenital syphilis (Table 2), we must conclude that the extremely frequent and severe hypoplasia present in this series is largely attributable to this disease. The 74 individuals with apparently normal dentitions include perhaps half a dozen victims of congenital syphilis in whom the teeth were not affected with recognizable lesions of this disease. However, these individuals will have experienced poor health throughout the period of formation of the permanent dentition visible as linear or pitting hypoplasia. Although weaning stress is demonstrable in the *timing* of disturbances of enamel development in this population (Corruccini et al., 1985; Handler and Corruccini, 1986), we believe that the *severity* of these disturbances is substantially the result of a specific infectious disease.

TABLE 3. Mean number of disturbances of enamel development per person in the Newton Plantation series[1]

	N	LEH	MGA	Pit	Total
Congenital syphilis	3	3.67	.66	2.67	7.00
Normal dentition	74[2]	1.47	.34	.36	2.18
Combined	77	1.90	.35	.45	2.36
Ratio CS/ND		2.49	1.94	7.42	3.21

[1] Categories for coding disturbances of enamel development are those of Corruccini et al. (1985:701). LEH refers to episodes of linear enamel hypoplasia considered by most observers to include the grades mild, moderate and severe. MGA refers to wide bands of hypoplastic enamel that are deeply inset with respect to the tooth surface and represent unusually severe episodes of linear enamel hypoplasia. Pit refers to horizontal bands of discontinuous, pitted depressions in the enamel surface.

[2] Two individuals in the original sample of 104 are combined with NP 55; an additional 18 were not included in this analysis because of advanced wear, antemortem tooth loss, or incomplete recovery. The 77 individuals represented in this table consist of those meeting scoring criteria for both Corruccini et al. (1985) and the present study.

DISCUSSION

The implications of this study for the ongoing scholarly debate about slave health are substantial. If perhaps nine of 96 slaves in the Newton Plantation series suffered from congenital syphilis, we can attribute much of the high infant mortality in this population (Handler and Lange, 1978) to this disease as well. Under the far better health care conditions that the U.S. general population enjoyed during the first one-half of the twentieth century, the infant mortality rate in congenital syphilis was 25–50% (Moore, 1941). The stillbirth rate was as high as 25% (Curtis and Philpott, 1964). These data reflect the use of various effective heavy metal compounds in treating syphilis but predate the introduction of penicillin; hence we can expect the mortality experienced by the Newton Plantation slaves to have been even higher. The 10% or so of the Newton Plantation burials who reached adolescence or young adulthood with congenital syphilis represent a far larger cohort lost before birth or in the first year of life, who are unrepresented in our sample. Adult mortality and disability due to acquired syphilis will have been a major problem for the Newton Plantation population as well. In addition, the remarkable infertility of slaves on Barbados (Dirks, 1978) may well have been due in part to syphilis.

Historical records for the plantation fail to shed any light on these issues, both because diagnostic labels of this era are unreliable and because yaws and syphilis are discussed only as chronic problems, not as causes of death. However, congenital and acquired syphilis could contribute to most of the causes of death listed in the plantation records. Such causes as "consumption, convulsed, dropsy, fever, fits, inflammation, invalid, joint evil, leprosy merasmus [sic], rheumatism, scrofula, sore throat, teething," and "worms" (Handler and Lange, 1978:99) could easily include the diverse symptoms of acquired and congenital syphilis.

Higman's (1984) broader study of mortality in Caribbean slaves is likewise uninformative on these issues. However, Higman points out that reports of yaws deaths are largely confined to children under 10 years of age in the historical records for the Caribbean. Prior to the 1940s, there were no objective means for distinguishing among the treponematoses (Quétel, 1990). Because there is no appreciable mortality in childhood yaws (Grin, 1956), these accounts may refer to skin and bone lesions of congenital syphilis. It is highly unlikely that they refer to yaws in the modern sense. Swados (1941:471) comments on the rarity of references to syphilis among slaves in medical journals published in the American South: "Like tuberculosis, syphilis is a 'white man's disease' unknown to the Africans when they were first brought here." Several historians have argued that much of the so-called syphilis in slaves was actually yaws and that yaws may even have conveyed cross-immunity to venereal syphilis (Parramore, 1970; Kiple, 1984:244). While our data do not permit us to inquire into the origins of syphilis among Barbadian slaves, they reveal that it was common by the eighteenth century. Discussions of the history of the treponematoses in African-American populations will require revision in this light.

In a broader context, historians have discussed slave mortality largely as a direct consequence of poor nutrition, poor living conditions, and high infectious disease load. Neonatal tetanus (Swados, 1941; Kiple, 1984; Dirks, 1978), malaria (Steckel, 1988), respiratory diseases (Gibbs et al., 1980), and malnutrition (Gibbs et al., 1980) have been implicated in the very high infant mortality seen in many slave populations. We argue that congenital syphilis must be added to this list.

Studies of dental conditions in three nineteenth century U.S. African-American samples are of interest here. We have had an opportunity to examine dentitions from the First African Baptist Church series, a free Black community from Philadelphia (Angel et al., 1987), a plantation slave cemetery in South Carolina (Rathbun, 1987), and the Cedar Grove Cemetery from Arkansas (Rose, 1985). In the first two groups, there are no examples of Hutchinson's incisors or Moon's molars, and the general health of these populations appears to be good on most measures. This is not the case at Cedar

Grove. Rose's report on Cedar Grove includes a good example of Hutchinson's incisors in a 10-year-old child (Burial 83). This case has been examined using histological techniques (Marks, 1984). The age-specific pattern of periostitis at Cedar Grove confirms the identification of congenital and acquired venereal syphilis in this population, and congenital syphilis is reflected in both high stillbirths and high neonatal mortality (Rose, 1989).

We examined dental casts from Cedar Grove and discovered Moon's molars and canine abnormalities resembling those in *NP 54* in a Cedar Grove female aged 30–39 years (Burial 94). Rose (1985:114) noted that these molars were abnormal in size and shape but attributed the changes to the regional effects of agenesis. A second adult, a male aged 35–39 years (Burial 89), has notched and somewhat contracted upper central incisors and canines reminiscent of the changes seen in congenital syphilis. The upper first molars appear normal, but both lower first molars are missing antemortem. Rose (1985:109) notes that this individual had generalized periostitis. He presents tuberculosis as a diagnosis, but we might suggest syphilis instead or in addition. A female 30–39 years of age (Burial 47; see illustration in Rose, 1985:78) shows modifications of both the upper central incisors and the first molars that suggest congenital syphilis (Rose, 1985:78). Cranial lesions in this individual that Rose attributes to porotic hyperostosis may reflect syphilitic periostitis. If four of the 80 individuals recovered at Cedar Grove show signs of congenital syphilis, a minimum figure for frequency in this population at birth is 5%. This is roughly comparable to what we observed in the Newton Plantation series. This figure is, of course, subject to the constraints of survivorship, frequency of dental defects, and loss of affected teeth that we have discussed previously and suggests to us an underlying frequency at birth of 15% or more. We underscore Rose's (1985:154) comments on congenital syphilis as an aspect of disease load and infant mortality in this community, and we suggest that syphilis may be even more important than Rose proposes. This overview of three U.S. African-American samples suggests that presence or absence of congenital syphilis may account for much of the variability in health and mortality seen among nineteenth century African-American populations.

An exercise in simulating the infant mortality due to congenital syphilis at Newton Plantation may be instructive here. We have argued that congenital syphilis was present in 10% of the scorable remains recovered from this site. If we assume a 50% neonatal mortality rate from congenital syphilis (Moore, 1941), a conservative assumption given the conditions of slave life, then another nine individuals will have died of congenital syphilis as infants. Only 16 infant deaths are registered in the Newton Plantation records (Handler and Lange, 1978:99). Higman (1984:658) reports age-specific death rates for age groups 0–4 years of 29–50 per 1,000. Infant mortality among Caribbean slaves is little known but may have been as high as 50% and was especially severe during the first month of life (Kiple, 1984:54, 112–114). Although both our sources of data on Newton Plantation clearly display underrecording, the mortality we expect from congenital syphilis clearly more than accounts for the infant deaths for which we have records in this population.

We return to the question with which we began this paper: Why has congenital syphilis been recognized so infrequently by paleopathologists? A major obstacle has surely been the lack of appropriate published examples in our literature and the rather skimpy illustration in the clinical literature, much of it old and obscure. We hope that this paper helps to remedy these impediments. We look forward to reports of other examples that show the dental characteristics of congenital syphilis: reduced dimensions and thin enamel in the permanent incisors and first molars, crowding and infolding of the first molar cusps, notching of the upper incisors, and apical hypoplasias of the canines.

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