DENTAL ENAMEL HYPOPLASIAS IN PREHISTORIC POPULATIONS

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ABSTRACT

Recent years have witnessed an impressive increase in research on enamel hypoplasias in archaeological populations. By reviewing a series of studies of enamel hypoplasias at Dickson Mounds, Illinois, North America (950-1300 A.D.), a prehistoric site involved in the transition from gathering-hunting to agriculture, this paper provides an illustration of this type of research.

The location of linear hypoplasias on labial tooth surfaces of 111 adults was studied with a thin-tipped caliper, and this location was converted to an age at development. Most defects developed between two and four years of developmental age. Hypoplasias increased in prevalence from 45% in the pre-agriculture group to 80% in the agricultural group ($p < 0.01$). The transition to agriculture occurred at a cost to infant and childhood health. Defects are associated with decreased longevity. Individuals with defects have a life expectancy of nearly ten years fewer than those without defects, suggesting that the development of a defect marks a significant and lasting health event. Enamel hypoplasias occur most frequently on anterior teeth, polar teeth in developmental fields, and the middle developmental thirds of teeth. Analysis of these data suggests that enamel may be differentially susceptible to growth disruption and that susceptibility varies both within and among teeth.

The study of enamel defects at Dickson provides insights into the health and nutritional consequences of the economic change from hunting and gathering to agriculture. More generally, with the availability of teeth from genetically homogeneous populations, studies of enamel hypoplasias in prehistory should provide a useful complement to research on this condition in contemporary peoples.

INTRODUCTION

The convening of an interdisciplinary conference on developmental defects of dental enamel is timely for one studying these defects in an archaeological context. Closer connections to the increasing body of research on dental defects in experimental animals and contemporary human populations will help improve prehistorians' understandings of the significance of these defects in archaeological contexts. Conversely, those studying dental defects in contemporary humans may not be aware of the impressive growth in research on these defects in prehistoric groups.

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Where skeletal remains are available for analysis, enamel hypoplasias are now very often studied as indicators of physiological perturbations (stress) experienced during tooth development (Rose *et al.*, 1985). For example, in a recent volume on health and nutritional changes at the transition from hunting-gathering to agriculture (Cohen and Armelagos, 1984), 15 of the 19 (79%) regional case studies included enamel defect data. The annual meetings of the American Association of Physical Anthropologists regularly include a number of papers on enamel defects, and the 1988 meeting featured a symposium devoted to this subject.

The use of enamel hypoplasias as non-specific, systemic stress indicators in prehistoric studies seems to be well-justified. On the one hand, most defects are unlikely to be due to hereditary factors or localized disruptions (traumas), and these few cases are usually identifiable (Cook, 1980). On the other hand, the large number of possible causes of these defects (Cutress and Suckling, 1982) precludes ascribing a more specific cause. Especially where the fluoride content of drinking water is low, and bouts of undernutrition

and disease during infancy and childhood are frequent, most defects are likely to be secondary to the stress imposed by these factors on the developing enamel.

This use of enamel hypoplasias as indelible recorders of stresses during a critical period of dental development fits into prehistorians' increased concerns with health and nutritional status. Temporal changes in health and nutrition are now viewed as important keys to our understanding of human evolution. Further, the distribution of disease and malnutrition over ecological and sociopolitical terrains is increasingly seen as critical to our understanding of the human condition, past and present. Dental researchers may not share all the prehistorians' evolutionary concerns. However, most might at least appreciate that the archaeological database provides a greater time depth and a varied set of ecological settings in which to study a condition that is common today.

The purpose of this paper is not an exhaustive review of the study of dental defects in prehistoric and archaeological populations, but rather to highlight trends and perspectives in a way that might inform those working with contemporary populations. This will be done *via* a review of a series of studies of enamel defects from one archaeological site: Dickson Mounds, Illinois, North America (950-1300 A.D.).

In this paper, I will first provide a brief introduction to Dickson archaeology and the methods employed for studying enamel hypoplasias. Subsequently, results of three applications of the study of enamel defects at Dickson are presented: (1) temporal changes in the prevalence and age at development of defects; (2) associations between enamel hypoplasias and longevity; and (3) distribution of defects within and among teeth in relationship to patterns of sensitivity to developmental disruption. The chief contribution of the first application is toward understanding shifting patterns of health and nutrition over time. The second application has implications for the validity of dental hypoplasias as "stress indicators". An association between enamel defects and mortality would suggest that the formation of these defects is a meaningful event. Finally, the third application raises fundamental biological questions about the nature of normal enamel formation and how differences in sensitivity to developmental disruption may help to explain the distribution of enamel hypoplasias within and among teeth.

MATERIALS AND METHODS

The Dickson Mounds are a multicomponent, habitation-burial complex located near Lewiston, Illinois. The skeletons from Dickson have been associated with three roughly sequential cultural groups: Late Woodland (LW), Mississippian Acculturated Late Woodland (MALW), and Middle Mississippian (MM). The Late Woodland group (*ca.* 950-1100 A.D.) is characterized as one involving a relatively small (74-125) and semi-sedentary gathering and hunting population, with seasonal camp sites and an economy directed toward the use of a broad spectrum of local fauna and flora. The MALW group (*ca.* 1050-1200 A.D.) reflects a transition during which local populations came under the influence of Mississippian cultures further to the south and already involved in maize-based agriculture (Harn, 1978, 1986). During the MM time period (*ca.* 1200-1300 A.D.), the "Mississippianization" of local populations became complete with the culmination of trends toward: (1) extended and intensified trade networks; (2) increased population density, size, and sedentarism; and (3) greater reliance on maize agriculture (Goodman *et al.*, 1984b). In sum, in a short period of time the Dickson population experienced a revolutionary change in lifestyle, from nomadic and rather self-reliant gathering-hunting to agriculture, village life, and increased contact with outside groups.

The mounds and associated cemeteries are estimated to include over 3000 skeletons, of which nearly a third have been excavated and made available for paleo-epidemiological analysis. Cultural group affiliations of these individuals were determined by association with grave goods and burial position indicative of the different groups and locations within the mounds (Goodman *et al.*, 1984b). Developmental age of adults was based on the agreement of multiple standard measurements, including the degree of epiphyseal closure and changes in the pubic symphysis (Goodman *et al.*, 1984b; Moore *et al.*, 1975).

The temporal changes at Dickson have been shown to be associated with increases in nutritional, infectious, degenerative, and traumatic pathologies and a decrease in growth status and life expectancy (Goodman *et al.*, 1984b; Goodman and Armelagos, 1985a). For example, porotic hyperostosis, an indication of iron deficiency anemia, is four times as prevalent among MM subadults as compared with LW subadults (64% to 16%) (Lallo *et al.*, 1977). Periosteal infections in subadults increase from 27% in the LW to 81% in the MM (Lallo *et al.*, 1978). Life expectancy is lower at all age intervals in the MM when compared with the combined LW and MALW samples (Moore *et al.*, 1975).

One hundred and eleven adults and adolescents were examined for the presence of enamel hypoplasias so that our understanding of changes in nutrition and health would be improved (Goodman *et al.*, 1980). Enamel hypoplasias were operationally defined as having circumferential pitting and lines or bands of decreased enamel thickness (see Fig. 1). Identification was aided by the cleaning of teeth and use of a dental probe and binocular microscope.

All teeth were studied except for third molars, which were considered to be too variable in developmental timing for estimation of age at hypoplasia formation. Loss of the tooth (*pre-* or *post mortem*), as well as loss

of enamel (due to attrition and other causes), was recorded so that overestimation of the enamel available for study would be avoided. For approximation of the developmental ages of individuals at the time of hypoplasia formation, the locations of defects relative to the cemento-enamel junction were measured with a thin-tipped caliper to 0.1 mm. These location measurements were then converted to a developmental age based on the tooth development chronology of Massler and co-workers (1941). Defects were "clustered" into half-year developmental periods. For example, the maxillary central incisor was divided into nine half-year periods between birth-0.5 and 4.0-4.5 years of age. Finally, defects on different teeth were "matched" to others that occurred during the same time period so that it could be ascertained that the hypoplasias were due to a systemic disruption. By use of this method, a chronology of stress by half-year developmental periods was achieved for each individual from birth to seven years of age (Goodman *et al.*, 1980, 1984a).

We should caution that this chronology provides only an estimate of *developmental* age at time of formation of enamel hypoplasias. Variation may be expected among individuals, genders, and populations. Eruption may be delayed by stresses such as chronic undernutrition and parasitism (Alvarez and Navia, 1989). Therefore, matrix formation and calcification times might also be affected. More information is needed on population differences in and environmental effects on dental matrix formation and maturation. Nonetheless, the chronology of Massler and co-workers (1941) has been used in all other studies of the chronology of enamel hypoplasias (Goodman, 1988) and is close to other calcification standards (Cameroon and Sims, 1974). It is a useful starting place for the development of information on the timing of enamel hypoplasias.

The first study reviewed utilizes the entire data set, while the last two use a portion of the data set. The second focuses on defects occurring between 3.5 and 7.0 years, a period of time that was available for study on all individuals. The third study is based on the 30 individuals who had a complete and uneroded set of teeth.

RESULTS

Temporal Changes in Prevalence and Age at Development

Nearly two-thirds (66%) of individuals had one or more hypoplasias. A clear trend of increased hypoplasias *per* individual is evident through prehistoric stages. The mean frequency of enamel defects increases from 0.90 in the LW (n = 20) to 1.18 in the MALW (n = 45) and 1.61 in the MM (n = 46). There is an increased frequency of individuals with one or more hypoplasias—from 45% in the LW to 60% in the MALW and 80% in the MM. The difference in prevalence between the LW and combined MALW and MM and the MM alone is significant ($p < 0.01$ level, Chi-square test with Yates' correction) (Goodman *et al.*, 1980).

The chronological pattern of enamel defects for the pre-agricultural groups (combined LW and MALW) and the agricultural group (MM) is presented in Fig. 2 (Goodman *et al.*, 1984a). The developmental patterns of defects in both the pre-agricultural and agricultural groups follow a roughly normal distribution, with highest frequencies between two and four years of age. However, there is some evidence for a slightly earlier distribution of defects in the MM compared with the combined LW/MALW group. The median is earlier in the MM (2.5-3.0 years) than in the LW/MALW (3.0-3.5 years). At 2.5-3.0 years, 57% of the MM's 93

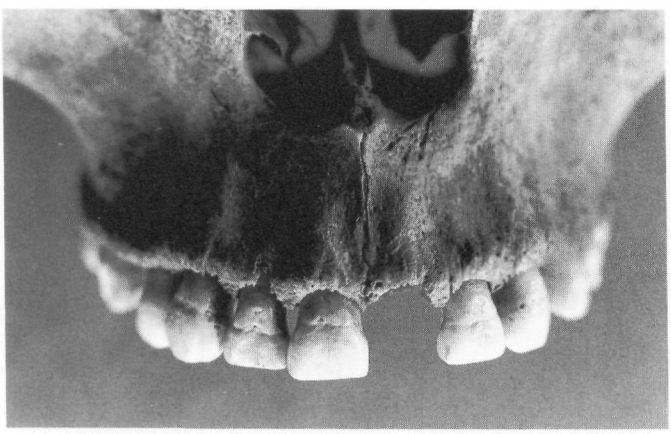

Fig. 1—Chronologic enamel hypoplasias on maxillary, anterior permanent teeth. Hypoplasias are observable on the right central incisor, the right and left lateral incisors, and the right and left canines. All hypoplasias occur between 3.0 and 3.5 years of developmental age and appear to be the result of the same systemic disruption (stress).

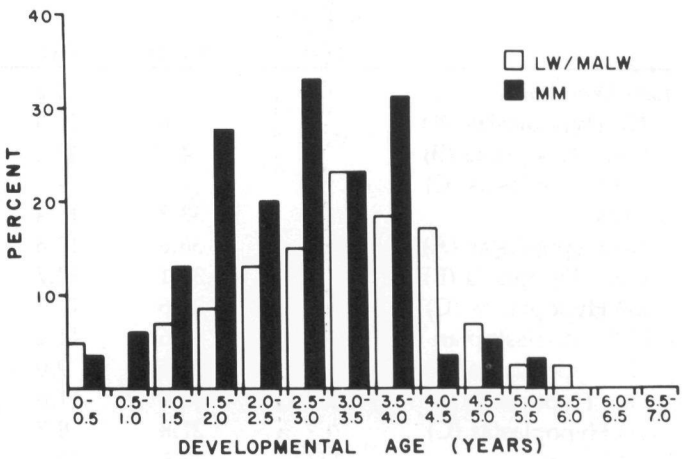

Fig. 2—Frequency distribution of enamel hypoplasias by half-year periods in two Dickson Mounds populations: combined Late Woodland (LW) and Mississippian Acculturated Late Woodland (MALW) and the Middle Mississippian (MM) (Goodman *et al.*, 1984a).

defects had occurred, compared with only 34% of the 50 LW/MALW's defects. This difference approaches significance (two-tailed $p < 0.10$) *via* the Kolmogorov-Smirnov test (Goodman *et al.*, 1984a).

Enamel Hypoplasias and Longevity

The mean age at death of individuals in the three cultural groups and in the total sample is compared for those individuals with no hypoplasia-stress periods between 3.5 and 7.0 years, one hypoplasia-stress period, and 2-3 hypoplasia-stress periods (Table, Fig. 3). In the LW, no individual had more than a single defect. Those with one hypoplasia had a slightly but not significantly greater mean age at death (34.7 years) than individuals with no hypoplasia-stress periods (31.6 years) (F ratio = 0.35; Goodman and Armelagos, 1988).

This association between hypoplasias and longevity is reversed during the MALW periods. The mean age at death of individuals without hypoplasias was 36.6 years, or 5.5 years greater than those with one hypoplasia-stress period (31.1 years) and 8.0 years greater than those with two or more stress periods. This inverse association between stress periods and mean age at death is most pronounced during the MM. The mean age at death of individuals without hypoplasias was 37.5 years, or 7.3 years longer than those with one hypoplasia and 15.7 years longer than those with two or more hypoplasias. A one-way ANOVA, testing for the statistical significance of differences in ages at death among hypoplasia-stress period groups, yielded an F ratio of 6.52 ($p < 0.01$; Table).

There is also a significant decrease in longevity with childhood stress periods in the total sample (Table; Fig. 3). The mean age at death of individuals without hypoplasia-stress periods was 35.8 years, or 4.4 years greater than for those with one hypoplasia-stress period (31.4 years) and 10.2 years greater than for those with two or more hypoplasia-stress periods (25.6 years). The ANOVA yields a significant F ratio (4.99, $p < 0.01$). The most significant *a priori* contrasts are between the no-stress-period group and the one-or-more-stress-periods and two-or-more-stress-periods groups ($t = 3.04$ and 3.08, respectively; $p < 0.01$; Goodman and Armelagos, 1988).

Factors Affecting the Distribution of Enamel Hypoplasias

Variations in defect frequency among teeth are presented in Fig. 4. The maxillary central incisor is most hypoplastic, followed by the mandibular canine, the maxillary canine, and the other incisors. Despite similar ages at development, the maxillary central incisor has nearly twice the frequency of defects as the other incisors. Furthermore, this tooth is 4-5 times as hypoplastic as first molars, which are developing at roughly similar ages.

Although there is extreme variation in defect frequency across teeth, we also noted that the distributions of defects within teeth are remarkably similar, despite variations in age at formation (Fig. 5). Specifically, when the tooth crown is divided into developmental thirds (incisal/occlusal, middle, cervical), the lowest frequency of defects is invariably found on the incisal/occlusal third, and the highest proportion of defects is nearly always found in the middle third. For all 14 teeth, from zero to 25% of defects *per* tooth are found on the incisal/occlusal third, from 40

TABLE

COMPARISON OF MEAN AGES AT DEATH FOR INDIVIDUALS BY CULTURAL HORIZON AND NUMBER OF HYPOPLASIA-STRESS PERIODS BETWEEN 3.5 AND 7.0 YEARS OF DEVELOPMENTAL AGE

	Sample Size	Mean	S.D.	One-way ANOVA (F Ratio)	*A priori* A *vs.* B	Contrasts A *vs.* C	(T-Values) A *vs.* B+C
Late Woodland	20	33.0	11.5	0.35	0.55	---	0.55
No Hypoplasias (A)	11	31.6	10.4				
One Hypoplasia (B)	9	34.7	13.0				
2-3 Hypoplasias (C)	-	--	--				
MALW	45	33.3	13.4	1.44	1.22	1.53	1.69
No Hypoplasias (A)	22	36.6	12.8				
One Hypoplasia (B)	14	31.1	14.7				
2-3 Hypoplasias (C)	9	28.6	11.7				
Middle Mississippian	46	31.6	11.2	6.52**	2.25**	3.50***	3.52***
No Hypoplasias (A)	17	37.5	9.0				
One Hypoplasia (B)	22	30.2	11.0				
2-3 Hypoplasias (C)	7	21.8	8.7				
Total Sample	111	32.5	12.1	4.99**	1.84+	3.04**	3.08**
No Hypoplasias (A)	40	35.8	10.1				
One Hypoplasia (B)	45	31.4	12.7				
2-3 Hypoplasias (C)	16	25.6	10.8				

+ = 2-tailed $p \leq 0.10$; * = 2-tailed $p \leq 0.05$; ** = 2-tailed $p \leq 0.01$; *** = 2-tailed $p \leq 0.001$.

to 80% on the middle third, and from 18 to 60% on the cervical third (Fig. 5).

DISCUSSION

The increased frequency of enamel hypoplasias in the MM at Dickson supports other paleopathological data in suggesting that the temporal changes experienced by the Dickson populations led to an increase in infant and childhood stress (Goodman *et al.*, 1980, 1984a). Although the exact cause of these defects cannot be ascertained, the peak in hypoplasias between two and four years of age suggests that, along with the associated parasitism, the weaning diet may not have been adequate.

More generally, enamel hypoplasias are ubiquitous in prehistoric populations (Rose *et al.*, 1985). This is consistent with the view that these groups were experiencing a variety of bouts of poor health and undernutrition, with minimal medical intervention. The high prevalence of hypoplasias suggests that they are due to systemic perturbations secondary to stresses of undernutrition and infection (Suckling *et al.*, 1983, 1986).

The developmental distribution of defects in prehistoric samples consistently peaks between two and four years of age (Goodman, 1988). This contrasts to Sarnat and Schour's (1941) study of a modern clinical sample from Chicago, in which a much earlier peak was reported. The difference suggests that the pattern of defects is due not to universal biological factors, but rather is related to patterns of stress. In peasant populations, it is common to have a peak in infection and malnutrition after the first year (*cf.* Chavez and Martinez, 1982; Mata *et al.*, 1971). The hypoplasia peak found in archaeological populations seems to follow this developmental pattern.

There are at least three processes which may account for the association between enamel hypoplasias developing between 3.5 and 7.0 years of age and decreased life expectancy in adolescence/adulthood. First, this association may result from differential lifelong patterns of biological susceptibility to physiological disruptions. An increased susceptibility to stress may cause both an increased frequency of childhood hypoplasias and an earlier age at death. That is, individuals who are ill during childhood may continue to fall ill as adults, due to a "weak constitution". Second, individuals who were exposed to and survived a period of severe childhood stress may suffer a loss in ability to respond to other stresses. In a sense, these individuals are "biologically damaged" by the early stress. The wear and tear of stresses during development may render them less fit to respond to and survive subsequent stresses. Third, these data may result from differential lifelong patterns of behaviorally and culturally based exposure to stressors. An increased lifelong potential for exposure to stres-

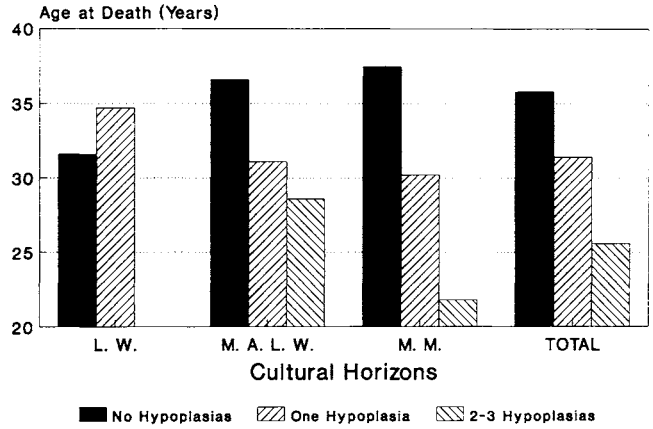

Fig. 3—Mean ages at death of Dickson Mounds adolescents/adults by number of hypoplasia-stress periods between 3.5 and 7.0 years of developmental age. LW = Late Woodland, MALW = Mississippian Acculturated Late Woodland, MM = Middle Mississippian (Goodman and Armelagos, 1988).

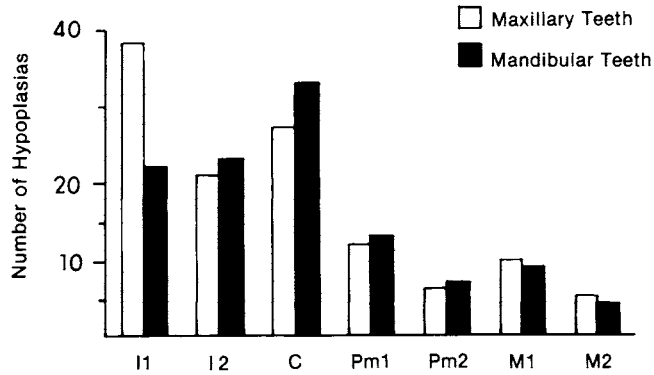

Fig. 4—Comparison of the frequency of enamel defects by tooth type based on 30 individuals from the Dickson Mounds (Goodman and Armelagos, 1985b).

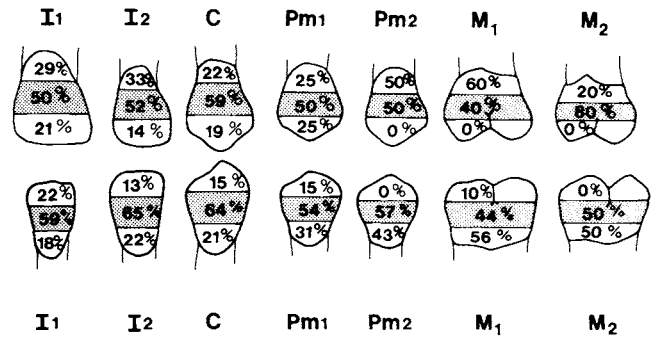

Fig. 5—The frequency of enamel hypoplasias by incisal/occlusal, middle, and cervical crown thirds of maxillary (upper row) and mandibular (lower row) permanent tooth type, I1 to M2 (Goodman and Armelagos, 1985b).

sors may cause both an increased frequency of childhood stress and earlier ages at death.

While these data have been largely unable to distinguish among these mechanisms, it is suggested that a cultural buffering hypothesis is most congruent with the patterns of association. The greatest difference in mean longevity between those with and without hypoplasias/stress is in the MM, the horizon in which status differences are likely to be greatest (Rothschild, 1979).

Although the cultural buffering hypothesis is supported, this does not exclude a role for a "biological damage" mechanism. Low socio-economic status during childhood may promote disease and undernutrition, which leaves individuals less able to rally from future insults. Most important, the data strongly support the notion that enamel defects are indicators of stress and that this stress is highly meaningful in terms of life expectancy. In sum, because enamel defects are relatively indelible, they may provide a unique tool for analysis of the long-term consequences of stress during early development.

A commonly held view contends that all teeth developing at the time of a disruption will be equally likely to develop a hypoplasia (*cf.* Cameroon and Sims, 1974; Sharawy and Yaeger, 1986). Our data, however, suggest that there are wide differences in susceptibility to hypoplasias among teeth developing at similar times (Goodman and Armelagos, 1985b).

A variety of unexplored factors, other than age at crown formation, may explain this apparent difference in susceptibility. The differences in frequency of defects among teeth developing at the same time may be due to differences in "genetic canalization" and developmental stability (*cf.* Waddington, 1957). The more genetically stable or canalized teeth may be more hypoplastic because, in a sense, they are unable to alter growth by changing velocity of development. Thus, matrix apposition occurs on time, but the quality of enamel suffers. Also, the polar and most stable teeth of developmental fields (*cf.* Butler, 1939; Dahlberg, 1945) are generally the most hypoplastic.

The paucity of defects on the incisal/occlusal thirds of teeth could be due to attrition. However, this pattern holds in this population even for the youngest individuals with recently erupted teeth. Thus, it is suggested that all teeth display similar within-tooth hypoplasia patterns due to common morphological or physiological factors, such as within-tooth variation in rate of enamel apposition, length of prisms, prism direction and angle, and number of active secretory-phase ameloblasts. We have suggested that long prisms are most consistently associated with enamel defects (Goodman and Armelagos, 1985b). However, we are unable to determine the physiological significance of long prisms. For example, are ameloblasts which secrete at a high velocity most at risk of disruption, or is the risk of a defect simply proportional to the amount of matrix formed by an ameloblast?

The study of enamel defects in anthropology is expanding on many fronts. For example, Skinner (1986) has shown that enamel defects are very common in the gorilla. However, these defects are far less common in both Old and New World monkeys (Vitzthum and Wikander, 1988). This variability across species may be significant to our understanding of the above-noted variations within species.

Anthropological analyses of enamel hypoplasias as indicators of stress have spread from studies of archaeological to historical (Goodman, 1988) and contemporary groups (Goodman *et al.*, 1987). Studies of hypoplasias in contemporary groups living under marginal conditions may add perspective to the prehistoric data. However, since methods for the analysis of general stress during infancy and childhood are generally poor, these contemporary studies may prove most important if we find that enamel defects are highly reflective of stress levels. This is one area of study where anthropologists are beginning to work with nutritional scientists, and where cooperation with other dental researchers is needed.

In summary, with increased interest in the ecology and evolution of humans, enamel hypoplasia has emerged as a frequently used indicator of dietary and disease stress. Recent studies have effectively discriminated levels of stress, indicated by the frequency of hypoplasia, between sexes, age groups, social classes, and time-successive populations (Rose *et al.*, 1985). A variety of benefits can be seen in cooperation between prehistorians and other dental researchers. Prehistorians need more precise information on the causes of enamel defects, while "dry skull" studies may provide unique research opportunities, including an ability to study the evolution of nutrition and health.

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