THE CHRONOLOGICAL DISTRIBUTION OF ENAMEL HYPOPLASIA IN HUMAN PERMANENT INCISOR AND CANINE TEETH

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Summary—The frequencies and chronology, based on a standard tooth development chart, of enamel hypoplasia derived from permanent upper central incisors and mandibular canines were compared for 42 prehistoric Amerindians. Between 0.5 and 4.5 years, when the crowns of both these teeth are developing, hypoplasias were 1.36 times more common on the incisors (34 hypoplasias/incisor; 40 hypoplasias/canine). Hypoplasias on incisors occurred earlier (mean = 2.50; median at 2.0-2.5 years) compared to the canine (mean = 3.51; median at 3.3-4.0 years). Differences in published frequencies and chronologies of hypoplasias may be explained, in part, by an indefinable variation in the teeth studied. The highest density of hypoplasias on both tooth crowns was just cervical to the midpoint, suggesting that developmental rates and crown geometry may influence the ability of the crown to record stressful events.

INTRODUCTION

Chronologic or systemic enamel hypoplasia is a decrease in enamel thickness resulting from a temporary disruption in enamel matrix formation (Pindborg, 1970, 1982; Sarnat and Schour, 1941; Yaeger, 1980). As a wide variety of nutritional deficiencies and diseases may cause enamel hypoplasias; they can be put in the general category of non-specific physiological disruption (stress; Kreshover, 1960; Pindborg, 1982).

As enamel once developed remains more or less unchanged and in man its time of development can be ascertained, enamel hypoplasia has great potential as an indicator of stress during the prenatal to childhood periods (Sarnat and Schour, 1941). The frequency of enamel hypoplasia has been used to assess the general health status of living (Infante and Gillespie, 1974; Sweeney et al., 1969) and prehistoric populations (Cook and Buikstra, 1975; Goodman, Armelagos and Rose, 1980; Swärdstedt, 1966). The chronological distribution of enamel hypoplasia in populations has been used as evidence for peak periods of stress (Goodman Armelagos and Rose, 1984; Sarnat and Schour, 1941; Schulz and McHenry, 1975; Swärdstedt, 1966).

Sarnat and Schour (1941) presented the chronological distribution of hypoplasias in 60 individuals from the Chicago area. Two-thirds of the episodes of hypoplasias occurred within the first year of life, approximately one-third occurred during the next 22 months and less than 2 per cent occurred between 34 and 80 months. Sarnat and Schour (1941) concluded that this distribution of hypoplasias is more a reflection of a biologically-universal pattern of host resistance, than of the chronological pattern of environmental stressors (Massler, Schour and Poncher, 1941).

If Sarnat and Schour's conclusion is correct, enamel hypoplasia chronologies from other populations should accord with their findings. Their findings have not been repeated for other contemporary or industrial populations but hypoplasia chronologies have been constructed for at least four prehistoric populations. The chronological distribution of hypoplasias in a medieval Swedish population had a peak at two to four years, with low frequencies before two years (Swärdstedt, 1966). Similarly, chronological distribution of hypoplasias in two time-successive ancient American Indian populations from Dickson Mounds had few hypoplasias before two years of age and after four years, with a peak frequency between two and four years (Goodman et al., 1984). Finally, a prehistoric California population showed few hypoplasias in the first two years and increases in frequency to a peak between four and six years (Schulz and McHenry, 1975).

The differences in enamel hypoplasia chronologies do not support Sarnat and Schour's (1941) contention referred to above. Rather, these data support the view that hypoplasia chronologies are a reflection of different chronological patterns of environmental stressors affecting these populations. Still un-evaluated, however, is the potential role of methodological factors as causes of the differences and similarities observed among these hypoplasia chronologies.

Our purpose was to determine whether upper-central incisors and lower canines, the two teeth which are most often studied (Rose, Condon and Goodman, 1985) and most frequently hypoplastic (Goodman et al., 1980), differ in susceptibility to hypoplasia and yield different chronologies.

MATERIALS AND METHODS

The sample is derived from the Dickson Mounds, a multicomponent habitation and burial site (A.D. 950-1300) located near Lewiston, Illinois (Harn, 1980). In a study of 111 individuals with permanent dentitions, Goodman et al. (1980) found that 73 (65.8
per cent) had one or more systemic enamel hypoplasias. Forty-two individuals with at least one intact and unworn upper-central incisor and mandibular canine were included in our study.

Hypoplasias were defined as circumferential lines, bands or rows of pits of decreased enamel thickness (Plate Fig. 1). Using a binocular microscope and dental probe, these crown-surface lesions were easily distinguished from other surface irregularities.

In order to determine the age of the individual when the lesions developed, their distance from the cementum-enamel junction was measured. These measurements were converted to a half-year period by reference to the chronology of enamel-crown development of Massler et al. (1941), slightly modified by Swärstedt (1966). This method involves the division of the incisor crown into 9 (birth to 4.5 years) and the canine crown into 12 (birth to 6.0 years) half-year developmental periods.

RESULTS

There were more hypoplasias on the incisor than on the canine. Of the 378 half-year periods (9 periods times 42 teeth) in respect of incisors, 54 were hypoplastic (1.29/incisor; 0.14/half-year interval). Of the 504 half-year canine periods (12 periods times 33 teeth), 46 were hypoplastic (1.10/canine; 0.09/half-year period). The significance of the greater frequency of defects on the incisor was tested by applying the Wilcoxon matched-pairs sign test (Siegel, 1956) and yielded a two tailed 

\[ z = 2.21 \]

The two-tailed probability of this difference being due to random variations is near zero (\( p < 0.001 \)), as estimated by the sign test (\( z = 3.47 \)).

The occurrence of hypoplasias in both teeth showed a normal distribution (Text Fig. 2). However, the distributions differed in the place of their means and modal ages. The incisor-chronology mode was at 2.0–2.5 years with a mean of 2.30 years; the canine mode was at 3.5–4.0 years, with a mean of 3.51 years.

The incisor and canine hypoplasia distributions differed significantly even within the common 0.5–4.5-year period. By 3.0 years, 39 of 54 (72.2 per cent) incisor lesions had occurred compared to 12 of 40 (30.0 per cent) canine lesions. The significance of the 42.2 per cent difference between these cumulative frequencies was tested by applying Kolmogorov-Smirnov test for differences in distributions (Siegel, 1956). The 42.2 per cent difference is far greater than the critical value of 29 per cent for rejection of the null hypothesis (distributions are not significantly different) at the \( p > 0.01 \) level of confidence (two-tailed).

For all 12 occurrences of a hypoplasia on a canine before 3.0 years, a corresponding hypoplasia was found on the incisor in the same skull. However, before 3.0 years there were 27 hypoplasias found on the incisor without a corresponding hypoplasia on the canine. Between 0.5 and 3.0 years of age, the incisor yielded over three times the frequency of defects as did the canine.

After 3 years of age, the distribution of defects on these teeth changed. Between 3.0 and 3.5 years there was an equal frequency of lesions on each tooth class. However, between 3.5 and 4.5 years 20 canines had lesions, whereas only 7 incisors had lesions. For this time period, the canine yielded nearly three times the frequency of defects found on the incisor.

![Fig. 2. Percentage of hypoplasias by half-year intervals for maxillary central incisors and mandibular canines (n = 33). Numbers below the graph are frequencies of hypoplasias for each half-year period during which these tooth crowns were developing.](image-url)
DISCUSSION

The greater frequency of hypoplasias on the incisor does not appear to be due to local factors, random variation, or methodological errors. When a hypoplasia was present only one of these two teeth, other teeth were examined to determine whether the defect might be due to a local factor. It was decided that one defect was due to local trauma and it was not included in the analysis. Attrition is the only factor, we are aware of, which could limit our ability to record defects. However, we excluded all teeth from study in which attrition had begun to wear into the incisal edge (beyond stage 2 attrition; Brothwell, 1981).

Differences in the frequency of defects by tooth have been noted (Cutress and Suckling, 1982; El-Najjar, DeSanti and Ozebek, 1978; Pindborg, 1970). In general, the anterior teeth in series are mostly hypoplastic. Because they are the earlier developing teeth, these differences are usually attributed to the difference in time of mineralization (Pindborg, 1970; Yaeger, 1980). In our study, however, variation was found while both tooth crowns are mineralizing. Thus, we propose that variations in frequency may also be due to differential susceptibility of tooth crowns to hypoplasias.

The reason why teeth might be differentially susceptible to hypoplasias can only be speculated on. One possible explanation is that teeth which are most stable in developmental timing, the teeth at the polar centres of dental fields (Bailit, 1975; Butler, 1939; Dahlberg, 1945), are most susceptible to disruption. Teeth, in which the genetic control over development is strongest, may be more likely to be hypoplastic because they are less able to alter developmental timing. In a sense, different teeth may have different developmental reactions to stress, depending upon the degree of genetic control over their development. If this hypothesis is correct, recognition of variations in susceptibility of enamel to developmental disturbance should aid in understanding patterns of genetic control over and evolution of the human dentition.

Between-tooth differences in the chronology of defect could be partly related to systemic error in the mineralization standard and its application to prehistoric Amerindian teeth. However, the majority of the difference in chronology is not reasonably attributed to the method. The choice of the Massler et al. (1941) standard is justified for a variety of reasons. First, it has been used in previous studies, therefore, its use allows for direct comparisons. Secondly, this standard is in general agreement with other mineralization standards; thus large errors are not to be expected (Gustafson and Koch, 1974). Thirdly, it contains data on the development of the anterior-permanent teeth before 3 years of age. Information on the early stages of development of these teeth was critical to our study, but is infrequently found in studies based on serial radiographs. Finally, the modification of Swärdstedt (1966) accounts for variation in the rate of mineralization of tooth crowns (Moorrees, Fanning and Hunt, 1963a) by allowing for differences in the amount of enamel which is equivalent to a half-life interval.

Although teeth differ in their chronology of enamel hypoplasias, the distribution of defects is remarkably similar in regard to the position of the defects on tooth crown. For both the incisor and canine, there is a low frequency of defects in the incisal third and gingival sixth of the crown, and a high frequency in the remaining half of the crown. The peak frequency is near the midpoint of the crown. The enamel prisms at this point on the tooth crown are most nearly perpendicular to the surface. This orientation may render it easier to discern variations in rod lengths. Gleiser and Hunt (1955) and Moorrees et al. (1963a, 1963b) noted that there is an increase in velocity of crown development at this time. The greater rate of development may render the tooth more vulnerable to disruption.

These data may help to explain variations in results of studies which use different teeth. For example, among the prehistoric hypoplasia chronologies, the late-peak frequency in the California population may be due to Schulz and McHenry's (1975) exclusive use of mandibular canines. The California chronology is more similar to the Dickson Mound chronology for the same tooth (Fig. 1).

Our findings warn against comparing results of enamel-hypoplasia studies which do not use the same teeth. In order to begin to understand the importance of host resistance and environmental factors in the aetiology of hypoplasias, differences in susceptibility of various teeth to hypoplastic disruption should be ascertained. Once these sources of variation are known, teeth may prove to be better recorders of previous physiologic stresses than has hitherto been realized.

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REFERENCES


Plate 1.

Fig. 1. Chronologic enamel hypoplasia on the maxillary incisors and canine in a prehistoric Amerindian. Note that the position of the hypoplasia on the teeth suggests a common time of formation around 3 years of age.
Plate 1.