Problems in the Methodology of Dental Enamel Hypoplasia Analyses

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Abstract: This article summarizes the issues and problems surrounding the subject of dental enamel hypoplasia (DEH). The methodology of several research studies, ranging from paleopathological to studies of living children, have been analysed and critiqued. The critiques are used to demonstrate where weaknesses may occur in this type of paleopathological study, and how they might be improved. This summary indicates that age formation and enamel development standards, and severity of the enamel disruption must be more carefully and universally applied to paleopathological and anthropological data. Standardized techniques for measurement of DEH are also essential for accurate and reliable research conclusions.

Introduction

Studies of dental enamel hypoplasia (DEH) have become increasingly popular in paleopathological research. DEH studies of both past and present populations have taken place and each has influenced the methodology of the other. In an attempt to discuss and evaluate more fully the issues and problems surrounding the methodology of DEH analysis, it is important to recognize that there are many biases and assumptions which might be introduced into studies of hypoplastic defects. Concepts, classifications, methods, and chronologies are all influenced by these deeply maintained assumptions. The insights anthropologists and other researchers have been able to derive from research in DEH have been limited thus far, as there is yet to exist a consensus among researchers as to the precise modes and rates of formation of teeth and structures within teeth, and population differences one might find among these processes. This lack of consensus contributes further to "variations in the perceptions of what constitutes dental disease and lack of standardized methods for data collection and analysis..." (Lukacs 1989:283). Only when these methods and assumptions are subject to scrutiny will the validity and reliability of the study and interpretation of DEH improve. This paper will focus primarily on the issues and problems surrounding DEH analysis rather than interpretation. There remain many important debates surrounding the interpretation of DEH which lie beyond the scope of this paper. Some of these issues will

briefly be considered, however, when problems in analysis create further problems in interpretation.

Tooth enamel is a durable structure both within the body during life and postmortem. The durability of dental enamel makes it one of the most informative hard tissues for studying adaptations of past and contemporary peoples to their physical and sociocultural environments (Skinner and Goodman 1992:153). Hypoplasia is a condition that is relatively easy to identify macroscopically, and often in the case of archaeological specimens, teeth are the best preserved and most prevalent material available (Hillson 1986:140). "As an indicator of general, non-specific stress it is the paleontologically best preserved portion of the anatomy" (Ogilve *et al.* 1989:25) being the least affected structure in the body during interment (El-Najjar *et al.* 1978:185). And because mature enamel is unalterable by internal biological events (Blakely *et al.* 1994:371; Hillson 1986: 129), hypoplasia may provide a permanent "memory" of chronologic occurrence of environmental stress during childhood (Moggi-Cecchi *et al.* 1994:299).

Hypoplasias are often labelled as indicators of stress and are defined as "deficiencies in enamel composition" (Goodman 1991:281). These defects occur while enamel is developing, and remain as a permanent record of stress into adulthood (Sarnat and Schour 1941). It should be noted that DEH can only occur during a period of enamel matrix formation (El-Najjar *et al.* 1978:186). Linear enamel hypoplasia (LEH) is the most common form recognized by anthropologists (Skinner and Goodman 1992:151). LEH can be described as marked horizontal grooves or undulations of decreased enamel thickness (Goodman *et al.* 1980:518) (see figure 1). These occur as bands running around the crowns of each tooth, and when matched with similar bands on other teeth formed at the same time, can be related to particular episodes or periods during the developmental history of the individual (Hillson 1986:129).

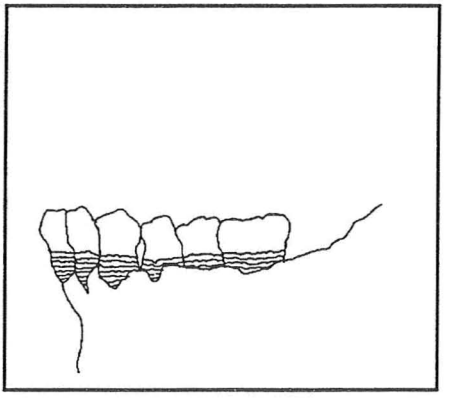

Figure 1. *Dental (Linear) enamel hypoplasia manifest on mandibular teeth.*

DEH has been associated with a variety of disease and nutritional deficiencies (Goodman *et al.* 1984:287). It is often difficult to determine their exact cause as many conditions can result in DEH (refer to Pinborg 1982, cf. Hillson 1986). While a specific cause of a particular hypoplastic defect might not be isolated, the mere existence of a defect indicates a stress of sufficient potency to interrupt normal enamel development (Goodman and Armelagos 1985:479; Blakely *et al.* 1994:372).

The Process of Amelogenesis

Amelogenesis (or enamel formation) takes place in two stages: 1) matrix production and 2) maturation. The matrix (the basic structure of the enamel crown) is formed by the productive actions of a closely linked sheet of cells known as ameloblasts (Hillson 1986:113). In the second stage, the cells take on a resorptive and transport function, removing proteins from the matrix which results in full maturation including hardened enamel (Skinner and Goodman 1992:153). The secondary stage of enamel development occurs shortly after the commencement of the first so that the two occur concurrently throughout the greater part of the period of mineralization (El-Najjar *et al.* 1978:187). Each ameloblast produces matrix for a given thickness of enamel and brings about its maturation (Hillson 1986:114). The first transverse bands (or "rings") form the circumference of the cuspal or incisal aspect of the developing crown, followed by a regular succession of the bands transgressing towards the cemento-enamel junction (CEJ) where crown development ends (Blakely *et al.* 1994:371).

Primary structures of enamel are called prisms. Prisms are created by the combined activities of four adjacent ameloblasts which secrete a keyhole-shaped rod that elongates away from the dento-enamel junction (Skinner and Goodman 1992:153). Enamel is initially deposited in successive increments that cover the previous layer completely and can be described as domes (Hillson 1986:122). Successive sleeves of enamel are deposited around the last dome, overlapping towards the cervical part of the crown (Skinner and Goodman 1992:154). The sleeves become narrower and thinner as the crown nears completion (see figure 2).

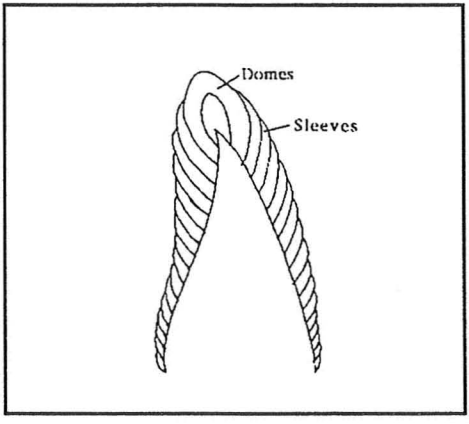

Figure 2. *Layering of enamel.*

Amelogenesis, as with all growth, is subject to pathological and physiological disturbances (Hillson 1986:130). DEH is the result of disturbances to ameloblasts during enamel matrix production (Skinner and Goodman 1992:154) when the cells which are nearing completion of matrix production switch over to maturation earlier than normal (Hillson 1986:130). In linear DEH this can form a relatively wide, deep groove in the enamel. Environmental structural anomalies, like DEH, are primarily horizontally patterned, as opposed to genetic anomalies, which are vertically arranged (El-Najjar *et al.* 1978:185).

Methods

The first problem in the analysis of DEH is defining its presence. Many different researchers employ equally as many different operational definitions of the condition. According to Skinner and Goodman (1992:157), "enamel hypoplasia occurs on the external surface of the teeth as reduced enamel thickness macroscopically visible as more or less confluent horizontal pits or actual grooves." Another definition is provided by El-Najjar *et al.* (1978:187) who describe it as a "transverse line on the enamel surface... [which] can be read micro-macroscopically...." While Goodman and Armelagos (1985:482) described "circumferential lines, bands or pittings of decreased enamel thickness...," and Blakely *et al.* (1994:374) refer to it as "transverse linear reduction in enamel thickness... [where] moderate/severe hypoplasia (or MGA) are relatively deep/wide defects... [and] slight hypoplasias (LEH) are normal/shallow lesions...." Finally, DEH is defined as "clear developmental defects of the dental enamel..." by Ogilve *et al.* (1989:26), whereas Hillson (1986:129) sub-defines two types, in which M-hypoplasia appears as isolated pits, grooves and washboard defects and irregular pitting is referred to as Ghypoplasia. Multiple definitions of its presence can therefore create some confusion in analyses of DEH.

This non-universality of the definition of DEH makes for awkward interstudy comparisons. One primary problem is the lack of means for describing a gradient of severity of hypoplastic defects (Skinner and Goodman 1992:157). There are also few published tests of intra- and inter-observer error in recording the presence, location and size of enamel defects (Skinner and Goodman 1992:157). Various scoring systems have been devised, including one by Sarnat and Schour (1941), which distinguishes between narrow and wide, smooth and pitted defects, occurring singly or in multiples. There are some new scoring indices currently gaining popularity. One used in contemporary studies known as the DDE, was developed by the Federation Dentale International (Hillson 1986:135). It might also prove valuable to archaeologists. Another index was devised by Hargreaves *et al.* (1989). Called the Hypocalcification/Hypoplasia Index or HHI, it was used to describe defects in groups of living people. It classifies the hypoplasia or hypocalcification by assigning a number to the degree of severity (1 through 9). This index was found by researchers to be easy to interpret and apply plus allow for the rapid assessment of individuals.

Once the the DEH has been operationally defined by the researchers, identification and analysis can begin. Surface defects can be described fairly unequivocally in terms of defect type, number of demarcations, and location (Skinner and Goodman 1992:157). Hillson (1986:132) claims that the usual method for diagnosis is to run a sharp dental explorer or probe over the tooth surface, and suggests that all observations should be carried out under bright, oblique lighting to accentuate any unevenness in the crown surface. Lukacs (1989:267) describes the optimum method of macroscopic hypoplasia analysis. He claims it is best done with a 10x hand lens and dental probe "and [that] the position of the defect on the tooth crown, type of hypoplasia (linear, pitted or both), and the surface of the crown affected should be

recorded. Linear hypoplasia distance should be measured from the CEJ with Helios dial calliper rounded to the nearest tenth of a millimetre" (Lukacs 1989:267). No mention is made about the type or positioning of a light source. Roberts and Manchester (1995:60) claim that the recording of enamel defects is usually undertaken microscopically using a binocular microscope with "good" lighting.

These descriptions assume all researchers are working under the same conditions, or with the same technology. In practice, this is often not the case. Moggi-Cecchi (1994) describes how he and his colleagues measured the distance of the initial and final margins of each groove from the CEJ using a digital calliper with 0.05mm precision, but only used a 4x magnifying glass. Goodman and Armelagos (1985:482) confirmed the presence of a defect using a binocular microscope. Goodman and colleagues (1980) used the Helios calliper 0.01 millimetre rule, but observed defects with the aid of a zoom binocular microscope. Hargreaves *et al.* (1989:128) examined the teeth of living children under either natural light or anglepoise 60 watt lamps and used sickle probes. In his study of living children in rural Pakistan, Lukacs (1991) was aided by the use of a small flashlight. In this study, no magnification was used and no attempt was made to record the position of the lesion on the surface of the crown (Lukacs 1991:515). He noted the difficulty of observing minimal expressions of the defect in the field without the aid of magnification (Lukacs 1991:519).

Some researchers achieve success using comparative casts rather than examining the teeth directly (Hillson 1992; Ogilve *et al.* 1989). A recent study (Propst *et al.* 1994) has shown that recording defects is easier, more productive and more accurate when performed on casts rather than teeth. As Hillson (1986:136) points out, "more precise methods of the location of hypoplastic bands could be obtained from microscopic examination of the tooth surface or casts of it." This would allow the perikymata to be counted between hypoplastic episodes and the last dome/ first sleeve or CEJ (Hillson 1986:136). These figures would then be directly comparable to data for other teeth.

As noted, investigators use various methods in the recording and identification of DEH. Some use casts, others use teeth directly. Some use probes, lights and microscopes, while others do not. Data should be reported by tooth to ensure comparability (Goodman and Armelagos 1985:491). As Lukacs (1989:273) states, "imprecision can be avoided only if investigators carefully specify the methods used in their research." Only then will results be suitable for comparison.

An important factor in the discussion of DEH analysis is the materials. The materials, in this case, are the teeth themselves. For instance, which teeth are examined, their condition, and in what numbers they exist in the sample are important factors which can greatly influence study results. It appears as though different teeth have varying sensitivity to stress. Lingual enamel, thin enamel and smaller teeth may be less vulnerable to DEH (Skinner and Goodman 1992:163). Also, "anterior teeth are more hypoplastic than posterior teeth" (Goodman and Armelagos 1985:479). Goodman *et al.* (1980:526) noted that maxillary central incisors and mandibular canines are the most frequently hypoplastic. Some authors claim

it is important to gather information from all available teeth in each individual (Hillson 1986:139). Others study only one side of the dentition or its antimere (e. g. Goodman 1988; Moggi-Cecchi *et al.* 1994; Ogilve *et al.* 1989). In the case of Ogilve and colleagues (1989:27), antimeres were treated as separate data points though it artificially inflated the sample sizes. Goodman and his colleagues (1980:526) suggest that future hypoplastic analysis might be limited to the two maxillary incisors and two mandibular canines, saving great time and allowing for minimal loss of potential information.

Early hypoplasias form somewhat occlusally on the surface on the tooth (Skinner and Goodman 1992:158). Often, the occlusal/incisal surfaces of the teeth are worn. This enamel attrition can affect the description and analysis of DEH. Most studies attempt to avoid including heavily worn teeth in their calculations and results (e.g. Goodman *et al.* 1984a; Moggi-Cecchi *et al.* 1994), other studies do not even make mention of this possible circumstance. Goodman (1989:270) admits that the paucity of defects on the incisal/occlusal thirds of the teeth in his study could be due to the affects of attrition. Blakely *et al.* (1994:374) used moderately, but not severely worn teeth. Goodman (1989:266) examined all teeth, but took into account pre- and postmortem tooth and enamel loss so that overestimation of the enamel available for study would be avoided.

In making casts of the teeth of Neandertals, Ogilve *et al.* (1989:26) avoided moulding the CEJ to prevent damage to the roots or alveolus. They also included all anterior teeth that had 3.0 mm or more of crown remaining. For both of these reasons, a great portion of the enamel surface of their materials could not be examined, potentially biasing the results of the study.

There is a tendency to report results without explicitly stating the procedure employed in calculating DEH prevalence (Lukacs 1989:273). Several authors debate the issue of which teeth should be included in the studies of hypoplasias in past and present populations. There is a problem with the use of multiple teeth, such as canines and incisors, in calculations to infer age of stress. The different teeth yield two different peak ages at stress in a single population (Skinner and Goodman 1992:166). Most researchers use a variety of teeth, or all those available for study. Some studies examine only a specific class of teeth, such as the deciduous canine. A study such as this can be used to demonstrate localized rather than systemic stress upon an individual or population. Data from multiple classes of teeth have to be calculated in order to demonstrate a systematic stress evident in enamel defects. It is important when using the tooth as a unit of analysis, not to translate a peak or trough in the occurrence of enamel hypoplasia directly into a time of heightened or lessened stress (Skinner and Goodman 1992:164). This problem can be minimized by using a few selected teeth or, preferably, the individuals as the unit of analysis (Skinner and Goodman 1992:164). It is evident that choice of tooth or teeth can significantly affect the results of an analysis.

Various methods have been devised for recording the position of the defects on the crown and then converting this measurement into age (e.g. Massler *et al.* 1941; Sarnat and Schour 1941). "Perhaps the most important factor in the determination

of the chronology of enamel hypoplasia refers to the choice of the developmental standard and how it is interpreted and employed" (Goodman and Rose 1991:287). However, the interpretation of the chronological distribution of hypoplasias is controversial (Goodman *et al.* 1984b:260). The validity of the choice of tooth/teeth and the choice of chronology depends upon whether or not there are reliable standards of development for these teeth. Anthropologists generally use relatively vintage standards for the stages of development of dental enamel (e.g. Massler *et al.* 1941; Sarnat and Schour 1941). In Sarnat and Schour's paper (1941:1996), age at development of defects was determined by noting the "exact position and extent of aplastic enamel defects," and comparing these with a tooth development standard. Massler *et al.*'s (1941) method of estimating the developmental age of individuals at the time of line formation (i.e. stress) "divides the tooth crown into half-year developmental periods of equal width, corresponding to the time of crown development" (Goodman and Armelagos 1985:482) (see figure 3). The chronology by Massler *et al.* (1941) is acknowledged only to provide an estimate of age at time of formation of DEH (Goodman 1989:267). Only when appropriate and effective standards are established can researchers create and utilise accurate chronologies.

In the past twenty years there have been at least five DEH chronologies published based on permanent teeth from archaeological and non-industrial populations and all use the same developmental standard as Sarnat and Schour (1941) (Goodman 1988:783). Goodman (1988:783) has concluded on the basis of his results, that it is unlikely that the chronology of defects reported by Sarnat and Schour (1941) is an accurate estimate of the chronology of defects from most populations. He disagrees with those who feel the Sarnat and Schour (1941) chronology is reflective of all industrialized populations. The chronology used by Goodman *et al.* (1980) assumes a constant pattern of crown formation timing and constant crown dimension, which may not be entirely safe assumptions (Hillson 1986:134). It appears as though additional chronologies of tooth formation are sorely needed.

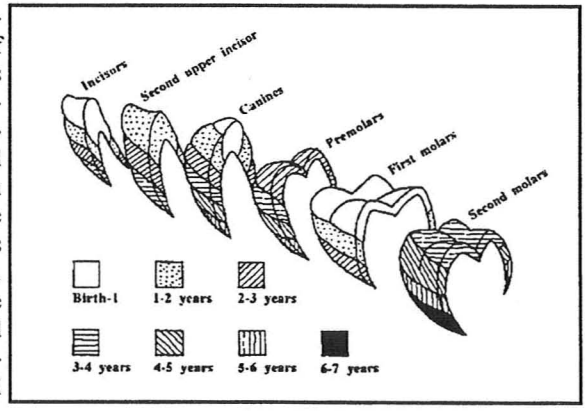

Figure 3. *Permanent crown formation (drawn from Massler et al. 1941).*

Another problem with the use of standard chronologies is that standards may not be accurately representative cross-culturally. Both Tompkins (1996) and Owsley and Jantz (1983) have found that differences do exist between modern populations of different biological affinities. Tompkins (1996:97) looked at tooth eruption and

calcification "schedules," and found that black southern Africans, French-Canadians and Native Americans followed different formation patterns. The author concluded that these differing patterns resulted from different relative development between tooth type. The Arikara Indians whom Owsley and Jantz (1983) studied had some teeth, including maxillary incisors, which appeared older by 0.5 to 1.1 years than the formation standards used for American whites. These observations were systematic, implying that they reflect population differences in tooth-formation timing, and not simply differences among individuals (Owsley and Jantz 1983: 466). When groups of different biological affinity are compared using the same tooth formation standard, one group may erroneously appear to be delayed or advanced in its development relative to the others. Obviously, the assessment of ages in studies of DEH are complicated by these type of results.

Scott and Symons (1974) have developed a more recent crown formation age chronology. New radiological standards based on modern criteria for sample representativeness are being sought. In addition to this, the age of defects could be exactly determined from the incremental structure sequences of enamel (perikymata) when viewed in profile in sections under the microscope (Hillson 1986:136). These techniques "seem to provide age estimates for crown formation that are significantly different from those provided by earlier workers" (Skinner and Goodman 1992:165). It should be noted that there are different standards for the formation of permanent and deciduous teeth. Therefore, the appropriate standards must be applied when examining evidence of DEH in these two very different types of teeth. Deciduous dentitions can demonstrate stress prenatally, since teeth begin to form even before birth.

In studies of dental paleopathology, the method of data presentation is often not explicitly stated (Lukacs 1989:271). A prime technique used in this type of analysis is the calculation of frequency of individuals in a sample exhibiting a particular dental lesion. This frequency is obtained by dividing the number of individuals with at least one case of DEH by the number of individuals that could have yielded evidence of the disease (Lukacs 1989:273). This technique is known as the individual count method, and should be reported for each sex and age subset of a skeletal sample in order for the statistics to have any anthropological or comparative value (Lukacs 1989:273). Attrition on the teeth of older individuals may influence the appearance of the distribution of DEH in the population. This is known as the "cohort effect"; only those adults who die young would be selected for analysis, a choice that would result in the false inference of high levels of childhood stress within a population (Skinner and Goodman 1992:168).

In the study of DEH, particularly LEH, researchers may want to calculate the number of lines or grooves on teeth. These calculations can be transformed into number of hypoplastic episodes per age in years per individual, or per tooth (Goodman and Armelagos 1985). This second type can demonstrate the sensitivity of a tooth relative to another (Goodman and Armelagos 1985:485). The first type is considered to be especially valuable in studies attempting to prove systematic childhood stress, such as weaning (e.g. Corruccini *et al.* 1985; Goodman *et al.* 1984a; Moggi-Cecchi *et al.* 1994). Goodman *et al.* (1984a:285) also calculated the mean

number of hypoplasias per individual. This can show the prevalence of this type of defect within a population. Authors have often conducted statistical analyses of differences in sex or age distribution. If the results appear to be statistically insignificant, the data can be pooled for further analysis.

Assumptions

The methodologies of those who studv DEH are influenced by many assumptions. A review of these assumptions will highlight those which are reasonable and those which may not be accurate. The first, and most basic assumption many researchers hold in studying DEH, is that these defects accurately and reliably reflect periods of environmental or disease stress during the period of enamel formation. Variations in DEH resulting in stress between populations are assumed to be due to variations in the experience of a physiological disruption (Goodman *et al.* 1984a:277). "These, in turn, are assumed to be a function of the amount of cultural buffering and host-resistance subtracted from the severity of culturally and ecologically produced constraints" (Goodman *et al.* 1984a:278). It should then follow that DEH will result from all periods of childhood stress during the time of enamel production, and that these defects will/can be accurately read to provide reasonable estimates of time, duration and severity of stress (e.g. Corruccini *et al.* 1985; El-Najjar *et al.* 1978; Goodman *et al.* 1984a).

This first assumption is not necessarily valid. While there is a well recognized link between episodes of malnutrition and marked groove defects on the anterior teeth, it is not always easy to link DEH to specific episodes of stress (Hillson 1986:130). Sarnat and Schour (1941) found that in only half of the cases they examined could hypoplasias be matched with a specific occurrence of disease. "It is not entirely clear how malnutrition and fever, for example, affect the normal secretory activities of ameloblasts; consequently, we cannot predict which particular stressors will produce enamel hypoplasias" (Skinner and Goodman 1992:160). It is also unclear what size and form of the DEH have to do with the causal episode (Hillson 1986:139). Therefore, "interpretations of defects in ancient hard tissues are inferences rather than precise diagnosis. They are probability statements based on the best available scientific evidence" (Skinner and Goodman 1992:160).

At this point in the discussion, aspects of Wood and colleagues' (1993) "Osteological Paradox" need to be considered. Two osteological populations or groups may have low rates of DEH, one because they were rarely stressed, the second because they were so consistently unhealthy they never rebounded to increased ameloblast production while enamel was being produced. In both of these cases, low prevalence of DEH in the skeletal population would exist, but it would be difficult to interpret which was the stressed population. Thus, problems in the methodology of interpretation, and not merely analysis, have serious implications for the study of DEH.

One valid assumption most authors make is that evidence of hypoplasia in multiple teeth in an individual dentition probably reflects systematic rather than local stress.

Once the appropriate standard of enamel formation is chosen, systematic stress can be demonstrated in the appearance of DEH lesions on multiple teeth, at enamel levels that indicate a similar time in development.

The issue of susceptibility, which occurs both within and between tooth classes, must be considered when investigating methodology. This variability in appearance is reflected in the types of teeth researchers choose to examine. Goodman and Armelagos (1985:491) suggest that there are differences in susceptibility to hypoplasias among teeth developing at similar times. They contest a commonly held assumption that all teeth developing at the time of a disruption are equally likely to develop a hypoplasia (Goodman 1989:270). This contested "time of development" hypothesis maintains that the frequencies of hypoplastic defects are dependent upon which crowns are developing at the time environmental insults are the most active (Goodman and Armelagos 1985:481). The assumption underlying this hypothesis is one of a biological process of constant magnitude and response to physiological disruption among all teeth (Goodman and Armelagos 1985:480). A number of studies have demonstrated that some teeth are more susceptible to DEH than others, even when developing at the same time (e.g. Goodman *et al.* 1980).

In general, the anterior teeth show more enamel defects than the posterior teeth (Goodman 1988:788; Hargreaves *et al.* 1989:128). Goodman and colleagues (1980:526) found that canines provide the best records of stress from ages 3 to 6.5 years; whereas, from birth to 3 years the incisors generally (maxillary specifically) are best. In this same study, the authors found that no information would have been lost had the premolars and molars not been examined, since hypoplastic lines found on these teeth were matched to more severe lines on anterior teeth. Hargreaves *et al.* (1989:128) found that maxillary central incisors showed the most enamel defects. In a further demonstration of between teeth susceptibility, Lukacs (1991) found that localized hypoplasia of deciduous canines occurs more frequently in the mandible than in the maxilla.

Skinner and Goodman (1992:491) suggest that researchers concentrate on only a few easily accessible and frequently hypoplastic teeth, such as the maxillary central incisors and mandibular canines. These recommendations emerge as a result of other studies (i.e. Goodman 1988; Goodman and Armelagos 1985). They state, "the formation of a gradient of susceptibility may provide a method of estimating severity of physiological stress. The appearance of hypoplasias on less susceptible teeth may be more indicative of a more physiological disruption" (Skinner and Goodman 1992:489). As discussed earlier, the choice of teeth for examination and analysis is crucial and can lead researchers to interpretations never before considered.

This difference in susceptibility is important. Deciduous anterior teeth develop earlier than deciduous posterior teeth. Despite overlapping times of development, earlier developing teeth have earlier peak frequencies than later developing teeth (Goodman 1988:788). Thus, anterior teeth, which are developing during periods in which host resistance is low and environmental insults are great (as they are during early childhood), are more likely to be hypoplastic (Goodman and Armela-

gos 1985:489). This holds implications for the interpretations of results which seem to indicate that most hypoplastic defects occur between the ages of two and four (or weaning age).

Goodman and Armelagos (1985) propose an explanation for the differences in hypoplasia frequency by tooth, suggesting that more developmentally stable teeth will be more susceptible to ameloblastic disruption. The more stable teeth (ie. upper central incisors) are genetically unlikely to grow larger or smaller, and enamel hypoplasias may be the only available response to environmental disturbance (Goodman and Armelagos 1985:490).

Turning to within tooth susceptibility, data collected on the location of hypoplasia on the tooth crown itself has demonstrated that it is not randomly distributed within tooth crowns either. These distributions appear to be similar for all tooth crowns, regardless of time of development. The highest frequencies of hypoplastic defects occur in the middle third of the crown (Goodman and Armelagos 1985:488). The lowest frequencies occur on the incisal/occlusal third. In not acknowledging these susceptibility results, many researchers have made erroneous assumptions. If only certain teeth were examined it might falsely appear as though certain specific stresses, such as weaning, cause the most severe enamel defects between the ages of one and four.

Katzenberg *et al.* (1996:184) argue that the commonly held assumption that weaning stress results in increased periods of DEH formation is likely to be an artifact of "coincidental associations between 'calculated' ages of occurrence of the two phenomena." They point out that in many studies weaning stress and DEH formation are simultaneous, while in others the stress precedes the markers. Also, researchers often never discuss the reasoning behind the assumption that weaning actually occurs between the ages of one to four years when associating it with DEH.

There is an explanation for these within-tooth frequency chronologies. According to the pattern of crown formation, enamel is laid down initially in dome-like increments at the incisal edge. The newer increments bury those formed earlier, along with any environmentally stress-cased defects that might have formed within them. This results in observations like those by Skinner and Goodman (1992:165). The first year and a half of incisor and canine crown formation are not expressed on the surface of the tooth from a single individual. "This phenomenon seriously weakens previous efforts to infer timings of occurrence, since customarily the most occlusal level has been taken to coincide temporally with initial mineralization that has been detectable radiologically" (Skinner and Goodman 1992:164). Researchers have to be careful when considering the peak-ages at occurrence of hypoplastic defects.

It is also suggested by some authors that all teeth display similar within-tooth hypoplastic patterns due to common morphological or physiological factors. These include within-tooth variation in the rate of enamel apposition, prism length, prism direction angle and number of secretory ameloblasts at a certain location on the tooth crown (Goodman 1989:270). It appears as though long prisms are most

likely to be associated with enamel defects (Goodman and Armelagos 1985:482). The longest prisms occur in the middle third of the tooth. Formation slows down in the cervical region of the tooth, and this area is therefore likely to have an increased susceptibility to stressful episodes. Yet this cervical enamel also becomes increasingly thin and it becomes progressively difficult to discern and score hypoplasia with confidence as one nears the cervix of the tooth (Skinner and Goodman 1992:165).

One study of modern children has shown conflicting results. Hargreaves *et al.* (1989:128) found that DEH of the incisal/occlusal third of a crown is the most dominant condition in a study of modern South African children. This raises questions about further assumptions "since changes in susceptibility to stressors are assumed to be a relatively constant biological phenomena, this position implies that the distribution of hypoplasias should be similar in all populations" (Goodman *et al.* 1984a). Some studies (especially those dealing with weaning) and researchers (e.g. Neiburger 1990) assume that standards for crown formation were the same in the past as they are today, and that there is no variation between and within populations of different time periods and geographic areas (Roberts and Manchester 1995:60). Ogilve and colleagues (1989:27) assume that Neandertal crowns took the same amount of time to develop as do those of modern humans, despite the fact that they are much larger when complete. In one study, Moggi-Cecchi *et al.* (1994) assumed that the skulls of unclaimed indigents they examined were representative of the population they wished to investigate. However, as Goodman (1988) has shown in his reappraisal of the Sarnat and Schour (1941; 1942) chronologies, two populations are not necessarily similar in their distribution of DEH.

The last problem in the methodology of DEH analysis is the assumption surrounding the sudden diminution of hypoplasias during the middle of childhood (around age 6-7 years). This diminution is often interpreted as the beginning of decreased episodes of stress, but actually reflects a sharp decline in scorable enamel (Ogilve *et al.* 1989:32; Skinner and Goodman 1992:167). It is this time in childhood that enamel formation slows and finishes: no more hypoplasias can develop (Skinner and Goodman 1992:167). It is, therefore, possible that methodological patterns are creating "the perceived problem of weaning" (Goodman and Skinner 1992:167), despite an inevitable "heaping up" of observations of hypoplasia at age three. These authors claim that periods of peak stress are most likely to be a statistical artifact of flawed methodology.

Conclusions

The large number of possible causes of DEH prevents the attribution of a more specific cause in individual cases. Despite this fact, Skinner and Goodman (1992:162) think that the comparison of DEH prevalence among prehistoric and modern samples is both valid and informative. DEH may provide a general index of infant-childhood health (Sarnat and Schour 1941). A non-specific indicator of stress can be valuable to anthropologists in order to interpret the adaptive and

functional consequence of such stressors. While there may be a lack of perfect prediction from a measurement in individual cases, this should not preclude its use on a population level (Goodman 1991:462).

Despite the problems with methodology, the study of DEH is still a valuable and informative tool for understanding the health of past and present populations. There are solutions to these methodological problems in DEH studies. An important first step is for anthropologists "to be able to classify a variety of pits and grooves encountered, both for descriptive and etiological purposes" (Skinner and Goodman 1992:157). A methodology must be developed for the study of DEH that is "easily performed, is repeatable and is a valid indicator of stress" (Goodman *et al.* 1980:526). Perhaps an index like the Federation Dentale International's (1983) DDE or Hargreaves *et al.* (1989) could be systematically employed by all researchers studying DEH. Furthermore, data should be reported by tooth to ensure comparability between teeth with high variability (Goodman 1988:789; Goodman and Armelagos 1985:491).

A potential source of error can be eliminated by controlling for within and between tooth variation in susceptibility of enamel to hypoplastic disruptions (Goodman and Armelagos 1985:491). As Blakely and colleagues (1994:380) note, "structural and random factors must be methodologically considered in addition to nutritional and disease variables that influence the observed age distribution." Differences in susceptibility of teeth must be taken into consideration when comparing data from different populations, as data from different teeth are not strictly analogous (Moggi-Cecchi *et al.* 1994:302).

It must be reasserted that diseases and nutritional disturbances affect only those portions of the enamel being formed and calcified at the time of stress (El Najjar *et al.* 1978:189). "When a study seems to show different classes of teeth yielding different ages at peak stress, we should perhaps question our methods rather than the sample" (Skinner and Goodman 1992:166). This is demonstrated by the "heaping up" problem. To solve this problem, a researcher might separate the analysis of first and subsequent LEH on single teeth from individuals, then compare the chronological distribution of each (Skinner and Goodman 1992:169).

If comparative studies of dental pathology in modern and archaeological samples are to yield meaningful conclusions, results that are not reproducible and high levels of inter-observer error cannot be tolerated (Lukacs 1989:264). Comparability of growth disruptions, such as DEH will not be obtained until several factors are addressed. These factors include some issues mentioned in this paper: the age structures for samples must be similar, appropriate standards of enamel crown development must be used, minimal severity needs to be standardized, the same type of tooth should be studied, and criteria for the conversion of hypoplasias to growth disruptions need to be standardized (Goodman *et al.* 1980:527). Once these steps are taken, the application of dental enamel hypoplasia studies will become ever more prevalent and useful in paleopathology and other disciplines.

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