1981

An Analysis of Hypoplasia and Hypocalcification in Deciduous Dentition from Dickson Mound

Michael L. Blakey
University of Massachusetts - Amherst

Follow this and additional works at: https://scholarworks.umass.edu/anthro_res_rpt20

Part of the Anthropology Commons

Retrieved from https://scholarworks.umass.edu/anthro_res_rpt20/5

This Article is brought to you for free and open access by the Anthropology Department Research Reports series at ScholarWorks@UMass Amherst. It has been accepted for inclusion in Research Report 20: Biocultural Adaptation Comprehensive Approaches to Skeletal Analysis by an authorized administrator of ScholarWorks@UMass Amherst. For more information, please contact scholarworks@library.umass.edu.
AN ANALYSIS OF HYPOPLASIA AND HYPOCALCIFICATION IN DECIDUOUS DENTITION FROM DICKSON MOUND

Michael L. Blakey
Department of Anthropology
University of Massachusetts

Teeth are the most tenacious human tissues and are well preserved in the archaeological record. Systemically affected by metabolic stress elsewhere in the body, teeth are useful as indicators of the general status of prehistoric human health.

Dental defects, resulting from cessation of enamel-calcium apposition caused by metabolic insult, provide an immutable record of metabolic stress. Defects occur only in the transverse ring of enamel being laid down at the time of upset. The period during which individuals endure systemic insult can be discerned by this evidence and, since teeth do not remodel upon recovery from insult, defects remain unaltered. The present study is an analysis of two commonly found defects—enamel hypoplasia (linear grooves and pits) and hypocalcification (linear discoloration) in deciduous dentition of a prehistoric population from Illinois.

The sample was recovered from the Dickson Mound Site in Illinois, and consisted of skeletal remains of an Amerindian population that lived between the tenth and fourteenth century. Differences were observed in the incidence of enamel defects and associated metabolic stress occurring from the fifth month in utero to the tenth to twelfth month of life.

Clinicians and skeletal biologists have studied hypoplastic defects in the dental enamel of a number of species (see Molnar and Ward 1972:9 for a review of primate studies). Both interpopulation and age differences in stress characteristics have been identified. Though the etiology of hypoplastic defects is not completely understood, research in this area suggests an association with stress. These studies have pointed consistently toward
a much higher frequency of hypoplasia in nutritionally stressed than nutritionally sufficient groups. Sweeney's (1969) study of malnourished Guatemalan village children recorded hypoplasia among 45 percent of the individuals observed. Infante (1975) reports an incidence of 19.4 to 38.9 percent for a group of nutritionally stressed Apache school children. Sarnat and Schour (1941) discovered a high incidence of hypoplasia among nutritionally stressed American children, with two-thirds of defects occurring during infancy.

Enamel defects appear to indicate severe nutritional and/or disease stress. Deficiencies of vitamins (such as A, C, and D) can result in arrest episodes in calcium-enamel formation in two ways: either by causing degeneration or atrophy of ameloblasts responsible for enamel apposition, or by decreasing available calcium to ameloblasts. Infectious disease stress and compounding nutritional deficiency often have a synergistic effect which has been closely associated with enamel defects (Sweeney et. al., 1969).

The association between hypoplasias and metabolic insult, together with their permanence has made these useful for analysis of health and disease in archaeological populations. Swardstadt (1966) in an excellent study of enamel defects in skeletal material from a medieval Swedish site found hypoplasia frequency differences emerged from social strata. He found the highest incidence among the Swedish "slave" population and the lowest among "landowners," with "peasants" intermediate. Hypoplasia and hypocalcification are present even in the fossil record. Defects in hominids from Swartkrans and Sterkfontein with an incidence (per dental sample) of 17.1 and 8.4 percent respectively has been reported (White, 1979).

Hypoplasia research with archaeological populations support the expected relationship between hypoplasias and metabolic stress, either between social strata or cultural groups and modes of subsistence (Sciulli, 1976; Goodman et. al., 1980). However, where frequencies of hypoplasia have been plotted for age groups using adult skeletal remains, results differ from clinical data.

Schulz and McHenry (1975:913), in a study of prehistoric California Amerinds, detected no hypoplasias during the first year and found the highest frequency occurring between the ages of four and five. Sarnat and Schour (1941) in clinical study which appears to include deciduous teeth found the highest incidence of hypoplasia during the first year. Schulz and McHenry and others suggest that the disparity between these clinical and skeletal results is a function of early mortality for stressed infants (Schulz and Mc Henry, 1975; Goodman et. al., 1980). Consequently, fewer infants would have lived long enough to have infancy derived enamel defects.
in adult dentition and would therefore be under represented.

Environmental stress differences across cultural horizons at Dickson have been repeatedly demonstrated by investigators using a number of pathology indicators. Goodman, Armelagos, and Rose (1980) have demonstrated an increase in hypoplasias of the permanent dentition from the Late Woodland Period to the Middle Mississippian Period at Dickson. The increase in hypoplastic episodes, they feel, is due to an increase in nutritional and disease stress with the appearance and intensification of maize agriculture: concurrent with increase population pressure, dependency on maize, and protein insufficiency. Using permanent dentition from Dickson Mound, they observed hypoplasias which had occurred during the formation of these teeth in childhood. Results indicate lowest frequency during the first one and one-half years of age. Their study also shows that stressed individuals display an increase in mortality.

The present study has been conducted in part to ascertain the validity of the "stressed infant attrition hypothesis" for under-representation of infancy-derived defects in adult dentition. Deciduous dentition (the later development of which overlaps early adult enamel formation) was used. In addition, analysis revealed the systemic maternal, prenatal, and infant stress for combined cultures at Dickson.

**Materials and Methods**

Dental remains of 129 sub-adults from the Dickson Mound Site were available for study at the University of Massachusetts. Of these, fifty individuals between one and twelve years of age (with fully-developed crown enamel) were of sufficient preservation, and selected for study.

A sliding needle caliper was used to measure from the dentino-enamel junction (or inferior border of completely developed enamel) to the center of the hypoplastic or hypocalcified line (if these were no more than 1.5 mm in width). Wider linear defects (usually hypocalcification) were measured to their superior and inferior borders. In this way, the entire period of pathological enamel formation was recorded from onset to conclusion, respectively. This technique has not been used in previous studies. It is appropriate to the analysis of hypocalcification which often persists over several months of enamel development. That method commonly used (measuring to the middle of the defect) is probably accurate for defects such as hypoplasias which are thinner.
After all measurements of defects were taken, these were repeated on one-half of the dental arcade to enhance accuracy. During the second set of measurements each crown height was ascertained for tooth-specific timing of defective segments. Other data recorded included the color of the hypocalcified stain, the type of hypoplasia (pit or line), and the presence of caries. Defects on all teeth, and all teeth present were noted.

To determine the absence of defects, at least four unaffected teeth had to be present, due to the possibility that missing teeth might have contained hypoplasias. This criterion follows the methodology of Goodman et. al. (1980). This selection criterion was not used to determine the presence of defects. Defective teeth were easily recognized with the use of a hand lens and probe. Only one affected tooth was necessary to determine the occurrence of a stress episode. If four teeth per individual were necessary for this determination, error might be skewed in favor of a less than representative frequency of defects, since these are highly susceptible to caries and subsequent loss. Fewer teeth would in all likelihood be present among stressed individuals.

Some researchers have attempted to use the frequency of hypoplasias for age groups at death as an indication of the health characteristics of the targeted ages. This method seems more likely to reveal the survival rate of stressed individuals, but not the age at which insult occurred. The approach used in this study was modeled after Swardstedt (1966) and modified to include the developmental sequence for deciduous dentition.

The height of each tooth was divided by the number of months during which its enamel developed. Thus, all crowns were divided into the horizontal segments encompassing 16 months of enamel formation (averaging the conclusion of formation at the eleventh post-natal month). The distance measured from the dentio-enamel junction to the defect(s) was then divided by the width of a monthly increment for that tooth. This became an expression of the number of months between the occurrence of the defect and the last month of enamel development. Finally this figure was subtracted from the developmental month at which enamel development ended for specific teeth. By this method, the coordinates of defects were plotted by the month of occurrence. The incidence of stress episodes (frequency of stress occurring during a given month and not time of onset) during each month, was plotted for the sub-adult sample (Figure 1). As expected, many individuals evidenced defects on different teeth, with overlapping developmental timing, which had occurred during the same month. Only one month episode was recorded in such cases. Defects (usually
hypocalcification) spanning several developmental months, were plotted as one episode for each month in which they occurred.

The incidence and duration of episodes by month of onset were also calculated (Figures 2 and 3). Only primary episode onset is described, since secondary episode onset may be predisposed by previous stress in ways that are not understood.

Results

All defects in deciduous dentition occur by the twelfth month post partum. Percentages generated for the entire sample of fifty individuals give the frequency of individuals stressed by the end of infancy. Almost two-thirds (64%; N=32) had microdefects. This finding, even when combined with adult estimates, is far more comparable to clinical results than those obtained from studies of adults and adolescents alone.

Of those having defective dentition, 90.6% (N=29) evidenced hypocalcification. Hypoplasia occurred among 59.3% (N=19) of the individuals with 84.2% (N=16) of these co-occurring with hypocalcification. Thus, hypoplasias were predominantly accompanied by hypocalcification while hypocalcification often occurred alone (31.25%; N=10).

The relationship between hypoplasia and hypocalcification with respect to severity is uncertain. Some evidence from this study may suggest that hypocalcification is less severe in intensity (though often of greater duration if reflected by width of defect). When hypocalcification co-occurs with hypoplasia, the former almost invariably occurs as the earlier aspect of pathological enamel formation or the earlier and later aspects with hypoplasia between. One might expect individuals who survived to have experienced less severity in earlier and later phases of malnutrition or disease. Furthermore, the present study provides evidence of individuals who have survived stress--manifested as hypocalcification--nearly one-third more often, yet with higher incidence and earlier onset per individual, than hypoplasia. However, rather than having a greater survival rate, this may be a function of greater incidence of hypocalcification during development. Nonetheless, one would expect a greater number of less severe episodes among survivors. Hypoplastic pits are more disruptive to dental tissue than hypocalcification discoloration. Still, on the average, solely hypocalcified individuals are dead one year earlier than individuals with hypoplastic defects. The greater duration or chronicity of hypocalcification-producing stress may account for the earlier age of death. Experimental studies may be needed to demonstrate, with certainty, the relationship between defects.
Figure 1 demonstrates a clear pattern of incidence in metabolic stress occurrence. Survived stress—manifested as hypoplasia—increases over the pre-natal period, and peaks during the first month after birth with rapid subsequent decline. Hypocalcification incidence is highest during the eighth prenatal month and remains high for the first few months after birth.

Pre-natal stress (indicative of a maternal stress as well) is of the highest incidence. The significance of pre-natal and maternal stress is perhaps best characterized by the timing of primary stress onset. Figures 2 and 3 illustrate onset patterns for each of the indicators. Using the first defect to appear on an individual (usually hypocalcification) to indicate primary onset we find that 28.1 percent of all primary stress begins by the fifth month in utero: 71.8 percent of primary stress onset occurs prenatally, with 89.8 percent occurring by the second month of life. Furthermore, stress duration appears to be greater when onset occurs prenatally.

Discussion

Sciulli (1976:79) has pointed to such high levels of pre-natal hypoplastic stress as "... notable since the pre-natal period of development is usually well buffered from stress by the intrauterine environment and most upsets capable of affecting maternal metabolism severely enough to precipitate stress in the fetus commonly result in abortion."

With so many stressed foeti, abortion may have been very common at Dickson. Indeed, many of the study cohort may have been premature (a rough equivalent of survived abortion). Sweeney et. al. (1969:1278) found that 80 percent of Santa Maria Cauque, Guatemalan children less than 40 weeks in utero, had an infection within the first three months. Seventy-five percent of these had hypoplastic lesions. They report that Rosensweig and Sahar (1962) found that 25 percent of premature children in a "developed country" had hypoplasias. Sweeney et. al. attribute susceptibility to low cord-blood levels and liver stores of vitamin A, compounded by infection (1969:1278-1279). Perhaps some of the stress which appears to be occurring late in the prenatal period (calculated by assuming "normal" developmental time span) actually reflects neonatally stressed prematurity.

The Native American population at Dickson Mound was severely stressed. Foeti stressed by maternal insufficiency usually continued to be stressed into infancy, a characteristic of about two-thirds of the sample Dickson children.
As far as the trend in defect incidence is concerned, the pre- and neo-natal increase probably represents either increased metabolic stress frequency with periods of greater foetal demand on maternal nutrition or a high proportion of more developed foeti surviving stress to display defects.

Hypocalcification is more prevalent in the infant population than hypoplasia, and the incidence of either defects brings skeletal results closer to the pattern of childhood stress which clinical studies that include children would predict. Greater mortality of stressed individuals appears to be taking place between infancy and adulthood. Therefore, this study supports the notion that the surviving adult population, while exhibiting infancy-developed enamel, underrepresents the degree of stress experienced by infants. It is evident that hypocalcification and hypoplasia in deciduous dentition, e.g., identified during the earliest indication, may provide more detailed and accurate information regarding infant stress rates than other methods of enamel defect analysis.
REFERENCES CITED


Figure 1

HYPOPLASIA and HYPOCALCIFICATION
Incidence by Month of Occurrence

n = 233
HYPOPLASIA

Primary Onset Incidence by Month

Figure 2
Figure 3

HYPOCALCIFICATION
Primary Onset Incidence by Month