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Dental Enamel Hypoplasias as Indicators of Nutritional Status

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INTRODUCTION

We know a great deal about protein-energy malnutrition, almost everything in fact, except what causes it, how to prevent it, and what it costs society not to do so. (Calloway, 1982:744).

Teeth have long held a central place in the anthropological study of nutrition and diet. Because of the inherent and close relationship between teeth and diet, the dental structures have incorporated a variety of characteristics that reflect what was placed in the mouth and presumably consumed (Kay, 1985; Scott and Turner, 1988). Caries rates, for example, have long been known to reflect the degree of consumption of refined carbohydrates (Mummery, 1870; see also Chapter 10, *this volume*). Similarly, the degree and pattern of dental wear may be used to infer a number of aspects of food preparation and consumption (Powell, 1985).

Teeth may also reflect the nutritional status of an individual during tooth development. Nutritional status, defined as "the state resulting from the balance between the supply of nutrients on the one hand and the expenditure of the organism on the other" (McLaren, 1976:3), is the end result of numerous factors which affect both access to and utilization of nutrients. Although nutritional status is certainly a function of diet and nutrient intake, a variety of other factors such as work load and disease status interact

balance of the individual, and it is a function of a variety of interacting factors, including political-economic and ecological conditions. Finally, as Calloway (1982) suggests in the opening quote, poor nutritional status places functional limits on the community.

The purpose of this chapter is to provide a current assessment of our ability to read the record of nutritional status via the study of dental enamel hypoplasias, deficiencies in enamel thickness due to a cessation in ameloblast activity (Sarnat and Schour, 1941). Ultimately, our goal is to better assess whether enamel hypoplasias might become a useful addition to the toolkit of measures of nutritional status of both contemporary and prehistoric populations. In order to answer this question, however, one needs to address a variety of subsidiary issues, and the indicator has to meet a number of criteria that are essential to any epidemiological measure. Is this indicator easily and reliably identified and measured? What is its sensitivity and specificity? Is unique and useful information, not readily available from another source, provided by this indicator? How well do we know what metabolic and physiological states are outwardly and permanently reflected in the development of an enamel hypoplasia?

In a sense, the current epidemiological status of enamel hypoplasia, potentially useful indicator of past nutritional status, is comparable to a stage that has been transversed by a variety of other anthropometric measures of nutritional status. Although some

enamel hypoplasias might fit as measures of past nutritional status. How well do enamel hypoplasias reflect nutritional status? Are they particularly reflective of specific nutrient deficiencies? What other physiological conditions might be causative of enamel hypoplasias? Do enamel hypoplasias provide a unique perspective on an individual's history of nutritional adequacy?

In order to better understand the relationship between enamel formation and the occurrence of a developmental defects, we first present a brief summary of dental development and histology. Second, we review experimental and epidemiological studies of enamel defects that have helped establish their meaning and validity. Third, we discuss issues relating to the establishment of the time of formation of enamel hypoplasias. Other significant epidemiological issues, such as choice of teeth for study and classification and reliability of defects, are not focused on. (For a discussion of choice of tooth, see Goodman et al., 1980; Goodman and Armelagos, 1985a,b; and Rose et al., 1985. For discussion of issues in classification and reliability of defects, see Cutress and Suckling, 1982; FDI, 1982.) The main theme that we wish to stress throughout this chapter concerns the state of current knowledge regarding the meaning of enamel hypoplasias, and research directions which might help clarify the meaning and potential use of enamel hypoplasias.

ENAMEL HISTOLOGY

Normal Enamel Development

Although a variety of specific questions relating to the control of enamel formation have yet to be answered, the general pattern of enamel development is well understood (Jenkins, 1978; Warshawsky, 1985). Enamel development commences as an inductive process in which ameloblasts (epithelial cells that form enamel) line up at what will eventually become the dentin-enamel junction (Fig. 1). At the border between ameloblasts and odontoblasts (mesenchymal, dentin-forming cells), enamel and dentin formation begins (Kollar and Fisher, 1980). At a time that appears to be under strong genetic control, the odontoblasts begin to form the dentin matrix (Slavkin et al., 1984). The ameloblasts follow in secreting enamel proteins which form the enamel matrix. This process of secreting enamel and dentin pro-

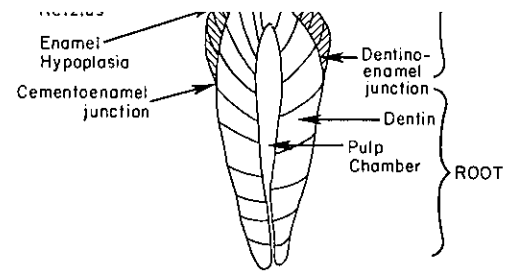


Fig. 1. Diagrammatic representation of a longitudinal section through a human mandibular canine. Note the major structures of enamel: enamel prisms and striae of Retzius. (From Rose et al., 1985.)

teins begins at the occlusal tips of the tooth crowns. As the process continues, more cervically located ameloblasts and odontoblasts are recruited to secrete protein and make enamel and dentin matrix, respectively (Shawashy and Yaeger, 1986).

The most essential structure of the enamel matrix are enamel prisms or enamel rods (Fig. 1). The enamel prisms transverse the enamel from the dentin-enamel junction to close to the surface of the crown, as most teeth have a thin layer of aprismatic enamel at the enamel surface. Human enamel prisms in cross section are characteristically keyhole in shape (Boyde et al., 1988; Shawashy and Yaeger, 1986). The secretion of enamel matrix by ameloblasts helps to form the enamel rods. However, the precise relationship between ameloblasts and enamel rods is uncertain. It is unlikely that one ameloblast makes one prism. However, it may be possible that the enamel matrix from four ameloblasts contribute to the matrix of four prisms (Shawashy and Yaeger, 1986).

A characteristic series of *constrictions* are frequently observed in long section of enamel prisms, usually spaced out at $\sim 2-6 \mu\text{m}$. These constrictions are most often called cross-striations and appear to suggest a rhythm to enamel matrix secretion. Many researchers have assumed that these cross-striation represent intervals of 24-hr growth episodes (Beynon and Wood, 1987; Bromage and Dean, 1985). If this hypothesis proves true, counting cross-striations may provide an important means for determining the length of time involved in enamel matrix formation.

Active secretory ameloblasts differ in the length of time they have been operative, with the more cervical

ameloblast having secreted enamel matrix for the shortest period of time. The "front" of new enamel matrix is, therefore, tangential or curved in relationship to the dentin-enamel junction (DEJ). It is farthest away from the DEJ at the occlusal end of the crown and closest at the cervical end of the crown. The exact shape of this developing front is maintained in the pattern of striae of Retzius, the second main enamel structure (Fig. 1). The first striae of Retzius do not reach the surface of the tooth until the full thickness of enamel has been reached. However, once the full thickness of enamel has been reached, the striae of Retzius may be said to run from the DEJ to near the outer surface of the crown (Fig. 1).

As the secretory ameloblasts complete their function of forming the enamel matrix, they begin to undergo changes in both shape and ultrastructure that are consistent with their change in function from secretion (tall secretory cells) to resorption and transport (short, ruffle-bordered resorptive cells) (Reith and Cotty, 1967). Influenced by the functionally changed ameloblasts, the enamel matrix loses protein and water and becomes more completely calcified. Mature enamel consists of approximately 97% calcified salt by weight and volume (Shawashy and Yaeger, 1986). If during the process of matrix secretion a group of ameloblasts are disrupted to a degree that they lose their functional capacities, less matrix will be formed. The resulting enamel will be thinner.

Classification of Enamel Developmental Defects

Dental enamel hypoplasias are of a class of enamel developmental defects. As a general term, *enamel hypoplasias* refer to all defects in enamel thickness. These defects may differ from light single and multiple pits to small furrows to deep and wide troughs of decreased enamel thickness to entirely missing enamel. Enamel hypoplasias are quantitative defects, as opposed to enamel opacities (or hypocalcifications), the other major class of enamel developmental defects (FDI, 1982). All enamel developmental defects result from disruptions in the process of amelogenesis or enamel formation. Enamel hypocalcifications had long been held to be caused by disruptions in enamel maturation. However, this assumption has recently been challenged by the exper-

Based on the pattern of defects within and among teeth, hypoplasias can reliably be distinguished as to whether they result from one of three conditions: (1) an hereditary anomaly, (2) a localized trauma, or (3) a systemic metabolic stress (Shawashy and Yaeger, 1986). Defects resulting from hereditary causes generally affect entire tooth crowns and are the most severe. Defects caused by local trauma and other nonsystemic factors may also be relatively severe but will affect only one tooth or a few adjacent teeth. These defects may cover only a thin portion of the tooth crowns. Finally, defects resulting from systemic metabolic stress are likely to be found on a variety of teeth developing at the time of the stress, and the locations of the defects reflect the relative completeness of enamel crowns at the time of the disruption (Shawashy and Yaeger, 1986).

Individuals with hereditary defects are rare (<1%) in most contemporary populations (Winter and Brook, 1975). It is likely that they would be even less frequent in prehistoric populations because individuals with hereditary defects are frequently affected with other congenital problems (Winter and Brook, 1975; Stewart and Poole, 1982), and therefore, may have had little chance of survival. We are aware of only a single case of an hereditary enamel hypoplasia reported from a prehistoric sample (Cook, 1980).

Defects caused by local trauma are also rare in prehistoric populations. Goodman et al. (1980) assessed the prevalence with which enamel hypoplasias conformed to the chronologic or linear pattern which is diagnostic of systemic defects. Of 111 individuals studied for hypoplasias of the permanent dentition at Dickson Mounds, Illinois, only one hypoplasia conformed to a trauma-induced pattern. That is, it was clearly noted on one tooth but could not be matched to defects occurring at the same time on other teeth.

In the vast majority of cases, defects found in archaeological materials fit a chronologic pattern and appear to be due to systemic metabolic stress (Goodman et al., 1984; Rose et al., 1985). Thus, they are frequently referred to as chronologic or linear enamel hypoplasias (LEH), reflecting the linear and chronologic nature of the defects caused by systemic stress at a specific point in time during tooth development (Sarnat and Schour, 1941; Goodman et al., 1984).

but rather, what factors are most likely to cause a defect, which types of defect result, and how these studies improve our understanding of the sensitivity of enamel to disruption in humans.

A plethora of experimental studies of enamel hypoplasias were published in the 1930s to 1950s. These early studies demonstrated that a large number of nutritional deficiencies and excesses could potentially lead to an enamel defect in rats, mice, and other experimental animals (Schour and Massler, 1945). In addition, these studies succeeded in showing that other *stressors*, or factors that disrupt normal physiology and growth (e.g., hormonal imbalances and diseases), could lead to an enamel defect (Kreshover, 1960). The inference to be drawn from this early experimental research is that enamel defects are non-specific indicators of physiological perturbations. Kreshover succinctly summarized this research in stating:

Ample clinical and experimental evidence exists to suggest that developmental tooth defects are generally non-specific in nature and can be related to a wide variety of systemic disturbances, any of which, depending upon their severity and degree of tissue response, might result in defective enamel (Kreshover 1960:166).

It is easy to infer how the choice of rats and mice as experimental animals was likely to have been made. These animals are inexpensive and readily available. Moreover, to these early researchers the continuously erupting central incisors must have seemed like the perfect tooth to study. Unfortunately, the development of the enamel of this tooth is quite distinct from that of humans (Fejerskov, 1979). As a result, it is difficult to clearly compare pathological changes in rat incisor enamel with equivalent changes in humans. Thus, these animals are less useful as experimental models.

The most important recent experimental work on the etiology of enamel defects has been done in New Zealand on sheep. Suckling and co-workers have succeeded in producing pathological changes in sheep enamel that correspond to enamel hypoplasias and hypocalcifications (Suckling and Thurley, 1984a; Suckling, 1986). These changes have been produced via trauma (Suckling, 1980; Suckling and Purdell-Lewis, 1982a), fluoride supplementation

interesting. Suckling and colleagues do not find any evidence to suggest that the frequency of enamel hypocalcifications increase with the level of parasitism. Conversely, they find a fairly clean dose-response relationship between the formation of an enamel hypoplastic defect and parasite load. All sheep receiving a high dose of parasites for a short period (7-10 days) at 8-9 months, a load severe enough to cause weight loss and debilitation, developed an enamel hypoplasia (Suckling et al., 1983). There is also some evidence that wider defects are associated with a more severe parasite load. The results of the above studies support the earlier contention that enamel developmental defects are highly nonspecific in nature. It seems that relatively few stressors, if severe enough, will not produce pathologic changes in developing enamel. As is generally true, it is difficult to infer sensitivity from animal experimental research. Most of the manipulations in these studies were quite profound. Only in the more recent work of Suckling and co-workers do we have a sense of the severity of stress necessary to cause an enamel hypoplasia. The issue of sensitivity may be better answered via studies with humans.

Epidemiological Studies

Epidemiological studies of the frequency of enamel hypoplasias in contemporary populations support a general association between the prevalence of hypoplasias and general living conditions. Although the direct comparison of prevalence rates from different studies is problematic due to variation in method of diagnosis and sampling of individuals and teeth (Goodman and Armelagos, 1985a,b), individuals in developed countries tend to have lower rates of enamel defects as compared with those from underdeveloped areas.

The frequency of individuals with one or more hypoplasias is generally less than ten percent in most populations from developed, industrialized countries (Cutress and Suckling, 1982). In comparison, enamel hypoplasias are frequently quite common in children from Third World countries (Baume and Meyer, 1966; Jelliffe and Jelliffe, 1971; Moller et al., 1972; Enwonwu, 1973; Schamschula et al., 1980; Sawyer and Nwoku, 1985; Goodman et al., 1987). As an example, Anderson and Stevenson (1930) report that nearly 90% of Chinese children have some form of enamel defect.

It is not surprising that the frequency of individuals with enamel hypoplasias of the permanent dentition in prehistoric populations is comparable with frequencies found in studies of contemporary underdeveloped countries. For example, Goodman et al. (1980) found that 66% of the adults from Dickson Mounds, Illinois (AD 950–1300), had at least one hypoplasia. Black (1979) notes hypoplasia rates as high as 94% on canines from a Mississippian cemetery in Missouri. Corruccini et al. (1985) report hypoplasias in 54.5% of Barbados slaves and suggest that accounting for missing teeth would raise the true prevalence to around 75%. Hojgaard (1980) found that 84% of his sample from prehistoric Bahrain (2000 BC) had hypoplasias, and Hutchinson and Larsen (1988) note hypoplasia rates of greater than 70% for Amerindians from the coast of Georgia. Hillson (1979), impressed by the high frequency of defects in prehistoric Egyptian and Nubian populations, suggests that, in impoverished communities, stresses during tooth development will be great enough to produce a hypoplastic prevalence of >40%. He further suggests that this is most likely due to chronic nutritional and disease stress, and hypoplasia rates would be even higher during periods of particularly pronounced stress.

The pioneering works of Sweeney and co-workers (Sweeney and Guzman, 1966; Sweeney et al., 1969, 1971) and Infante (1974; Infante and Gillespie, 1974) have more firmly established a general association between enamel hypoplasias and socioeconomic status. Sweeney et al. (1971) found an association between enamel hypoplasias of the deciduous upper central incisor (teeth whose crowns develop from about six months prenatally to about three months postnatally) and the degree of malnutrition in Guatemalan children. Forty-three percent of the children aged 2–7, with second-degree malnutrition (61–75% weight-for-age) had hypoplasias, whereas 73% of children with the more severe third degree malnutrition ($\leq 60\%$ weight-for-age) had enamel hypoplasias. In another study of Guatemalan children aged 6–83 months, Infante and Gillespie (1974) note variations in prevalence by village from 18% to 62%.

Infante (1974) reports that 19.4% of White Mountain Arizona Apache children have hypoplasias on their deciduous central incisors. With the inclusion of carious lesions (believed to be secondary to hypoplastic involvement), the prevalence increased to

Goodman et al. (1987) studied the frequency and chronological distribution of enamel hypoplasias in children from five rural communities in the Solis Valley of the Mexican altiplano. These communities were selected for study because of the presence of endemic mild-to-moderate malnutrition (children at 60–95% weight for age). These investigators found one or more hypoplasias in 46.7% of 300 children examined. Among the unworn and completely erupted teeth, the highest prevalence was found on permanent teeth, especially the maxillary central incisor (44.4% were hypoplastic); 14% of the deciduous maxillary central incisors were hypoplastic. This prevalence is slightly less than reported by Infante and Gillespie (1974) and Sweeney et al. (1971) from rural Guatemalan children.

For the Mexican children, most deciduous tooth defects appear to occur around the last trimester and neonatally. This pattern is similar to that found by Blakey and Armelagos (1985) in their study of deciduous tooth hypoplasias from the prehistoric Dickson Mounds, Illinois, population. For the permanent teeth, there is a clear tendency toward hypoplasia occurrence at 18–36 months. Because weaning generally takes place in the second year in these Mexican communities (Allen et al., 1987), Goodman and colleagues suggest that the increased frequency of hypoplasias may result from stresses associated with weaning.

In support of the relationship between enamel hypoplasias and general living conditions, Goodman et al. (1988) assessed the relationship between enamel hypoplasia and current nutrition and socioeconomic status. Nutritional status was determined by height-for-age and socioeconomic status is based on the household's "material style of life." Individuals with enamel hypoplasias tend to fall into a lower percentile height-for-age, and their families tend to score lower on the material style of life scale. This cross-sectional study suggests a relationship between general living conditions and the occurrence of enamel hypoplasia within these communities.

Goodman and colleagues (1989, in press) have also recently studied the frequency of enamel hypoplasias in two groups of adolescents from the highland community of Tezonteopan, Mexico. One group of adolescents has been nutritionally supplemented since birth, and during permanent tooth develop-

This study begins to directly link nutritional status to enamel hypoplasia frequency. In terms of sensitivity, we can infer from this study that a change from mild-to-moderate nutritional deficiency (the control group) to adequate nutrition (the supplemented group) has a significant effect on the frequency of enamel hypoplasias.

Less can be inferred from these studies regarding the specificity of enamel hypoplasias. It is not clear which nutrient deficiencies are most likely to cause an enamel hypoplasia in any of the epidemiological studies. In the Tezonteopan study (Goodman et al., in press), the supplement contained calories, protein, and a soup of micronutrients. Thus, any of a host of nutrients might account for the variation in enamel hypoplasia prevalence. Furthermore, it is not clear that increased nutrition intake directly causes a decrease in enamel hypoplasias. The supplemented individuals experienced about half the number of days ill with respiratory and diarrheal illnesses (Chavez and Martinez, 1982). Thus, it is possible that the enamel defects are more directly related to illness than to nutritional intake, or to the synergetic interaction between undernutrition and infectious disease. Poor nutritional status decreases immune function and predisposes one to infection, while infectious diseases can further diminish scarce nutritional resources (Solomons and Keusch, 1981).

In summary, studies of contemporary populations have shown a consistent increase in the prevalence of enamel hypoplasias for groups living in poor and underdeveloped communities. Although the general increase in enamel defects at the time of weaning suggests that dietary intake is a causative factor, it is not clear how important nutrition is, which nutrients are most critical, and how diet interacts with other factors (e.g., infectious, parasitic, and respiratory diseases) in the etiology of enamel defects. Regardless, enamel hypoplasias have been consistently associated with malnutrition and disease. Furthermore, these indicators of physiological stress are relatively indelible and are easily diagnosed.

Research Directions

Despite an estimated 500 articles on the epidemiology and etiology of enamel defects, we are just beginning to understand the sensitivity and specificity of enamel to developmental disturbance. There is a great deal of animal experimental research which

poplasias and specific nutrient statuses. Designing an experiment to show the relative effect of protein versus calorie deficiency would be a relatively easy proposition. Moving a step further, it would be very useful to know whether certain combinations of nutrient deficiencies are especially likely to cause an enamel defect and finally, how nutrient deficiencies might interact with other common stressors, such as parasitism, in the etiology of enamel defects. Animal research is not likely to provide the final word on either sensitivity or specificity, but it certainly can provide a variety of useful insights, especially regarding the issue of specificity.

The above discussion of epidemiological studies has barely mentioned paleopathological contributions, despite the numerous paleopathological studies that have been conducted. The reason for this is clear. Although these studies provide a variety of interesting applications of the use of enamel hypoplasias, they do not provide the needed controls that can increase our understanding of the meaning of these defects. If paleopathologists desire to understand the fundamental meaning of enamel hypoplasias, then they may need to do their own experimental or epidemiological research.

The majority of future contributions to understanding the meaning of enamel hypoplasias is most likely to come from epidemiological studies in which nutrition and other conditions are well documented. A number of longitudinal studies of undernutrition have been undertaken in such countries as Bangladesh, India, Mexico, and Guatemala (Pebley, 1984). In these studies, a group of individuals are followed for a period of time, with repeat measures of nutritional status, food intake, disease episodes, and related information. The data from these studies provides an excellent backdrop from which one might now study the formation of enamel developmental defects. If one can relocate individuals who were studied during the time of tooth development, and if one can now study the erupted teeth for the occurrence of enamel defects, then the correlation between various aspects of nutritional intake and status and the occurrence of enamel defects can be assessed. For example, during the mid-1980s, a group of mothers was studied in rural Mexico during the last two trimesters, and then the infants were studied during their first 6 months (Allen et al., 1987). The dietary, nutritional, and disease status of mothers and their infants was as-

sessed at frequent (weekly, monthly) intervals. Presently, these infants are around three to five years of age and their deciduous teeth should be fully erupted. It is our intention to locate these individuals in order to examine their teeth and relate the record of enamel developmental defects to the data collected while the teeth were developing.

A related epidemiological opportunity is similar to the above scenario, with the added dimension of examining the effect of an intervention or supplementation. This type of situation is one that comes the closest to a controlled human study. For example, in the 1960s, the Institute for Nutrition of Central America and Panama (INCAP) began a longitudinal, supplementation study in El Progreso, Guatemala (Lechtig and Klein, 1979). Individuals were assigned to one of three groups—a control (nonsupplemented) group and two groups receiving different nutritional supplements. Following individuals such as these could provide a strong inference about the role of nutrition in the etiology of enamel hypoplasias.

Trying to predict what we might know about the meaning of enamel hypoplasias at some future point in time is a risky proposition. One possibility, however, that needs to be anticipated is that our epidemiological and experimental studies might not provide us with indisputable data on the precise cause of enamel hypoplasias. We have already noted a number of inherent limitations of experimental research. Epidemiological research is limited in other directions. It should offer a means of assessing sensitivity, somewhat counteracting the main limitation of experimental studies. However, given the co-occurrence of nutrient deficiencies and other related conditions in the real world, I am not sure that epidemiological studies will ever be successful in disentangling these related, potential causes of enamel developmental defects.

This scenario is not as disappointing as it might seem at first glance. If one wants to understand the adaptive and functional consequences of stressors, then a nonspecific indicator might be highly useful. In fact, this is exactly the role played by anthropometric indicators of nutritional status (Martorell and Ho, 1984; Sutphen, 1985; Zerfas et al., 1985). Furthermore, if the ultimate physiological effects are so similar, it might not be important to know precise cause. If enamel hypoplasias turn out to be sensitive

THE TIMING OF ENAMEL HYPOPLASIAS

In addition to determining the frequency of enamel defects, the location of defects on tooth crowns provides the needed raw data for determining the age of individuals at the time of defect development. Perhaps the most unique feature of enamel hypoplasias is that, unlike nearly any other possible indicator, one may infer the time of their formation. Because of the regular and ringlike pattern of enamel development, this chronometric potential has long been recognized by a variety of researchers (Sarnat and Schour, 1941; Swärdstedt, 1966). The following section traces efforts to provide a chronology of stress during tooth development via use of enamel hypoplasias, provides an assessment of some of the possible errors involved in the age determination process, and suggests some directions for future research.

The first group to focus on the chronometric potential of enamel hypoplasias was Sarnat and Schour (1941). In their very influential paper, these workers introduced the concept that enamel hypoplasias may be used as a kymographic record of stress during tooth development (Sarnat and Schour, 1941). This idea was then applied to a contemporary clinical series. Briefly, these authors examined the location of enamel hypoplasias on the dentitions of 60 individuals from the Chicago area. Locations of enamel hypoplasias were compared with the standard of enamel crown development of Massler et al. (1941). Sarnat and Schour estimated that most defects (66%) developed during the first year and the majority of the remaining defects developed during the second year.

For the next quarter century, no further research was published on the chronological distribution of enamel defects, perhaps because of two conclusions drawn by Sarnat and Schour (1941). First, Sarnat and Schour suggested that it was difficult to match specific diseases or insults with the development of an enamel defect. They could link a number of conditions, noted in medical histories, to the appearance of an enamel defect at the appropriate location on teeth. However, defects were also frequently found without a matching illness, or an illness occurred without a defect developing at the corresponding locations on tooth crowns. On the one hand, this might suggest low sensitivity and specificity of enamel hypoplasias



Fig. 2. Photograph of chronologic enamel hypoplasias on anterior, maxillary permanent teeth. Hypoplasias are observable on the right central incisor, the right and left lateral incisors and the right and left canines. All hypoplasias developed around 3.0–3.5 years.

when one is sick, but reserves are adequate and the metabolic consequences of the illness will be negligible. Conversely, at other times a relatively minor “stressor” can cause a significant physiological response, leading to growth disruption.

Second, and related to the above, Sarnat and Shour (1941) suggested that the pattern of enamel defects they found in their group of children and adolescents would likely be a universal one. They proposed that the constitutional changes which a child undergoes are more determinant of the development of an enamel defect than the age pattern of disease. Because these “constitutional changes” are biologically universal, it would be neither necessary nor useful to examine the pattern of enamel hypoplasias in other groups or populations.

Although this advice appears to have been well heeded by dental researchers, it was less universally complied with by those working on prehistoric human remains. Swärdstedt (1966) was the first person to specifically ignore this advice. In his study of dental pathology in human remains from Westerhus, Sweden, Swärdstedt found a peak frequency of permanent tooth enamel hypoplasias around the ages of two to five years. Subsequent studies of permanent

tooth enamel hypoplasias have found similarly “late” peaks (Corruccini et al., 1985; Goodman, 1988; Goodman et al., 1984, 1987; Powell, 1988; Schulz and McHenry, 1975; Yamamoto, 1988). These anthropological studies clearly demonstrate that enamel hypoplasias do not universally occur most frequently in the first year of life. Rather, the most common pattern is a peak in defects around two to four years of age (Fig. 2). A number of researchers have suggested that this peak may be related to stresses associated with a change to a weaning diet, and may even reflect the time of weaning in different groups (Corruccini et al., 1985; Goodman et al., 1987).

Swärdstedt (1966) is more precise than Sarnat and Schour (1941) in describing his methods for assigning the age at formation of an enamel hypoplasia. Although Swärdstedt (1966) follows Sarnat and Schour (1941) in using the developmental standard of Massler et al. (1941), he is clearer than these earlier researchers in his description of how to translate the position of an enamel hypoplasia on a tooth crown to a specific age of the individual at development of the defect. Swärdstedt estimated the average tooth crown heights for each tooth type and obtained the time

each crown begins and ends calcification. He then divided each tooth into half-year zones. For example, a tooth crown such as the upper canine, which develops over a 6-year period and has an average crown height of 9.6 mm, is divided into 12 zones averaging 0.8 mm in width (Fig. 3). In fact, however, zones vary in width from 1.07 to 0.64 mm. This variation from 0.8 mm is related to an assumption of difference in velocity of growth, an assumption that is discussed below. In general, Swärdstedt relies on a number of assumptions in developing a chronology of enamel hypoplasias, most of which are followed by other researchers and lay the basis for subsequent studies. The following section examines some of these assumptions and other potential sources of error in determining a chronology of stress from the pattern of enamel hypoplasias.

Sources of Variation and Error

Choice of developmental standard. Perhaps the most important factor in determination of the chronology of enamel hypoplasia relates to the choice of developmental standard and how it is interpreted and employed. All current research rests on the standard established by Massler and et al. (1941). This is a fact that is mainly related to historical precedent. Although the use of the same standard increases comparability between different studies, one should consider whether it is the most appropriate standard for each specific population. If the Massler standard is not appropriate for a specific population, then it might be advantageous to alter it.

The standard of Massler et al. (1941) rests on a paucity of data (see also Chapter 8, *this volume*). The actual sample size is a point of contention, but does seem to have been quite small (about 1,000 teeth and 33 individuals), and the use of autopsy material introduces a possible error on the side of delayed development. Moreover, no estimates of variation in development are provided. There are two main advantages of this standard when compared with those standards based on more recent and methodologically sounder studies. It provides data on all teeth, deciduous and permanent, at all developmental stages, and is based on the direct observation of enamel and dentin matrix formation. Recent studies, which rely on the radiographic appearance of calcification, introduce a systemic error because calcification occurs at

inal researchers are estimates of ages at the beginnings and ends of main events (e.g., crown calcification), and this information is provided in a series of diagrams. Swärdstedt (1966) interprets these diagrams to mean that there are differences in the rate of enamel calcification at different locations on the tooth (Fig. 3).

Although originally accepting Swärdstedt's interpretation of the Massler et al. (1941) standard, Goodman et al. (1980) subsequently chose to adopt an assumption of constant velocity (Goodman et al., 1987; Goodman, 1988). Some variation in rate of formation is likely to be correct, but there is no reason to suggest that it will conform to the pattern implied by Swärdstedt (1966). Until better information is obtained on variation in rates of formation, it might be best to continue to use a constant velocity of enamel development as a sort of null assumption.

More research is desperately needed on the basic pattern of dental development before one can confidently state that estimated enamel hypoplasia chronologies reflect the realities of ages of individuals at the time of hypoplasia formation and stress. In addition to the aforementioned systemic error of being based on calcification rather than matrix formation, all of the more recent radiographic standards tend to start around the age of ≥ 2 years (Demerjian and Levesque, 1980; Haavikko, 1974; and Moorrees et al., 1963), universally missing the early stages of crown development (see Chapter 8, *this volume*). As many hypoplasias develop before 3 years of age—this is an extremely fundamental limitation. Finally, the samples on which these radiographic standards are based, usually middle-class to upper-class North Americans or Europeans, are likely to be a poor choice for application to most anthropological groups. Until more research is completed on the pattern of enamel matrix formation, one might do best to continue to adopt the standard of Massler et al. (1941). While this standard does not share the scientific qualities of the more recent radiographic standards, it has commonly been used and is in substantial agreement with more recent research (Cameroon and Sims, 1974; Gustavson and Koch, 1974).

Estimating age at defect formation. There are a variety of means to translate locations of defects on teeth to times of development. One can attempt to be

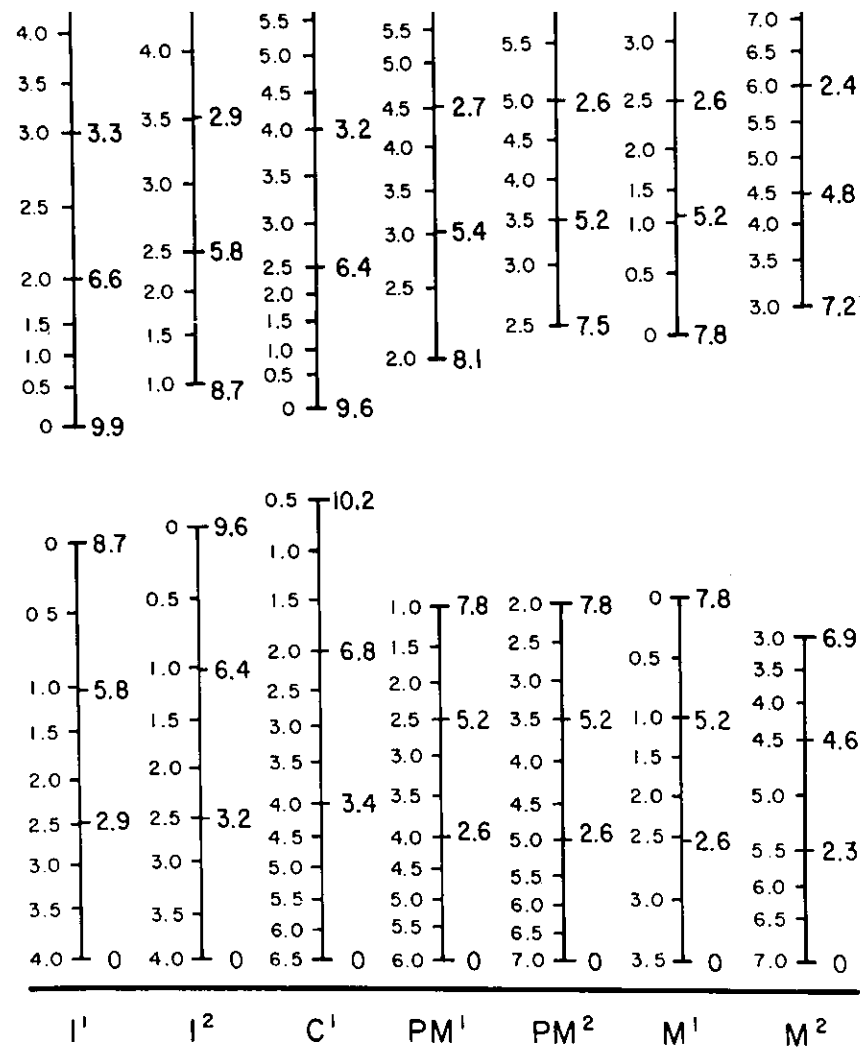


Fig. 3. Crown development diagram for the human permanent dentition used to estimate the age at development of enamel hypoplasias. The numbers to the right of each line are distances from the cemento-enamel junction to the midpoint of the hypoplasia in millimeters. The numbers to the left of each line are the corresponding ages at formation of the hypoplasia. Note that this chart reflects assumptions of variation in intratooth growth velocity. Furthermore, crown heights vary within and between populations. These sources of variation are discussed in the text. (From Goodman et al., 1980, modified from Swärdstedt, 1966, using the developmental sequence of Massler et al., 1941.)

riods, a length of time that seems more appropriate, especially with slow-growing permanent teeth. The point to remember is that the presented data are *estimates of ages at development*, with an error of unknown size and potential biases.

With agreement on the underlying chronology, it is a relatively simple procedure to develop a series of regression equations for converting a location of an enamel defect to an age at formation. In fact, Murray and Murray (1989) recently developed a computer

program that performs these calculations and saves the data as an ASCII file. This program, which is available from the authors, uses the Massler (1941) chronology and employs regression equations developed by Phillip Walker (University of California, Santa Barbara). For example, if we assume constant velocity and that a maxillary central incisor develops between birth and 4.5, then we can compute the age at development of a defect with a first degree polynomial:

$$\text{Age at formation} = \text{age at crown completion} - [(\text{years of formation/crown height}) \times \text{defect height (from CEJ)}]$$

Perhaps the most important issue is that researchers save the raw measurement data (locations of enamel defects from the cemento-enamel junction). If this is done, chronologies can be reconstructed when other standards are developed or a clearer estimate of bias is developed. At that time, parameters of the model may be changed and new estimates obtained.

One important bias involves the determination of the developmental time between initial matrix formation and striae of Retzius reaching the outer enamel. When a tooth begins calcification, the initial stria form partial circles which begin and end in the inner enamel (see Fig. 1). Only after an unknown length of time do the striae of Retzius end near the surface, and it is only at this time that one can determine a chronological record of disruption without sectioning teeth. Stated simply, there is an unknown length of time after initial matrix formation during which disruption can *only* be observed in section. Bromage and Dean (1985) estimate this period of time to be around a half year for permanent incisor teeth.

Individual and population differences in dental development. Even with a standard that meets stringent criteria, one will find variation in calcification timing among individuals and groups. It is possible to control for some of this variation. For example, most dental development studies that have provided separate data on males and females have found that development is somewhat advanced in females (Anderson and Thompson, 1973; Demerjian and Levesque, 1980). If one continues to use a standard such as that provided by Massler et al. (1941), which does not provide separate developmental times for males and females,

unit of enamel) or requires more time to develop (Blakey and Armelagos, 1985) than a smaller tooth. Moss and Moss-Salentijn (1977) suggest that larger teeth require more time for development. However, it may also be that they simply grow more rapidly.

Blakey and Armelagos (1985), for deciduous teeth, and Hodges and Wilkinson (1988), for permanent teeth, have estimated the error involved in estimating age at formation using an average tooth size. Assuming that smaller teeth take just as long to develop as larger teeth, both studies report that in populations where tooth size variation is relatively small, as might be expected in most anthropological populations, the error is minimal, usually less than a half-year. However, Hodge and Wilkinson (1988) suggest that this error might be quite substantial in a mixed series where variation in tooth size is relatively greater.

One of the more perplexing issues to consider is the fact that the same environmental factors under scrutiny, namely nutrition and health status, might affect the chronology of enamel development. It is well established that nutritional status influences the rate of tooth eruption (Alvarez et al., 1988; Alvarez and Navia, 1989). It may, therefore, be assumed that calcification might also be so affected.

In addition to the above noted factors, populations and individuals within populations may differ somewhat in the timing of enamel development for unknown or genetic reasons. Within a population, some individuals mature more rapidly than others. Among populations, some groups seem to have more rapid tooth eruption that is not explainable by environmental conditions alone (Garn et al., 1973a,b; see also Chapter 8, *this volume*).

Research Directions

A number of research projects need to be undertaken in order to better understand the basic pattern and timing of enamel development, as well as the relationship between enamel development and hypoplasia chronologies. One might be somewhat pessimistic about the availability of a newer developmental standard that might not fall victim to many of the shortcomings of prior standards. A radiographically based standard that includes the first months, or even the prenatal period, would be of tremendous value for research purposes, but not for the fetuses and infants exposed to the serial radiographs. Less

length of time that cervical enamel has been forming. They estimate an average of seven days between perikymata, based on assumptions that enamel prism cross-striations are daily events, and that there are seven to eight cross striations, on average, between perikymata. Hillson and Jones (1989) have recently reported on the development of instrumentation to automate the counting of perikymata. If this and similar procedures can be linked to information on the absolute age an event occurs, then they may provide a basis for a highly accurate developmental chronology.

Working with the standards and information at hand, it would be helpful to obtain a better sense of systemic biases and estimates of error. We are close to being able to estimate the affect of sex on formation times and the delay from initial matrix formation to the first occurrence of cervical enamel. On the other hand, we are further away in estimating the role of nutritional differences within and between communities and how to account for variation in tooth size. Basic research into these developmental factors will have implications for the epidemiology of enamel defects.

ENAMEL HYPOPLASIAS AND NUTRITIONAL STATUS

Although enamel hypoplasias of systemic origins are common and easily discerned from defects due to nonsystemic factors, it is difficult to attribute a more exact cause to these defects (Pindborg, 1982). This is true in clinical and contemporary epidemiologic settings (Cutress and Suckling, 1982; Pindborg, 1982) as well as in archaeological series (Rose et al., 1985). Cutress and Suckling (1982) suggest that nearly 100 factors are possible causes of enamel defects. The list of potential causes, including nutritional imbalances, drug toxicities, and almost any disease that severely stresses metabolism, gives credence to the view that enamel development is sensitive to a wide spectrum of physiological and metabolic changes. The great increase in research on the etiology of enamel defects seen in the last decade has perhaps only reaffirmed the earlier notions of Sarnat and Schour (1941) and Kreshover (1960) on the nonspecificity of these defects. Although it is relatively easy to identify chronological or linear enamel hypoplasias, and distinguish this condition from hypoplasias caused by local or hereditary causes, un-

progress has been made, however, in estimating the sensitivity of this defect. The relatively high frequency of defects in individuals who have experienced mild-to-moderate degrees of malnutrition during tooth development suggests that the indicator is relatively sensitive to undernutrition. Furthermore, enamel hypoplasias have a number of characteristics that suggest that they might be quite useful in a number of settings. In studies of contemporary groups, the ease of study and their nonspecificity might make them useful as surveillance level indicators. Similarly, in prehistoric studies, these indicators can provide an overall sense of metabolic state during tooth development.

The chronological and indelible properties of enamel hypoplasias highlighted in this review make them particularly suitable to studies of the long term consequences of early stress or to temporal changes in stress. For example, one might employ enamel hypoplasias as a means for assessing generational or secular changes in nutritional status.

A number of problems exist in trying to use enamel developmental defects as biological assays of past periods of nutritional and disease stress. Some of these problems, such as selection of tooth surfaces for analysis, reliably determining the type of defect, and quantification of dimensions, have not been considered in this review, whereas others, such as determination of sensitivity and specificity and timing of the event, have been highlighted in this review. Despite a number of extant methodological and theoretical problems, we propose that enamel hypoplasias, used in conjunction with other indicators of nutritional and disease status, can provide useful insight into the human condition. Commenting on the state of research into enamel developmental defects in contemporary malnourished children, Jelliffe and Jelliffe concluded an editorial in stating that "further studies of its (linear hypoplasia) etiology and public health consequences seem overdue" (1971:893). Nearly 20 years after this editorial, we have seen a revival of interest in enamel developmental defects, and we may soon understand more fully their potential as nutritional status indicators.

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