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# Assessment of Systemic Physiological Perturbations From Dental Enamel Hypoplasias and Associated Histological Structures

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*ABSTRACT* Dental enamel hypoplasias are deficiencies in enamel thickness resulting from physiological perturbations (stress) during the secretory phase of amelogenesis. The results of a wide variety of experimental, clinical, and epidemiological studies strongly suggest that these defects and their associated histological abnormalities (such as accentuated stria of Retzius and Wilson bands) are relatively sensitive and nonspecific indicators of stress. Because of the inability of enamel to remodel, and the regular and ring-like nature of their development, these defects can provide an indelible, chronological record of stress during tooth crown formation. For these reasons, along with the ease with which they are studied, enamel hypoplasias have been increasingly employed as indicators of nutritional and disease status in paleopathology, and their study has begun to extend into other subdisciplines of physical anthropology.

In order to provide the reader with a better understanding of the current issues in this field, we first review normal enamel development, historical advances in the study of enamel developmental abnormalities, and provide a threshold model to help conceptualize the etiology of enamel developmental defects. Specific attention is then centered on extant, fundamental issues in the use of enamel hypoplasias and histological structures as epidemiological indicators of nonspecific stress.

Most enamel hypoplasias are associated with abnormal histological changes (accentuated stria of Retzius or "Wilson" and "Cluster" bands). However, the lack of association of some mild surface irregularities, characteristically seen as thin, perikymata-like surface depressions, with abnormal prism morphology suggests that these surface features may *not* be evidence of physiological perturbation.

Methods now exist to reliably identify both histological and enamel surface defects. However, further research is needed on methods for determining the size of defects and the epidemiological significance of defect widths and depths. Similarly, the general relationship between the location of enamel hypoplasias and associated histological structures on the one hand, and an individual's age at the time of their development on the other hand, is also well understood. However, better estimates of intra- and inter-population variation in the timing of enamel matrix formation are needed

before these defects can reach their full potential as chronometric measures of stress.

Lack of understanding patterns of differential susceptibility of enamel to developmental disruption has likely hindered interpretations of the results of a number of past experiments. The seemingly strong pattern of differential susceptibility of enamel to disruption—within teeth and across tooth classes, dentitions, and taxa—may yield a number of significant clues to understanding basic issues in enamel development.

Populations that are exposed to a high degree of undernutrition and disease, from prehistoric to contemporary times, share high rates of linear enamel hypoplasias. While these defects seem to relate to bouts of undernutrition and infection, their specific etiology is still unknown. In the next decade we expect to develop more precise information on the specificity and sensitivity of secretory ameloblasts to disruption. A variety of research directions are suggested for further anthropological study.

> The growing dental structures, particularly the enamel and dentin, yield accurate, prompt, and permanent records of both normal fluctuations and pathologic accentuations of mineral and general metabolism. Fortunately, these records are easily read, by virtue of the orderly and rhythmic growth of these tissues in their daily ringlike succession. . . . . In enamel hypoplasia, the history of systemic disturbances is indelibly recorded by a cessation of ameloblastic activity (Sarnat and Schour, 1941:1989).

> Thus, the enamel and dentin in the formative and calcifying stages of their growth serve as kymographs on which are permanently recorded the physiologic or pathologic changes in metabolism that occur within the organism. This concept has been verified many times by histologic studies of the teeth of experimental animals and of man (Massler et al., 1941:36).

Linear enamel hypoplasias (LEH) and associated histological abnormalities have long been related to systemic physiologic disruptions during times of tooth development. A half century ago, Schour, Kreshover, and their colleagues clearly recognized that enamel's ease of observation, sensitivity, inability to remodel, and chronologic developmental pattern rendered it the perfect tissue for indelibly recording changes in physiology during development (Kreshover, 1940; Massler et al., 1941; Sarnat and Schour, 1941).

In the last half century a literature relating enamel quality to a variety of physiological disruptions (stress) has accrued at an increasing velocity, while the use of enamel defects as epidemiological indicators of physiological disruptions has spread through oral biology and into other fields of dental research (Suckling, 1989). Physical anthropologists frequently employ enamel defects to make inferences about patterns of development and stress across millennia and within populations. They have recently applied enamel defects as indicators of levels of stress in captive and free-ranging primate species (Skinner, 1986; Vitzthum and Wikander, 1988) and prehistoric hominids (Molnar and Molnar, 1985; Ogilvie et al., 1989). Enamel hypoplasias and other enamel developmental defects are most regularly used in a variety of analyses of skeletal and archaeological samples (e.g., Blakey, 1987; Blakey and Armelagos, 1985; Corruccini et al., 1985; Duray, 1990; Goodman, 1988, 1989; Goodman and Armelagos, 1988; Hodges, 1987; Hutchinson and Larsen, 1988; Lanphear, 1990; Mack et al., 1988; Powell, 1988; Smith and Peretz, 1986; Storey, 1988; Van Gerven et al., 1990; Yamamoto, 1988, 1989). Furthermore, enamel hypoplasias might be particularly useful in ascertaining pat-

terns of stress during early development in contemporary populations (e.g., Goodman et al., 1987; Hargreaves et al., 1989a,b; King, 1989).

As enamel is easily observed and studied in living as well as prehistoric individuals, analysis of enamel hypoplasias might provide those studying adaptation to marginal environments with a means for evaluating past levels of physiological perturbation (stress). Furthermore, because both deciduous and permanent tooth enamel is formed during critical developmental periods, the second trimester to about 10 years of age, information gained from such an analysis might be uniquely valuable.

In a broad sense, the biological basis and methodology for relating enamel hypoplasias (deficiencies in enamel thickness) to perturbations during tooth crown development has long been confirmed (Kreshover, 1940). However, there are a number of extant methodological and interpretive problems, along with more fundamental issues in enamel development, which have not yet been fully appreciated or studied (Goodman and Rose, in press; Suckling, 1989). What, for example, is the best method for estimating the age at development of a linear enamel hypoplasia, and how great is the error in such an estimate? What is the epidemiological significance of the width and breadth of an enamel hypoplasia? Can we ever ascribe a specific etiology to these defects? How long must a stress be imposed in order to form an enamel hypoplasia? This paper seeks to review these and other questions.

Because it is important to understand the underlying cellular mechanisms that lead to a surface defect, this review highlights the relationship between histology and surface manifestations. We further focus on enamel hypoplasias which are thought to be caused by systemic perturbation (see classification section below). While a review of enamel hypocalcifications (or opacities, the other main class of developmental defects of enamel) is surely warranted, enamel hypoplasias have been far more frequently studied in archaeological and anthropological populations and their etiology is more clearly understood and related to systemic perturbations (Suckling, 1986, 1989).

The specific purposes of this review are 1) to provide the reader with an historical overview of the study of enamel defects and an understanding of the current state of research on enamel hypoplasias and associated abnormalities; 2) to describe the relationship between histological structures and surface defects; 3) to outline current issues in method and interpretation; 4) to suggest directions for future research; and 5) to provide a sense of the potential contributions of this field of research to understanding both fundamental issues in enamel development and patterns of stress in humans and other species.

## ENAMEL HISTOLOGY

### *Normal enamel development*

Enamel, the whitish covering of the anatomical crowns of teeth, is the hardest tissue in the human body (Shawashy and Yaeger, 1986). Fully formed, mature enamel is totally acellular and almost completely composed of inorganic salt (97 + %), with small traces of remaining protein and water. Once formed, enamel is an essentially inert tissue, well suited to its role in mastication. The specialization of enamel, and the ameloblasts which make enamel, may explain its high degree of sensitivity to physiological perturbations.

Enamel organs (or tooth buds) are sequentially formed from the interaction of ectodermal and mesenchymal tissues. Continuing from the 6th week in utero to the 8th year, each of the deciduous and permanent tooth classes sequentially commences development through a series of morphological stages (bud, cap, bell). Toward the end of the bell stage, the inner enamel epithelium begins to reflect the eventual shape of the dentin-enamel junction (DEJ). The cells of the inner enamel epithelium undergo a series of differentiations in becoming tall, secretory *ameloblasts,* the enamel-forming cells. At this stage the underlying *odontoblasts,* the dentin-forming cells, have also sufficiently differentiated to begin the process of

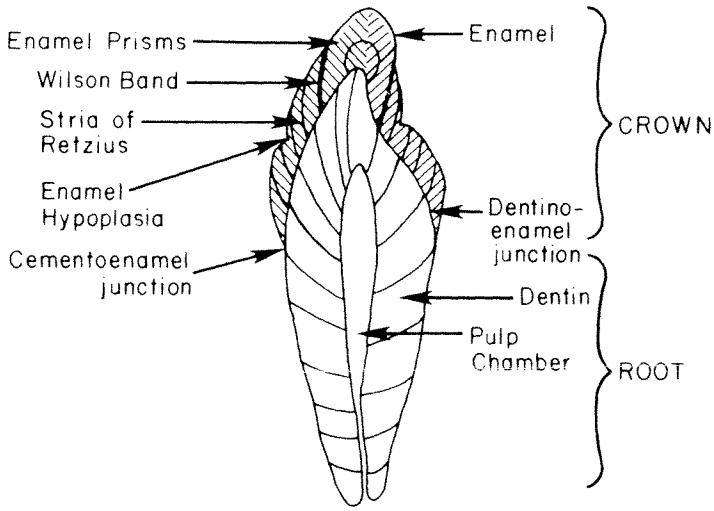


Fig. 1 Diagrammatic representation of a longitudinal section through a human mandibular canine. Note the major structures of enamel: enamel prisms and striae of Retzius (from Rose et al., 1985:283).

forming dentin, the tissues underlying enamel on the tooth crowns, and dentin on tooth roots (Fig. 1).

Although a variety of specific questions relating to the control of enamel formation have yet to be answered, its general pattern is well understood (Jenkins, 1978; Warshawsky, 1985). Enamel formation commences as an inductive process in which the cells of the inner enamel epithelium, while differentiating into ameloblasts, line up along what will eventually become the dentin-enamel junction (Fig. 1). Enamel and dentin formation begins at the border between ameloblasts and odontoblasts. At a time which appears to be under strong genetic control, the odontoblasts begin to secrete the dentin matrix, or predentin, and somehow stimulate the ameloblasts to follow in secreting the proteinaceous enamel matrix (Slavkin et al., 1984). The experimental work of Kollar and Fisher (1980) suggests that the mesenchyme directs the oral epithelium to proliferate in a manner which results in the epithelium taking on the basic outline of each tooth. When molar epithelium is combined with incisor mesenchyme an incisiform tooth is produced. Conversely, when incisor epithelium is combined with molar mesenchyme a molariform tooth is produced (Kollar and Fisher, 1980; Slavkin, 1988).

This process of secreting enamel and dentin proteins begins at the occlusal (incisal, cuspal) tips of the tooth crowns. The odontoblasts first retreat toward the future pulp chamber leaving behind a secreted protein matrix. Mineralization of this predentin begins at widely separated foci that expand outward, consolidating to form a uniformly calcified dentin (Avery, 1987; Suga, 1983, 1989). Simultaneously, the ameloblasts retreat toward the future enamel surface, secreting a protein matrix behind them. As the process continues, ameloblasts and odontoblasts retreating from the DEJ and, respectively, leaving behind enamel and dentin matrix, more cervically located ameloblasts, and odontoblasts are enlisted to secrete protein and make more enamel and dentin matrix (Shawashy and Yaeger, 1986).

Sometime after the beginning of enamel matrix formation, calcification of the matrix commences. As the secretory ameloblasts complete their function of forming the enamel matrix, they undergo changes in shape and ultrastructure that are consistent with their change in function from secretion (tall secretory cells) to transport and absorption (short, ruffle-bordered cells) (Reith and Cotty, 1967; Suga, 1983). Aided by the functionally changed ameloblasts, the enamel matrix

loses protein and water and becomes almost completely composed of inorganic calcified crystals.

Although the process of making mature, fully calcified enamel is seen as a two-stage process—matrix formation and maturation—there does not appear to be a standard amount of time separating these stages. Furthermore, calcification is a much more complicated process than previously imagined (Suckling, 1989; Suga, 1983, 1989). In addition to an initial calcification at the time of matrix apposition, maturation of enamel may be best viewed as a further three-step process (Suga, 1983). Suga's experimental research with a wide variety of species shows a first maturation phase involving a heavy increase in mineralization from the surface to the deeper layers, a secondary phase of increase from the DEJ to the surface, and a third phase of increase which is restricted to the more highly mineralized surfaces (Suga, 1983). One consequence of the complex enamel calcification pattern is that it is difficult to ascertain the age of an individual at the time of development of a hypocalcification defect. We do not know the length of time that maturation phases follow matrix formation, and hypocalcification defects may relate to disruptions to ameloblasts at all phases (Suckling, 1989).

### *Enamel structure*

The most essential feature of enamel is its *enamel prisms* (also referred to as enamel rods) (Fig. 1). There are approximately 12 million enamel prisms in a typical human tooth (Osborn and TenCate, 1983), formed from an equal number of ameloblasts. Because approximately 70% of permanent teeth and all deciduous human teeth have a thin layer of aprismatic, structureless enamel at the enamel surface, most prisms terminate before reaching the tooth surface (Osborn and TenCate, 1983). In cross section, human enamel prisms are characteristically keyhole in shape (Boyde et al., 1988; Shawashy and Yaeger, 1986, Shellis, 1984; Simmelink, 1987), average about 4 μm in diameter at the DEJ, and increase in width to approximately 8 μm near the surface (Osborn and TenCate, 1983). The change in dimension is necessary because the surface area of the more outwardly located enamel is considerably greater than the surface area near or at the DEJ.

The enamel prisms are formed by the subsequent calcification of the enamel matrix. While there are equal numbers of ameloblasts and prisms, one ameloblast does not make one prism. Rather, it appears that four ameloblasts jointly contribute to the formation of one enamel prism and each ameloblast is involved in the formation of four separate prisms (Shawashy and Yaeger, 1986). One ameloblast might form the "tail" of the keyhole-shaped prism, while the remaining three ameloblasts might combine in forming the "body." The secretion rate and movement of the ameloblasts orients the hydroxyapatite crystals within the matrix. The orientation of the long axis of the crystals produces the microscopic characteristics of the prisms. Disturbances in enamel secretion and ameloblast movement produce microscopic abnormalities in the shape and configuration of the prisms (Rose, 1973).

A characteristic series of *cross striations,* separated by about 2 to 6 μms, is frequently observed in light microscopic observation of long sections of enamel prisms. With a scanning electron microscope (SEM) the cross striations are observed as constrictions in the prism widths. It has been assumed that these cross striations represent daily (24 hour) growth cycles (Beynon and Wood, 1987; Bromage and Dean, 1985). If this assumption is valid, then counting cross striations may provide an accurate though technically difficult means for determining the length of time over which a prism was formed. This information will then have implications for understanding the duration and sequencing of events in tooth crown development.

Active secretory ameloblasts differ in the length of time they have been operative. The cuspally located ameloblasts commence formation earlier than the more cervically located ameloblasts. Therefore, the "front" of new enamel matrix will be tangentially curved in relationship to the DEJ. The front of advancing, secretory

ameloblasts is furthest away from the DEJ at its occlusal end and still in contact with the DEJ at its cervical end. The exact shape of this developing front is maintained in the pattern of *striae of Retzius,* the second main enamel structure, seen as brown or dark lines with the light microscope (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 traverse the enamel from the DEJ to near the outer surface (Fig. 1). The *perikymata,* a step-like feature, observed on the outer surface of unworn teeth, may be considered to be the surface manifestation of the striae of Retzius (Osborn and TenCate, 1983).

## CLASSIFICATION OF ENAMEL DEVELOPMENTAL DEFECTS

Dental enamel hypoplasia is a class of developmental defects of enamel (DDE). As a general term, enamel hypoplasia refers to a deficiency in the amount or thickness of enamel (Suckling, 1989 after Weinmann et al., 1945). These defects may range in appearance from single and multiple pits or small furrows, to deep and wide troughs of decreased enamel thickness, and ultimately to entirely missing enamel (see Figs. 2, 7 for photographic examples). Enamel hypoplasia is a quantitative defect, as opposed to enamel opacity (or hypocalcification), the other major class of enamel developmental defects, which involves change in color and opacity of enamel, indicating differences in hardness or quality of enamel (FDI, 1982).

All enamel developmental defects result from disruptions in the process of amelogenesis. Enamel hypoplasia has long been held to be due to a disturbance to ameloblasts during matrix secretion, an inference that is strongly supported by the most recent experimental research (Suckling, 1989). Similarly, enamel hypocalcification had long been held to be due to disruption in enamel maturation. However, the assumption that an opacity is always due to a disruption to ameloblasts during maturation phase has recently been challenged by the experimental research of Suckling, who finds that hypocalcification can result from a disruption in matrix formation (Suckling, 1986, 1989).

Based on the pattern of defects within and among teeth, hypoplasias can be reliably diagnosed as resulting from one of three causal conditions: 1) hereditary anomalies, 2) localized traumas, or 3) systemic metabolic stress (Shawashy and Yaeger, 1986; Suckling, 1989; Weinmann et al., 1945). Defects resulting from hereditary causes generally affect all of the teeth in a set and are the most severe (Stewart and Poole, 1982; Weinmann et al., 1945). Defects due to local trauma, local inflammation, and other non-systemic factors may also be relatively severe, but will influence only one tooth or a few adjacent teeth (Andreasen et al., 1971; Ravn, 1975, 1976; Shafer et al., 1983; Skinner and Hung, 1989; Stewart et al., 1982; Weinmann et al., 1945). Finally, defects resulting from systemic metabolic stress are likely to be found on most or all 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; Weinmann et al., 1945).

Individuals with hereditary defects are rare (less than 1%) in most contemporary populations (Winter and Brook, 1975; Witkop, 1957). 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 these individuals may have had little chance of survival. Currently, we are aware of only a single reported case of hereditary enamel hypoplasia from a prehistoric skeleton (Cook, 1980).

Defects due to local trauma are also likely to be rare in prehistoric populations. Goodman and co-workers (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. A severe enamel hypoplasia was noted on an upper central inci-

sor. However, this defect was not matched by a parallel defect on the other upper central incisor, or by defects developing at the same time as this one on any of the other teeth which were forming at the time.

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., 1980, 1984a; Rose et al., 1985). Thus, they are frequently referred to as chronologic or linear enamel hypoplasias, reflecting the linear and chronologic nature of the defects caused by systemic stress at a specific time during tooth development (Sarnat and Schour, 1941; Goodman et al., 1984a).

### *The FDI classification of surface enamel developmental defects (DDEs)*

While there has been general agreement on the distinction between an enamel hypoplastic defect and an opacity, there has been a great deal of disagreement on methods for classifying differences within classes of defects (Clarkson, 1989). For this reason the Federation Dentaire International (FDI) recently commissioned a working group to ". . . propose a system of classification of developmental defects of enamel suitable as an international epidemiological index." (FDI, 1982). The subsequent descriptive classification divides enamel opacities by color (white/cream and yellow/brown; FDI types 1 and 2, respectively). While differences in border characteristics—diffuse versus demarcated opacities—are noted and displayed in the FDI photographs, there are no means for systematically recording these characteristics. Because differences between diffuse and demarcated opacities may have epidemiological significance, they merit greater attention (Clarkson, 1989; Suckling, 1989).

Hypoplastic defects are classified as pits (type 3), horizontal grooves (type 4), vertical grooves (type 5), or altogether missing enamel (type 6). The FDI type 4 defect is most like what is typically referred to as chronologic or linear enamel hypoplasia (LEH) in the anthropological literature.

One shortcoming of the FDI hypoplasia classification is that it does not clearly address the importance of defect linearity. A vertical defect is discussed (type 5), but an illustrative photo is not included and the etiology of this type of irregularity is undetermined. The method for evaluating pits (type 3) is also quite limiting. It may be important to distinguish a linear arrangement of pits, which is most common in our observations of archaeological teeth, versus a random distribution, as is shown in the FDI photo. It may also be important to distinguish a single pit from multiple pits (see Skinner and Hung, 1986, 1989).

A more general problem encountered by use of the FDI is that it does not systematically provide a means for assessing the severity (size, depth, width) of a defect. Defect type 6 (missing enamel), for example, is clearly a more severe manifestation of defect type 4 (linear hypoplasia). However, without information on the size of a defect, it is impossible to test questions concerning the epidemiological significance of defect dimensions. Furthermore, a minimum criterion for scoring a defect is needed to improve reliability and comparability.

The above problems aside, the FDI classification has proved reliable in a number of field studies (Goodman et al., 1987; King, 1989; Murray et al., 1984; Suckling and Pearce, 1984) and should be referred to in studies of skeletal and archaeological teeth. The FDI has provided an important starting point for developing a reliable and comparable epidemiological tool (Clarkson, 1989).

### Recommended terms

In the following discussion of *surface defects* we will use the generic terms of DDE or enamel defect to refer to any form of surface defect. Enamel hypoplasia (EH) will be used to refer to any deficiency in enamel thickness—a pit, line, band, or other form of missing or deficient enamel. A pit is comparable to the FDI type 3 and may be linearly arranged or non-linear, singular or multiple. LEH is comparable to FDI type 4 and is in our experience the most common hypoplastic defect. LEH is distinguished by a marked horizontal or nearly horizontal area of de-

creased enamel thickness (Goodman et al., 1980). Finally, an enamel opacity of hypocalcification (FDI type 1 and 2) refers to a change in color and opacity of enamel without a change in thickness (quantity).

### *The relationship between surface and histological structures*

Interpretation of the meaning of DDEs requires an understanding of the mechanism by which they are formed. Partial understanding has been achieved through experimental work with nonhuman mammals and histological examination of human teeth.

The most severe form of enamel hypoplasia is a total deficiency of enamel, enamel aplasia (FDI type 6). This deficiency may be circular in area or may be a groove of variable width with no enamel covering the dentin. This type of hypoplasia results from the death of the ameloblasts prior to or at the time of differentiation. The fact that normal enamel can develop cervically to the defect indicates that the insult has not irreparably damaged the developing inner enamel epithelium.

A more common, but still severe, form of enamel hypoplasia is a marked deficiency of enamel thickness observed as a circumferential groove, line, or series of pits (FDI type 3 or 4). Histologically, these typical hypoplasias display marked deficiency in the normal external enamel surface profile, convergence of the striae of Retzius as they approach the surface of the enamel, and an absence of normal prism structure along the surface of the defect (Rose 1977). The fact that the enamel within the hypoplastic defect is well mineralized, as evidenced by the normal appearance of enamel prisms in cut and ground calcified sections, indicates that the ameloblasts did not die, but recovered sufficiently to complete the maturation (calcification) phase of their life cycle.

This interpretation of human hypoplasia formation is also observed in experimental work with sheep (Suckling and Thurley, 1984; Suckling and Purdell-Lewis 1982). The convergence of the striae of Retzius suggests that matrix secretion by the ameloblasts slows considerably after the insult and that the ameloblasts do not die, but cease secretion after a period of reduced secretion. The development of a normal enamel thickness by ameloblasts located cervically to the macroscopic defect indicates that the ameloblasts which had just begun to secrete enamel matrix were not irreparably harmed by the insult. Suckling and Thurley (1984) suggest that irreversible harm to the ameloblasts most frequently occurs relatively late in their secretory phase; the longer they have been secreting enamel matrix the more vulnerable the ameloblast. This phenomenon of differential vulnerability may explain why even the more severe hypoplasias typically involve one-half or less of the potential enamel thickness.

A commonly observed histological correlate of enamel hypoplasia is the presence of an *accentuated stria of Retzius* (or Wilson band) leading off the defect. This stria can be observed to commence at the inital slope (cuspal or incisal) of the hypoplasia and to continue to its normal termination at the DEJ. This accentuated stria marks the position of the active ameloblasts (i.e., matrix secretion) at the time of the insult. Suckling and Purdell-Lewis (1982) describe this structure as a "marked incremental line" associated with experimentally induced hypoplasias in sheep. Suckling and Thurley (1984) report that incremental lines are always associated with hypoplasias in physically traumatized teeth of sheep. In our experience, a Wilson band is frequently, but not always, associated with enamel hypoplasia in humans.

In a study of 30 canine and premolar pairs, Condon (1981) noted that all hypoplasias were associated with a Wilson band, defined as a stria of Retzius exhibiting abnormal enamel prism morphology and an abrupt change in direction (Rose, 1973; Wilson and Shroff, 1970). Approximately one-half of the Wilson bands occurred at the beginning of the hypoplasia, and the remainder occurred randomly within the hypoplastic defect.

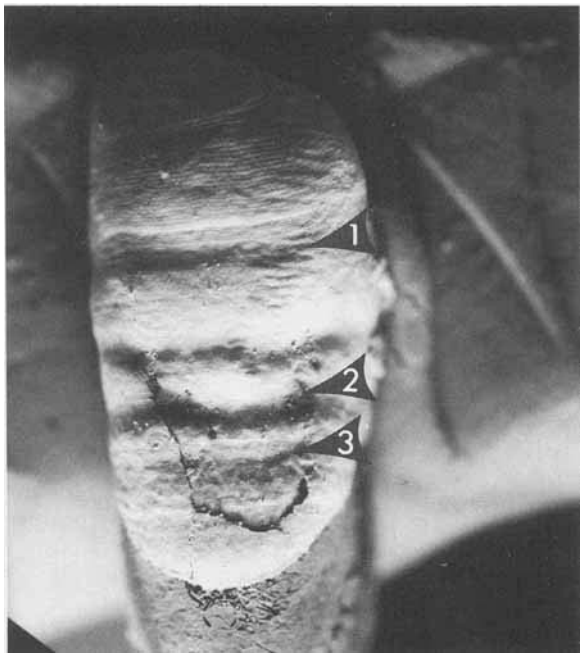


Fig. 2. Left mandibular canine with three of its hypoplasias labeled and numbered.

Following Condon (1981), we have evaluated the association of enamel surface and histological structure in a series of 32 teeth. Thirty linear surface depressions, or "hypoplastic-like" phenomena, were counted. Of the 30 surface features, 19 (63%) began with a Wilson band, five (17%) more contained but did not commence with a Wilson band, and the remaining six (20%) were not associated with a Wilson band.

One of the greatest difficulties in the study of surface defects involves distinguishing true, pathologic developmental defects from normal surface irregularities. Although pictures of "classic" hypoplasias are frequently published, no series of photographs of hypoplasias of differing dimensions has ever been published. To partly address this deficiency, the hypoplasias on the 32 incisor and canine sample were ranked from largest to smallest, and a representative sample is presented, along with associated micrographs and descriptions of their underlying histology. Enamel hypoplasias and the histological sections of the crowns with these hypoplasias, were first examined and photographed at 20× magnification. Enamel prism structure, striae of Retzius, and Wilson bands were then examined and photographed at 80× magnification. Only selected surface irregularities are discussed on any one tooth.

A mandibular left canine is the first tooth to be examined (Fig. 2). Hypoplasia number 1, the most incisally located of the three that are marked with arrows, is a relatively prominent hypoplasia. At 20× magnification (Fig. 3) it appears as a rather broad depression in the enamel surface which begins as a gradual slope from what would have been the normal enamel surface. Even at this low magnification a Wilson band can be seen leading off the hypoplasia (see arrow). The striae of Retzius converge near the enamel surface. A second Wilson band is associated with a further dip in the enamel surface approximately midway across the hypoplasia. Due to the clear association of the Wilson band and the band convergence, there is no question that this hypoplasia resulted from a developmental disturbance.

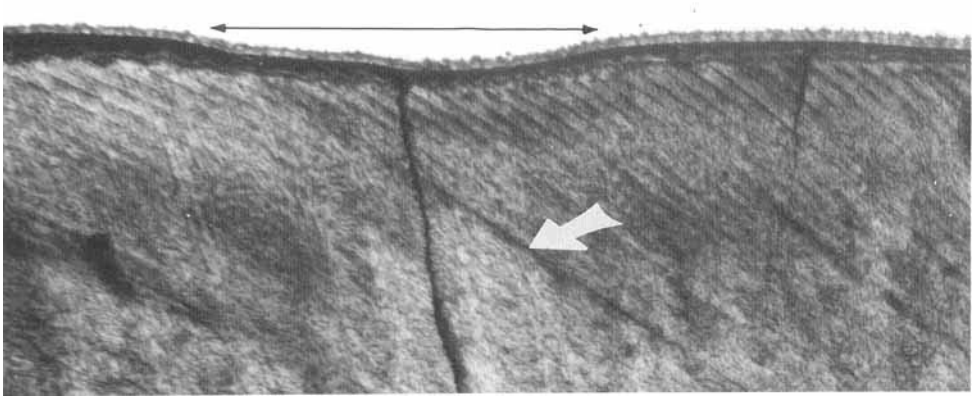

Fig. 3. Hypoplasia number 1 (from Fig. 2) underneath the double arrow and a Wilson band leading the formation of the hypoplasia labeled with an arrow ( ×20). A second Wilson band located at the base of the arrow follows and is associated with an undulation in the floor of the hypoplasia.

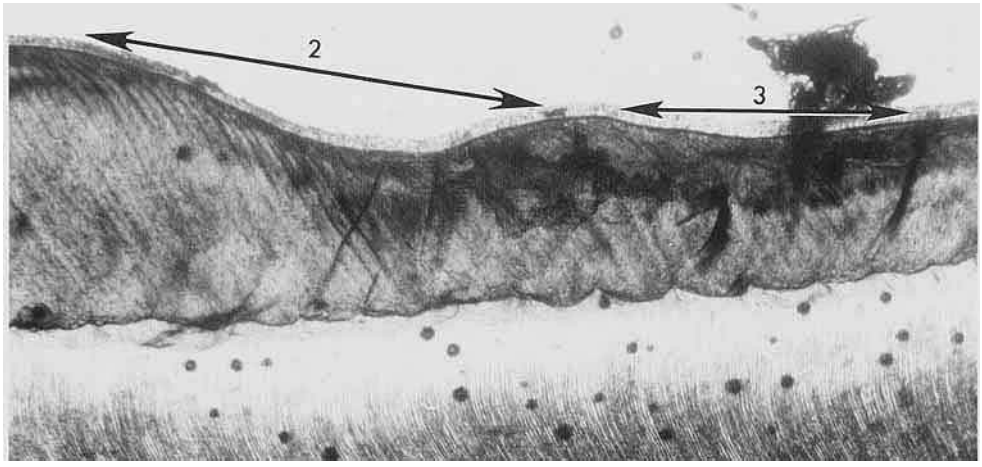


Fig. 4. Hypoplasia number 2 (from Fig. 2) under the lefthand double-headed arrow and hypoplasia number 3 under the righthand double arrow. Dirt adhering to the tooth surface partly obscures hypoplasia number 3. ×20.

Hypoplasia number 2 is the deepest and most easily observed on all the teeth examined in this study (Fig. 2). At 20× magnification, this hypoplasia appears as a significant deficiency in the enamel (Fig. 4). The cuspal slope is more gradual than the cervical and numerous closely spaced striae can be seen in the deeper enamel. At 80× magnification a Wilson band can be seen about halfway down the cuspal slope of the hypoplasia (Fig. 5). Convergence of the striae is evident and there is a loss of normal prism structure within the trough of the hypoplasia. Numerous prominent striae are found below the hypoplastic surface. However, they do not extend into the enamel adjacent to the DEJ, and are not considered to be Wilson bands, but rather, these are classified as "cluster bands." There is no question that this depression in the enamel surface is the result of a perturbation

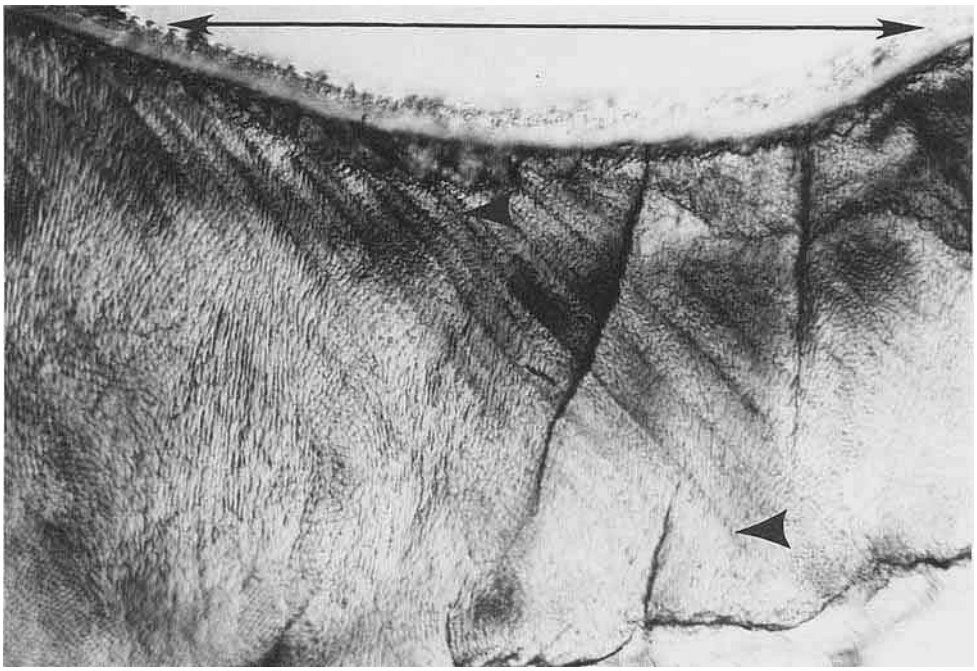

Fig. 5. Hypoplasia number 2 (from Fig. 2) located under the double-headed arrow and a Wilson band indicated by two arrowheads. ×80.

in amelogenesis. The series of cluster bands suggests that this hypoplasia was the result of a sequence of events during which ameloblasts repeatedly reached a threshold at which their function was temporarily disrupted. The general microscopic morphology of hypoplasias 1 and 2 are typical of macroscopically identified hypoplasias, observed regularly in the middle thirds of canine and incisor crowns.

Hypoplasia number 3 on this same tooth is macroscopically less prominent than the others (Fig. 2). At 20× magnification it is a broad, but shallow, dip in the enamel surface (Fig. 4). At 80× magnification, this hypoplasia appears as a shallow, almost unobservable, depression in the enamel surface with the striae converging sharply at the enamel surface (Fig. 6). This hypoplasia begins with a Wilson band and contains six other clearly defined Wilson bands. There are other prominent striae which do not meet the full definition of Wilson bands and should be classified as cluster bands. This hypoplasia is clearly associated with disturbed amelogenesis and, as previously noted, appears to be the result of an episodic or threshold phenomenon. A shallow hypoplasia, such as this one, associated with a high degree of histological abnormality, is typical of defects in the thin cervical enamel of incisors and canines.

Figure 7 is a right maxillary central incisor. The hypoplasias observed on central incisors appear more subtle than those on the corresponding canines. Hypoplasia number 1 involves a double depression in the enamel (Fig. 7). At low magnification (20×) this hypoplasia can be viewed as a broad, shallow depression in the enamel surface and numerous prominent striae can be seen within it (Fig. 8). At 80× magnification, a Wilson band can be seen just after the beginning of the hypoplasia (at the base of the right arrow) and is closely followed by a second Wilson band (left arrow; Fig. 9). All striae converge near the enamel surface.

Hypoplasia number 2 is macroscopically very similar to number 1 (Fig. 7). At 20× magnification it appears as a shallow depression in the enamel associated with numerous prominent striae (Fig. 8). At 80× magnification this hypoplasia

Fig. 6. Hypoplasia number 3 (from Figs. 2, 4) goes from edge to edge. The Wilson band leading the hypoplasia is indicated by the lefthand arrow and is followed by six additional Wilson bands (arrows) under the hypoplasia. ×80.

appears to begin with a Wilson band, with a second Wilson band closely following, and convergence of other striae at the enamel surface (Fig. 10). These shallow hypoplasias are typical of those found in the cervical enamel.

This next tooth, a maxillary right lateral incisor, exhibits one possible hypoplastic line and a series of slight depressions between the line and the cemento-enamel junction (CEJ) (Fig. 11). The surface abnormality is typical of those that are of greatest concern regarding whether they should be recorded as true hypoplasia. At 80× magnification the sharp depression in the enamel is clearly seen, but there is no evidence of disruptions in the underlying enamel (Fig. 12). The absence of Wilson bands and/or prominent striae, in conjunction with normal enamel prism morphology, suggests that this line is not a hypoplasia (e.g., due to ameloblastic disruption). We suggest that this is a prominent perikymata. All of the other lines observed on the tooth below line 1 are clearly perikymata associated with prominent, but normal striae of Retzius.

These descriptions of hypoplasias and their underlying histological structure are presented as representative examples of the range of hypoplastic expressions that one normally encounters. Our main counsel, based on the correlation of surface and histological structures, is that surface hypoplastic lesions must display a trough-like form. Sharp surface depressions with little width are virtually never associated with histological abnormalities. We recommended that hypoplasias be conservatively identified by demonstrating a deficiency in enamel thickness and having a breadth that is clearly noted without magnification. In epidemiological terms it may be better to have a few false-positives, and risk missing some minor hypoplasias, than to have to define hypoplasias as all surface irregularities and suffer the consequences of both poor validity and reliability.

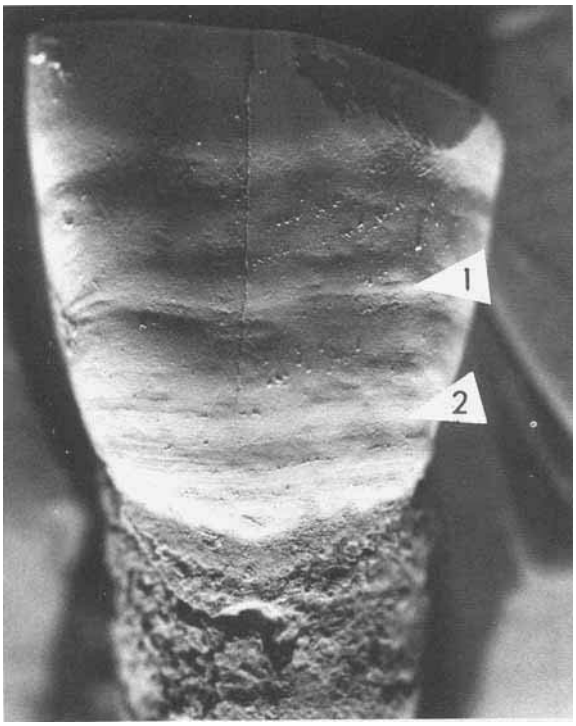


Fig. 7. Right maxillary central incisor with two of its hypoplasias indicated by numbered arrows.

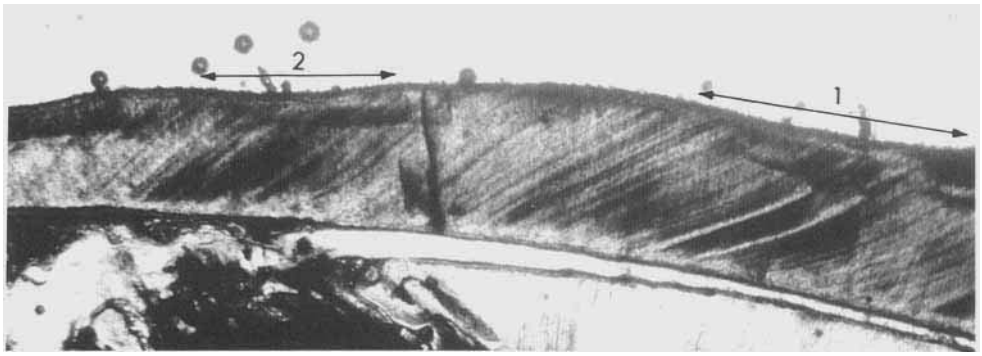


Fig. 8. Hypoplasia number 1 (from Fig. 7) is under the righthand double-headed arrow and number 2 is under the lefthand double-headed arrow. ×20.

## A MODEL OF ENAMEL DEFECT FORMATION

In order to better conceptualize the study of the wide variety and degree of enamel defects and associated histological changes, we have constructed two general epidemiological models (Figs. 13, 14). The first model (Fig. 13) highlights a chain of events leading to the formation of a DDE. Environmentally and culturally induced stressors and constraints encountered by the individual combine with the individual's level of host resistance to determine the type, duration, and intensity of systemic physiological perturbation (stress). If ameloblasts are active at the time

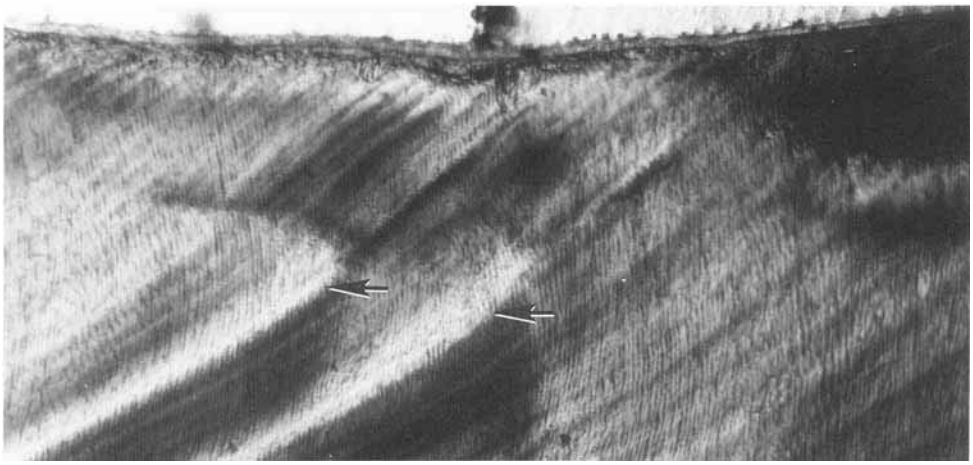

Fig. 9. Hypoplasia number 1 (from Figs. 7, 8) showing the first Wilson band (righthand arrow) starting close to the initiation of the hypoplasia and the second Wilson band (lefthand arrow) later in the hypoplastic sequence. ×80.

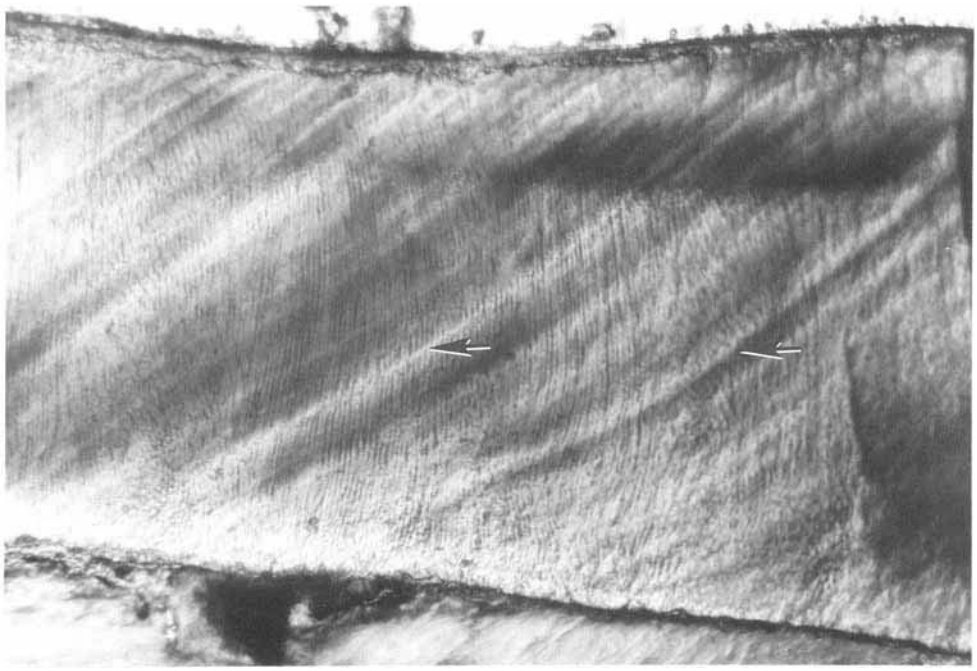

Fig. 10. Hypoplasia number 2 (from Figs. 7, 8) showing the deficiency in the enamel, the initial Wilson band indicated by the righthand arrow, and the succeeding Wilson band by the lefthand arrow. ×80.

of stress, then their physiology and metabolism will be affected. As has been previously noted, a level of physiological stress may be reached at which the ameloblast can no longer function. This "threshold" may be passed in an episodic manner or chronically.

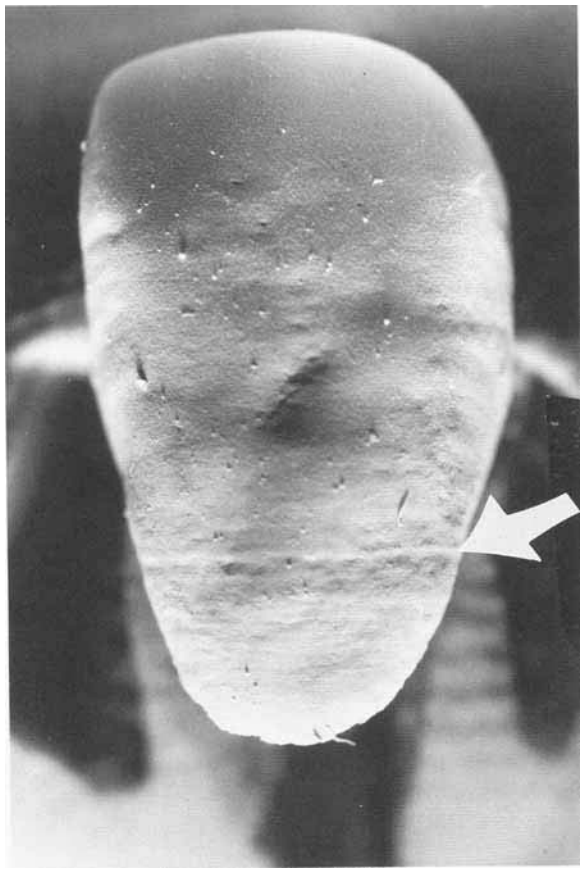

Fig. 11. Right maxillary lateral incisor with false hypoplasia (depression) indicated by an arrow.

Changes in ameloblast physiology, if long-lasting and severe enough, may induce a permanent developmental change in enamel structure, a DDE, and underlying histological changes. The type of defect found is determined by the functional stage of the active ameloblast at the time of disruption. Similarly, the size of a defect may relate to the severity and intensity of the disruption (Suckling, 1989). Finally, information on the epidemiological pattern of defects provides information on damage to concurrently developing systems and the patterns of environmental constraints and stressors encountered by the developing organism. The fundamental research questions in the study of DDEs concern understanding issues such as the specificity and sensitivity of enamel to different etiological factors (links from the first to next to last box). However, the interest in DDEs stems from their potential as epidemiological indicators of past periods of physiological perturbation (next to the last box).

While Figure 13 focuses broadly on potential differences in expression of enamel disruptions in relation to etiological factors, Figure 14 highlights a general method for reflecting upon the etiology of any enamel developmental defect. Figure 14 presents a *threshold model* for the formation of a defect, in this case with a hypothetical tooth, similar in developmental timing to a permanent incisor. The magnitude of ameloblastic disruption is presented on a y-axis and developmental age is presented on the x-axis. Enamel developmental defects result when ameloblastic

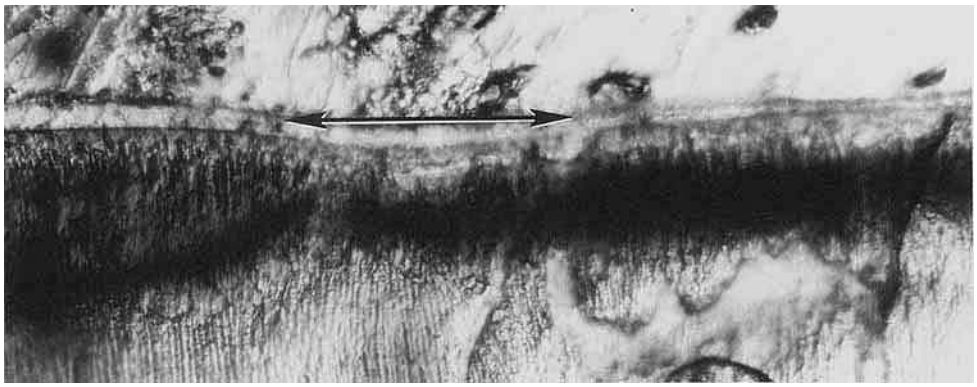

Fig. 12. The false hypoplasia indicated in Figure 11 is located under the double-headed arrow. Although suffering from severe postmortem damage, there is no abnormal prism structure underlying the depression. ×80.

disruption is pushed over a threshold line (see arrow) due to a combination of forces.

From the bottom, the first factors affecting ameloblastic disruption are unknown etiological/susceptibility factors. These may include genetic factors (potentially quantifiable) that render an individual particularly susceptible to stress, or may specifically weaken ameloblasts. The potential contribution of these factors has yet to be determined. Hypothetically, they have been placed at a point where they contribute to about 30% of the threshold, and their magnitude is constant by age.

The next set of factors that can raise ameloblastic disruption toward threshold levels is nutrition related. Depleted nutrient stores and inefficient utilization and availability of key nutrients is likely to have a significant effect on ameloblastic disruption and on whether short-term stressors can be met without pushing ameloblastic disruption over the threshold line. Nutritional factors are hypothesized to contribute an additional 30% to ameloblastic disruption. This contribution is likely to be somewhat greater, however, after the 1st year and during the time of weaning and immediately post weaning.

Finally, illness history in relationship to ameloblastic disruption is portrayed as a wavy line. The amplitude of each crest is an indication of the severity of the illness. Additionally, the base of the line, how far up it is on the y-axis, is related to the prior background genetic, nutritional, and other resistance factors. An enamel defect results when the illness is severe enough that ameloblasts are disrupted to a degree greater than the threshold line. Finally, note that the threshold line is curved. Because ameloblasts are most sensitive to disruption in the middle thirds of teeth, the threshold line is lowest, and easiest to surpass, toward the middle portion of the tooth, (Goodman and Armelagos, 1985a,b).

While presenting a conceptual basis for studying enamel defects, the above should also provide a sense of the variety of etiological questions and unknowns that can be considered. Clearly, enamel defects are most often due to combinations of factors and physiological processes. While the basic components, such as the role of undernutrition and infection, may remain relatively constant, and the end result of these processes is simple (whether, for example, ameloblasts stop or continue to secrete matrix), the significance and essentialness of different factors is likely to change and vary over time and environment.

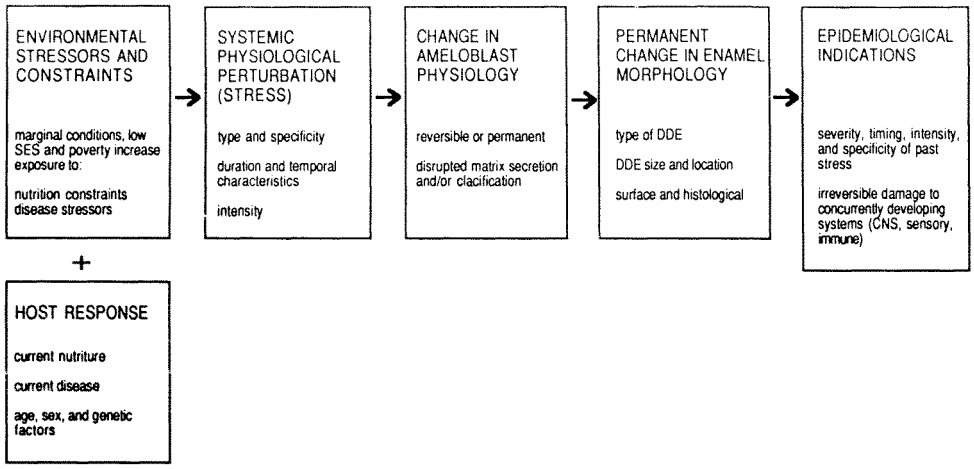


Fig. 13. Epidemiological model of the cause of enamel developmental defects.

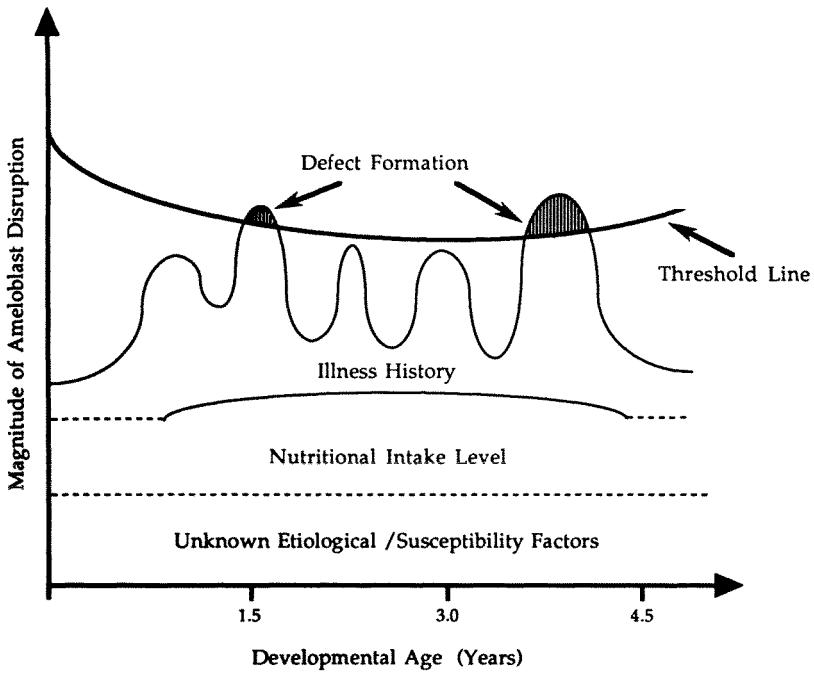


Fig. 14. Threshold model for the formation of enamel developmental defects.

Before turning to an examination of other extant, fundamental issues in the study of DDEs, the following section provides an historical overview of developments and research issues. Special emphasis is given to anthropological literature and research themes that *apply* DDEs as epidemiological indicators.

## HISTORY OF STUDY

### *Pioneering studies: research prior to 1930*

Early studies of enamel developmental defects focused on two related issues. The first concerned the relationship between enamel quality and subsequent suscep-

tibility to dental caries. The second issue, that enamel quality reflects underlying physiological disturbances due to poor periods of nutrition and disease, was originally a subsidiary concern, but began to emerge as an important research question in the 1930s.

Sarnat and Schour (1941) trace the history of enamel hypoplasia studies to the 1700s when Bunon attributed tooth "erosion" to rickets, measles, and scurvy. By 1785, Sanchez had linked dental alterations with syphilis (Sarnat and Schour, 1941), a finding that was later confirmed by Hutchinson (1857). Other early studies linked changes in enamel structure, such as pits and lines, to nutritional deficiencies and febrile disease (Sarnat and Schour, 1941).

The histological study of ancient and prehistoric teeth was undertaken to determine why modern peoples have a high prevalence of dental caries, while most ancient peoples did not. One hypothesis was that ancient peoples had dental enamel superior in quality to that found in modern groups. Concern with this problem led Tomes (1892) to describe the histology and postmortem changes of a tooth which he had found in a cemetery. In 1895, Berten provided evidence that disturbances in metabolism are reflected in the microstructure of developing teeth (Sheldon et al., 1945). Black (1906) then compared histological and surface changes due to a variety of local and systemic insults. Subsequently, Bodecker (1930) used ground histological sections of prehistoric Puebloan teeth to demonstrate that despite the presence of enamel fissures (i.e., very deep occlusal grooves), the prehistoric teeth were remarkably free of decay. In contrast, histological observations of both human clinical samples and laboratory animals were demonstrating a relationship between the progress of caries and defective enamel and dentin (e.g., Barker, 1931). While neither proving nor disproving the general relationship between enamel quality and caries susceptibility, this research established that archeological teeth could be sectioned and that histological observations could make contributions to our understanding of oral health.

The literature of the period shows a profound lack of consensus in the definition of an enamel defect. It certainly did not employ the current terminological conventions regarding enamel hypoplasia and enamel hypocalcification. A variety of terms were used in place of and along with enamel hypoplasias, including dental erosion, premature caries, notched incisors, enamel aplasia, and atrophied teeth (Marshall, 1936; Sarnat and Schour, 1941). As well, dental researchers were aware of the relationship between the position of a defect on teeth and the time of the causal disturbance by the middle of 19th century (Black, 1906; Hutchinson, 1857). However, they did not begin to examine systemically the relationship between defect location and the age of the individual at the time of the disturbance until the 1930s.

The single important contribution of these early studies was the demonstration that both enamel surface defects and microstructural defects of the enamel and dentin were associated with events that occurred during that period in the individual's life when the teeth were developing. A link between events during formation of teeth and caries susceptibility was expected to be found with new experimental methods (Mellanby, 1929) and clinical studies (Day, 1944). Somewhat paradoxically, the development of new experimental methods for relating events during enamel formation to subsequent abnormalities in formation did little to further agreement on the role of enamel quality in caries etiology. However, this new research greatly stimulated the acquisition of knowledge on the cause of enamel hypoplasias and microdefects.

### *Foundation studies: research from 1930 to 1960*

The middle portion of the twentieth century, particularly the period between 1936 and 1945, was a time of great activity and advancement in the study of enamel defects (see Suckling, 1989). From 1932 to 1945 Schour and colleagues at the University of Illinois authored a series of articles on the time of tooth crown development and the histology of enamel (Massler et al., 1941; Sarnat and Schour,

1941; Schour 1936; Schour and Massler, 1945; Schour et al., 1937; Schour and Smith, 1935; Schour and Van Dyke, 1932). They established the notion that enamel surface defects could provide a "kymographic record" of periods of physiological stress (Sarnat and Schour, 1941; Massler et al., 1941). Meanwhile, Kreshover and co-workers were mainly responsible for experimentally determining the range of insults which could sufficiently disrupt enamel formation and cause histological and surface defects in experimental animals (Kreshover, 1940, 1942, 1944, 1960; Kreshover and Clough, 1953; Kreshover and Hancock, 1956; Kreshover et al., 1953, 1954, 1958).

During this period the study of ancient teeth continued to be motivated by the idea that their superior enamel structure could prevent dental decay. Sognnaes (1955) used thin sections of 233 archeological teeth to describe the postmortem changes in the enamel and dentin. He determined that there were extensive changes in the histological integrity of the dentin with burial and fossilization. However, the enamel survived virtually unaltered in even the oldest teeth. This research helped to establish that prehistoric enamel was rarely superior in structure to modern enamel, despite vast differences in the frequencies of caries (Sognnaes, 1956). More important, Sognnaes concluded that defective enamel histology is the result of nutritional and other metabolic disturbances. Others also reached similar conclusions: Schuman and Sognnaes (1956) demonstrated that defective histological structure was common in free-ranging nonhuman primates and Moorrees (1957) attributed an increase in histological defects in Aleut teeth to a decline in the quality of diet associated with western contact and acculturation. The low prevalence of caries in native Australians was attributed to infrequent use of refined carbohydrates, rather than "intrinsic qualities of the tooth structure" (Cran, 1960:104).

These studies essentially refuted the notion that prehistoric and nonindustrialized individuals had a superior enamel structure which contributed to caries-resistant teeth (Clement, 1963). Conversely, the potential was established for using the microstructure of human enamel to assess the adaptive success (e.g., level of stress, morbidity, nutritional inadequacy) of prehistoric peoples. It should also be noted that none of these studies referenced the extant clinical and experimental literature. Anthropological and clinical/experimental research followed separate paths, despite the fact that both areas of study were being conducted by dental researchers.

In one of the first epidemiological studies in a less-developed country, Anderson and Stevenson (1930) found that up to 90% of their subjects from a Chinese village had a hypoplastic-type enamel defect. This study established the high prevalence of enamel defects in an agricultural society. Anderson and Stevenson (1930) also noted an increase in prevalence of defects in younger individuals and proposed a secular trend toward increased stress.

Day (1944), in a study of caries in northern India, found defects in up to 65% of the boys he examined, but was unable to link these defects to caries. Conversely, Davies, in a study of 770 public elementary school children, found that hypoplastic teeth were highly likely also to be carious (1939).

A notable sequence of studies in this period involved the clinical and histological study of "tooth rings." These studies were initiated by Schour and co-workers, and continued unabated from the 1930s into the 1960s (Rushton, 1933, 1939; Sarnat and Schour, 1941; Schour, 1936; Schour and Kronfeld, 1938; Swanson, 1931; Watson et al., 1964; Johnson et al., 1965). One of the first studies of a microdefect (not observable on the enamel surface) that is attributed to a specific stress (i.e., birth) is Schour's (1936) description of neonatal lines (previously identified by Rushton, 1933) observed in the enamel and dentin of human deciduous teeth (Schour and Kronfeld, 1938). Other histological investigations concerned the variable quality of enamel formed before and after birth (e.g., Rushton, 1939).

The pivotal work of Massler et al. (1941) provided a mechanism for establishing the age at formation of enamel defects and initiated the search for chronological

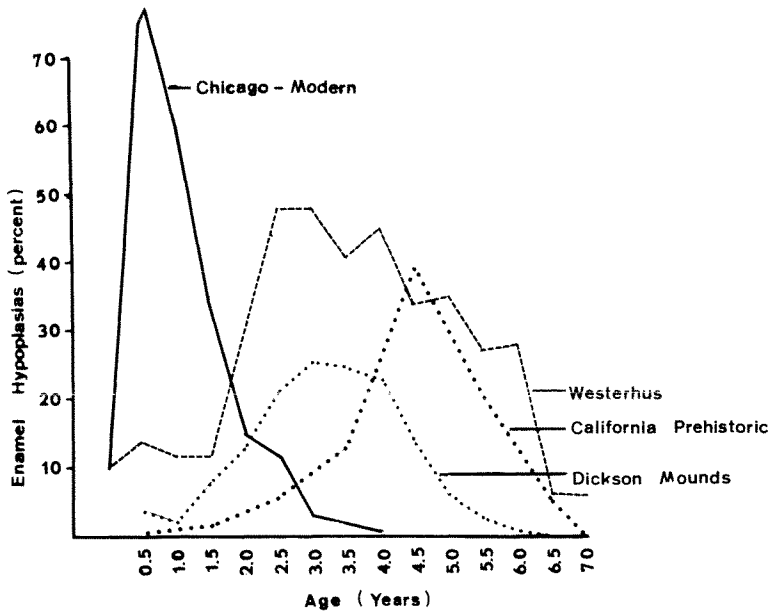


Fig. 15. Comparison of chronologies of linear enamel hypoplasia in different populations: Chicago historical (Sarnat and Schour, 1941), Dickson Mounds, Prehistoric Illinois (Goodman et al., 1984a), California Prehistoric (Shulz and McHenry, 1975), and Westerhus, Medieval Sweden (Swärdstedt, 1966).

correlations between enamel defects and causal agents. For example, Sheldon et al. (1945) used a clinical sample to demonstrate that 70% of enamel microdefects were associated with systemic disturbances evident in the subjects' medical histories and without considering nutritional status or other resistance factors. Conversely, Schour and Massler (1945) reviewed the already impressive literature on the relationship between diet and the developing human dentition.

Perhaps the most influential paper yet published on enamel defects is that of Sarnat and Schour (1941, continued in 1942). Using the sample of 60 individuals from the Chicago area with known hypoplasias, these authors established that many of the defects could be attributed to known bouts of disease. They also presented a cumulative distribution of enamel hypoplasias by individual's ages at time of development (Fig. 15). Nearly two-thirds of the defects developed during the 1st year and most of the remaining developed by the end of the 2nd year.

The clinical and experimental research of this period established a research paradigm which would eventually enable enamel histology to be employed in anthropological research. Kreshover (1940) provided an excellent summary of earlier research and described the histopathology of abnormal enamel development. He described three severity stages of ameloblast disturbance and associated them with observed enamel defects ranging from those only observed in histological section to severe linear enamel hypoplasia. By 1960 results had been presented from an extensive series of histological studies that examined the relationship between a variety of stresses (i.e., tuberculosis, alloxan diabetes, artificial fever, and lymphocytic choriomeningitis), abnormal amelogenesis, and both microdefects and hypoplasias of enamel (Kreshover 1942, 1944; Kreshover et al., 1953, 1958; Kreshover and Clough, 1953; Kreshover and Hancock, 1956). Kreshover (1960) associated enamel defects with a variety of agents, and noted:

> 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).

While Kreshover did not cite research on the non-specificity of the stress response that was published in the decade before his review (Selye, 1950, 1955, 1956), his writing appears to have been influenced by the general stress perspective. Kreshover's assertions on the sensitivity and non-specificity of enamel to developmental disturbance has survived to the present time. Research before 1960 established methods for chronologically relating enamel disturbances to the developmental age of the individual at the time of their development. It also clearly established a relationship between enamel defects and a series of specific illnesses, hormonal imbalances, and nutritional problems (Kreshover, 1960; Via and Churchill, 1959; Vila, 1949).

Although the general methods and the validity of the main assumptions involved in the study of enamel defects had been established by 1960, a number of important subsidiary issues remained. For example, no researcher had systematically studied differential susceptibility of teeth to developmental disruption, although this was a readily observed phenomenon. And, while most studies found a relationship between known stressors and defective enamel, a perfect association was never established. The sometime high frequency of false-positives (hypoplasia, but no apparent stress) and false-negatives (no hypoplasia, but apparent stress) could not be readily explained.

Two "uniformitarian" assumptions that most directly derive from the work of Sarnat and Schour (1941) may have contributed to misguiding researchers of the next generation. The first assumption is that teeth are universally susceptible to enamel defects. Accordingly, all developing teeth will be equally exposed and responsive to stress; therefore, the quality of their enamel will be equivalent. The second assumption is that Sarnat and Schour's (1941) chronological pattern of enamel defects was due to a universal pattern of susceptibility (see Fig. 15).

The first assumption clearly permeated the dental sciences, as evidenced by constant assertion in textbooks. In a textbook on forensic dentistry, Cameron and Sims stated: "Since the disturbance is of a general systemic nature, all of the teeth forming at the time of the disturbance will have an equally marked incremental line formed in the hard tissue undergoing mineralization." (1974:29). Similar assertions are found in texts in preventive dentistry (Nizel, 1981), pedodontics (McDonald and Avery, 1983; Stewart et al., 1982), oral embryology and histology (Shawashy and Yaeger, 1986), and oral diagnosis and pathology (Guita, 1984; Kerr et al., 1983; Pindborg, 1970; Shafer et al., 1983; Spouge, 1973).

The second uniformitarian assumption, that the chronological pattern of enamel defects found by Sarnat and Schour (1941) was due to a universal constitutional change in susceptibility to disruption (also see Massler et al., 1941), was also quickly reified. For example, in a frequently cited review article, Giro stated: "It is a *known fact* that at least 68% of all cases of hypoplasias reported have occurred during the 1st year of life." (emphasis ours) (1947:313). The universality of the chronology of enamel hypoplasias is also frequently cited in current dental texts, despite studies to the contrary (e.g., Pindborg, 1970; Shawashy and Yaeger, 1986; Guita, 1984; Spouge, 1973; Shafer et al., 1983; and McDonald and Avery, 1983).

While the first assumption has likely hindered and frustrated researchers who have tried to understand patterns of disruption, within individual studies and across studies, the second, stable chronology assumption may have altogether prevented researchers from studying the variations in chronologies of stress in different populations.

*Diversification: research from 1961 to 1979*

The period from 1961 to the end of the 1970s is characterized by an increased diversity of studies of enamel histology and developmental defects. Most notable is the exponential increase in the number of studies of archaeological and skeletal populations. As well, a series of key articles focused on the etiological relationship between enamel defects and nutrition and disease stressors in less-developed countries.

## Advances in dental histology

Profound advancements were made in understanding the embryology and histology of enamel during this period. Just prior to its onset, Gustafson (1959) provided one of the first extensive discussions of normal and defective enamel histology, including detailed descriptions of the morphology of enamel prisms, explanations for the formation of striae of Retzius, and a definition of pathological bands (i.e., enamel microdefects or Wilson bands). Although some of her conclusions have since been shown to be faulty (e.g., that the enamel prism cross striations are membranes and prisms are strings of boxes formed by the ameloblasts), this work laid the groundwork for extensive studies of striae of Retzius.

The rapid adoption of the transmission and scanning electron microscopes in dental research provided the opportunity to observe the morphology of enamel prisms at high magnifications and resolution (Ronnholm, 1962; Meckel et al., 1965; Helmcke, 1967). This knowledge and technology was quickly transferred to the study of striae of Retzius and microdefects. Weber and Eisenmann (1971) described neonatal lines with the aid of high magnification transmitted phase contrast light microscopy, microradiography, and transmission electron microscopy (also see Weber et al., 1974). Of particular importance was Osborn's detailed studies of enamel prism morphology and striae of Retzius with the light microscope (1968, 1971). He attributed the morphology and orientation of enamel prisms to the fluid dynamics of enamel matrix secretion and established that striae of Retzius are normal features of enamel (Osborn, 1973).

Wilson and Schroff (1970) described the light microscopic morphology of striae of Retzius and "pathological bands," thereby setting the stage for using enamel microdefects in the study of childhood stress in prehistoric populations. They established that etching sections with hydrochloric acid made it possible to focus only on the section's surface. Wilson and Shroff also developed the technique of using off-centered transmitted light to bring the enamel prisms and striae of Retzius into clear relief along the sectioned enamel surface (Wilson and Shroff 1970; Wilson personal communication to Rose). These authors classified striae into three types: line striae, band striae, and pathological bands. Both the line striae and band striae are characterized by changes in the direction of enamel prisms. In agreement with Osborn (1968, 1971, 1973), Wilson and Shroff (1970) attributed striae to normal, but abrupt, changes in ameloblast direction. *Pathological bands* were defined as those striae which also exhibited abnormal enamel prism morphology, in addition to an abrupt change in direction.

## Studies in physical anthropology

With the development of bioarchaeology and growing interests in the "new archaeology," a number of studies commenced in the 1970s on enamel hypoplastic defects and histological changes in teeth from archaeological populations. In the first systematic histological study of enamel defects in bioarchaeology, Rose (1973) employed the Wilson and Shroff definition of pathological bands to evaluate their utility for studying childhood stress in prehistoric samples. However, because the term "pathological bands" had unproven connotations, Rose introduced the term Wilson band as a neutral alternative. Using the criteria of abnormal enamel prism morphology and a change in prism direction, Rose (1973; Rose et al., 1978) found an increased frequency of Wilson bands in the permanent canines of individuals

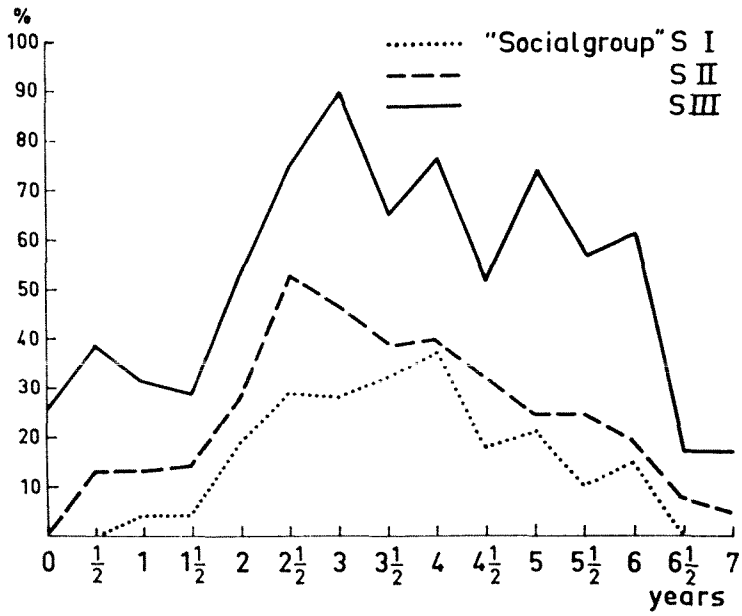


Fig. 16. Comparison of the chronology of linear enamel hypoplasia by social classes from Westerhus, Sweden (from Swärdstedt, 1966).

from the Middle Mississippian period at Dickson Mounds, Illinois, in comparison to an earlier group at this site and a Middle Woodland group from Gibson Mounds. Rose also found an increased frequency of Wilson bands in younger individuals and a peak age at development of defects between about 1.5 and 2.0 years.

In a significant early review of histological defects, especially in primate teeth, Molnar and Ward (1975) summarized the major stresses known to influence dentin and enamel microstructure. They employed light microscopy of unetched dental thin sections and used the degree of pigmentation to identify enamel microdefects. Subsequently, Rose systematically studied all enamel features (Wilson bands, striae of Retzius, and hypoplasias) with the light microscope, described three different types of pathological bands, and discussed their locational relationships within permanent human canine teeth (Rose, 1977). Finally, using a scanning electron microscope, Rose (1979) demonstrated that Wilson bands exhibited a progression of distorted enamel prism morphology. The studies by Molnar and Ward (1975) and Rose and co-workers (1978) employed and integrated, for the first time, the previously cited research on nonindustrial and prehistoric teeth with the clinical and experimental work on abnormal enamel development.

The first systematic study of linear enamel hypoplasias in an archaeological population was undertaken by Swärdstedt (1966), who assessed the frequency and age at development of these surface defects in Westerhus, a medieval population from the province of Jämtland. Swärdstedt discovered a variety of interesting paleoepidemiological patterns. Males displayed a higher prevalence of enamel hypoplasias than females; more hypoplasias were found in later than in earlier historical periods; individuals from the poorer socioeconomic classes displayed more hypoplasia than individuals from the better-off classes (Fig. 16); and the young displayed more hypoplasias than older individuals (Swärdstedt, 1966). Most defects developed between 2 and 4 years of age (Figs. 15, 16), and Swärdstedt utilized the chronologic nature of LEH to try to discern an annual cycle of stress, albeit statistical evidence for such a pattern was not found. The extreme difference in

frequency of hypoplasias by social class fit easily with the hypothesis that less-well-off individuals are exposed to greater stress (Fig. 16). The increased frequency of hypoplasias in the young parallels the previously noted findings of Anderson and Stevenson (1930) and Rose et al. (1978).

Swärdstedt's study (1966) also included a number of methodological innovations. The most significant of these involved assessing the age of the individual at the time of formation of a defect. Using the tooth crown formation charts provided by Massler and co-workers (1941), Swärdstedt demonstrated how the distance of an enamel hypoplasia from the CEJ can be used to estimate the age of an individual at the time of hypoplasia formation (also see Goodman et al., 1980; Rose et al., 1985). Swärdstedt also realized that a defect due to systemic perturbation should be found on all or many teeth developing concurrently. He "matched" defects occurring on different teeth in a given period. Unfortunately, he was less clear in describing the procedures used to match defects.

Other researchers have also found that the risk of enamel hypoplasias peaks at particular ages. Saul's (1972) observation on prehistoric Maya was similar to that reported by Swärdstedt (1966; also see Saul and Hammond, 1974). Schulz and McHenry (1975) found a slightly later peak, around 4 to 5 years of age, in permanent canines from prehistoric California Indians. Hillson (1979) also found that in prehistoric Egyptians and Nubians enamel hypoplasia and histological abnormalities tended to form around 2 to 4 years of age.

All of these studies of archaeological populations found relatively similar chronological distributions of defects, but ones that were markedly at odds with that of Sarnat and Schour (1941) (Fig. 15). Nevertheless, rather than challenging the universality of the Sarnat and Schour chronology, these authors mainly suggested that the differences they demonstrated might be due to sampling and methodological issues alone.

Work on the paleoepidemiology of enamel defects was also undertaken by Sciulli, for both deciduous (1977) and permanent teeth (1978) of prehistoric Ohio Valley Amerindians. In addition to having smaller teeth, he found that agriculturalists had a higher frequency of severe enamel hypoplasia on deciduous teeth. Sciulli (1978) also found that developmental abnormalities were more common on the permanent dentition and attributed this difference to a more favorable developmental environment (greater canalization) of the deciduous teeth.

In a series of studies of individuals from the Lower Illinois River Valley, Cook (1979, 1981, 1984; Cook and Buikstra, 1979) provided a set of epidemiological insights and methodological innovations. Her findings included demonstration of an increased weaning age mortality in individuals with postnatal defects (Cook and Buikstra, 1979). The occurrence of an enamel defect was associated with cribra orbitalia, a bony manifestation of anemia.

El-Najjar and co-workers (1978) analyzed the prevalence of hypoplasias in the Hammon-Todd skeletal series and compared their results to contemporary children from Cleveland. Overall, the frequency of defects was greater in the Hammon-Todd series (comprising mainly lower class individuals who died in the early 1900s in the greater Cleveland area). White males were found to have more defects than white females. While "whites" from Hammon-Todd had more defects than "blacks," the reverse was found for the contemporary children.

These data suggest a number of epidemiological patterns. The change in relative proportion of defects in "whites" and "blacks" speaks against genetic differences in susceptibility. The lower frequency of defects in blacks relative to whites in the Hammon-Todd collection could be due to intense selection against blacks who were stressed during infancy and childhood, or to the extreme poverty of the whites whose skeletons were found in the collection. Finally, the differences in the contemporary sample, drawn from schools of similar socioeconomic background (El-Najjar et al., 1978), question the notion that socioeconomic factors have uniform and singular effects on levels of stress in contemporary North America.

*TABLE 1. Comparison of proportions (%) of enamel hypoplasias by tooth type in different groups (highest proportion for each group is in bold)*

| | I1 | I2 | C | Pm1 | Pm2 | M1 | M2 |
|---|---|---|---|---|---|---|---|
| Maxilla | | | | | | | |
| Hammon-Todd[1] | 65.9 | 61.5 | **68.6** | 46.3 | 33.7 | 26.8 | 16.2 |
| Japan[2] | **86.7** | 50 | 60 | 33.3 | 20.0 | 30 | 16.7 |
| Dickson[3] | **29.8** | 20.0 | 24 | 4.9 | 3.6 | 10.2 | 5.9 |
| Neanderthals[4] | 44.4 | 50.0 | **54.5** | 36.7 | 35.3 | 8.6 | 23.1 |
| Mean (unweighted) | 56.7 | 45.4 | 51.7 | 30.3 | 23.1 | 18.9 | 15.4 |
| Mandible | | | | | | | |
| Hammon-Todd[1] | 71.0 | 71.7 | **75.1** | 40.0 | 29.5 | 23.6 | 13.9 |
| Japan[2] | 21.7 | 25.8 | **46.5** | 12.9 | 23.5 | 16.7 | 22.5 |
| Dickson[3] | 53.3 | 56.7 | **73.3** | 40.0 | 27.7 | 30.0 | 13.3 |
| Neanderthals[4] | 22.6 | 25.5 | **48.9** | 11.8 | 2.5 | 7.6 | 1.8 |
| Mean (unweighted) | 42.1 | 44.9 | 60.9 | 26.1 | 20.8 | 19.4 | 12.8 |

[1]Unweighted group means computed from El Najjar et al. (1978).
[2]Yamamoto (1988).
[3]Goodman and Armelagos (1985a).
[4]Ogilvie et al. (1989).

El-Najjar and co-workers (1978) also presented defect prevalence data by tooth type (see Table 1). These data indicate clearly that some factor, other than time of development, influences the prevalence of defect formation among teeth. Teeth with similar developmental periods, such as the incisors, have sharp differences in prevalence of enamel defects. Profound differences in prevalence of defects by tooth type were also found by Black (1979) in an analysis of teeth from the Turner Site, a Mississippian Cemetery from Southeast Missouri.

Research on fossil hominid DDEs and histological abnormalities was also undertaken during this period. Falin (1961) found a high proportion of enamel defects in Bronze and Stone Age teeth from eastern Europeans and attributed these defects to nutritional inadequacies. Brothwell (1963) surveyed the extant literature and found a great increase in the reported frequency of enamel hypoplasias in the mesolithic versus the late paleolithic. White (1978), following the previously published observations of Robinson (1956) and Tobias (1967), found a high prevalence of enamel hypoplasias on first molars of australopithecines. White also reported that australopithecines who died with hypoplasias seem to be at a younger developmental age than those without hypoplasias.

The paleoepidemiological studies of enamel defects during the 1960s and 1970s established general methods for analysis of surface and microscopic defects, including a methodology for estimating the developmental age of the individual at time of defect formation. A plethora of epidemiological insights were also made. Deciduous tooth defects were commonly found both prenatally and perinatally, while permanent tooth defects were found to peak most often between 2 and 4 years of age. The demonstration that enamel defects were associated with skeletal signs of nutritional deficiency and a decreased life expectancy provided support for the notion that these "benign defects" had adaptive significance.

## Studies in developing countries

While research was beginning on the paleoepidemiology of enamel defects, the first systematic studies were also commencing of enamel defects in less-developed countries. These focused on young children and deciduous tooth defects. Enwonwu (1973), for example, examined 872 Yoruba children under 7 years of age. He found that 21% of individuals from the lower socioeconomic status group had an enamel defect, compared to none in a high socioeconomic status group.

Similar findings were presented by Infante and Gillespie (1974) for children from Guatemala, and by Infante (1974) for White Mountain Apache children. Nearly one in five Apache children (19.4%) had one or more hypoplasias on their

deciduous maxillary central incisors. If carious lesions, believed to be secondary to hypoplastic involvement, were included then the prevalence doubled to 38.9%. Infante (1974) considered most of these defects to be prenatally developed and cited the "near poverty" conditions of the reservation as causally related to the high prevalence of enamel hypoplasias.

Sweeney was one of the first researchers to associate directly the development of LEH in humans with infectious disease and nutritional status (Sweeney et al., 1969, 1971). The prevalence of enamel hypoplasias on deciduous maxillary central incisor teeth of Guatemalan children was higher in those who had an infection during their 1st postnatal month (Sweeney et al., 1969). An increased prevalence of enamel defects was also found in children suffering from more severe grades of malnutrition. Nearly three-fourths (73.1%) of children with third-degree malnutrition (<60% weight-for-age) had an enamel hypoplasia, as did 42.9% of children with second-degree malnutrition (61–75% weight-for-age) ($P < .01$; Sweeney et al., 1971).

Derrick Jelliffe, a pioneer in the fields of nutritional assessment, international nutrition, and infant feeding, frequently noted the occurrence of enamel hypoplasias in deciduous anterior teeth of malnourished children in the Caribbean (Jelliffe and Jelliffe, 1971). In fact, this association was so certain to Jelliffe that he included a description and picture of enamel hypoplasia in his monograph on the assessment of nutritional status (Jelliffe, 1966).

### The end of the period

By 1980 few studies continued to focus on the relationship between enamel quality and caries. The study of enamel defects in experimental animals also slowed, perhaps because of the realization that rodent incisors were a poor choice for studying human dental development (Fejerskov, 1979). Conversely, clinical and epidemiological studies of enamel defects were on the rise by 1980. The most rapidly developing lines of research included studies of enamel histology and surface defects in archaeological populations.

Unfortunately still, a full appreciation of the utility of these defects as indicators of malnourishment was hindered by a variety of issues, including the lack of understanding of differential susceptibility by tooth class and crown location. The nonspecificity of enamel to disruption, a characteristic that might have disheartened researchers working within a particularistic medical paradigm, fit the growing interest in general stress mechanisms in human adaptability and bioarchaeology research (Buikstra and Cook, 1980; Goodman et al., 1980, 1988b; Huss-Ashmore et al., 1982; Martin et al., 1985; Rose et al., 1985).

## *Recent studies: 1980 to the present*

### Clinical and epidemiological studies: developed countries

The 1980s have witnessed a growing number of clinical studies of enamel hypoplasias, opacities, and microstructural defects. At this time the relationship between fluoride and dental defects became a public health issue (Rao, 1984; Richards et al., 1967). Work on "dental fluorosis" stimulated further studies of histological changes in enamel (Fejerskov et al., 1979; Yaeger, 1966) and aided the establishment of epidemiological indexes (Dean, 1942; FDI, 1982; Horowitz et al., 1984; and Pendrys and Katz, 1989).

The development of the FDI index was followed by a number of well-designed epidemiological surveys of enamel defects primarily in New Zealand and the United Kingdom (Cutress et al., 1985; Suckling and Pearce, 1984; Suckling et al., 1985; de Leifde and Herbison, 1985). Cutress and co-workers (1985) suggested that most defects in Auckland were *not* related to fluoride ingestion. Conversely, Suckling and Pearce (1984) found an increase in opacities with greater fluoride exposure in a study of 12 to 14 year old children from Richmond, New Zealand, and de Liefde and Herbison (1985) noted a similar pattern in children from Hawke's Bay.

Suckling and Pearce also found an increased prevalence of DDEs in individuals with a history of severe illness or accident.

A growing interest in neonatology also sparked interest in enamel defects. The relationship between neonatal complications and the development of deciduous tooth defects received considerable attention (see Bhat and Nelson, 1989, for a recent review). Most of these studies compared the teeth of control children with those who had diagnosed abnormalities (Grahnén and Larsson, 1958; Grahnén et al., 1969, 1972, 1974; Noren, 1983, 1984; Noren and Gillberg, 1987; Noren et al., 1978a,b, 1984; Magnusson et al., 1978). A variety of methods were employed and correlated, including macroscopic, light microscopic, and ion probe analyses.

Defective enamel has also been used as an indication of physiological disruption during the pre- or perinatal period (Judes et al., 1985; Levine et al., 1979). Murray and Johnsen (1985; Murray et al., 1987) found correlations between hearing deficits and the timing of systemic disturbances, as indicated by the position of a DDE on primary incisors. Children with hearing loss frequently had an enamel defect which occurred early in gestation (on incisal thirds of deciduous incisors). The more severe hearing losses were associated with the earlier occurrence of systemic disruption (Murray and Johnsen, 1985).

A similar relationship between neurologic impairment and the time of systemic stress has also been found (Murray et al., 1987). Jaffe and co-workers (1985) found that enamel defects occurred in over 75% of children with idiopathic brain damage, compared to less than 25% of control children. Most cases of brain damage could be traced to abnormal events during gestation. Jaffe et al. (1985) also suggested that because enamel and neurological tissues share a similar embryonic ectodermal origin, an insult to the developing brain, sufficient to result in damage, would leave evidence of the insult on the growing enamel.

Currently there is a tremendous rise in interest in enamel defects in clinical dentistry and dental epidemiology. A frequently debated issue concerns whether defects caused by excess fluoride (e.g., fluorosis) can be distinguished from defects due to other systemic causes. While some of the markings of "fluorosis," such as bilateral defects, are characteristic of other systemic insults, fluoride seems to differentially cause dental opacities. More severe opacities and hypoplastic defects occur only with higher fluoride burdens (Driscoll et al., 1983, 1986; Horowitz, 1986). Archaeological studies may be useful in resolving this controversy, as it is difficult to find a contemporary population that has not been exposed to exogenous sources of fluoride through nonlocal foods, medicines, and dental products.

## Histological studies of skeletal populations

The 1980s have witnessed a dramatic increase in the number of studies using Wilson bands and other enamel microdefects to establish the level of childhood stress in prehistoric skeletal samples. However, as studies increased, new methodological problems were encountered, including 1) variable definitions of Wilson bands and other microdefects, 2) calculation of the individual's age at formation of a microdefect, and 3) compensation for the influence of mortality selection upon microdefect frequencies. A fourth problem, not addressed in the literature, is the comparability of microdefect frequencies between different tooth types (e.g., incisors and canines).

The definition of a microdefect is influenced by the choice of observational instrument. The three criteria to date are presence of brown pigmentation (Molnar and Ward, 1975), variation of calcification evaluated with polarized light (Cook, 1981), and abnormal prism morphology (Rose et al., 1978). Rose's original definition of a Wilson band included both abnormal enamel prism morphology and an abrupt change in prism direction along the entire length of the band as it passes from the DEJ to the surface (Rose, 1977; Rose et al., 1978). This required tedious observation of enamel sections at high magnification (1,000×) and eliminated from consideration many potential Wilson bands.

Condon (1981) and Rudney (1981) asserted that Rose's definition was too restricted and difficult to apply consistently. Condon (1981) suggested that only an abrupt change in direction of the enamel prisms was necessary to identify a Wilson band, and that the proper use of oblique transmitted light will make them stand out as either ridges or troughs at relatively low magnifications (e.g., 160×). Rudney (1983) weighted bands with both changes in direction and abnormal prism structure twice that of bands exhibiting only abrupt changes in prism direction. Jablonski (1983) described the Wilson bands as exhibiting dark pigmentation, abnormal prism structure, and an abrupt change in direction.

One of the hindrances in arriving at a reliable and agreed upon definition of a microdefect concerns optical methods and equipment. Both Kohler illumination (parallel transmitted light) and Nomarski phase contrast optics were employed for Wilson band identification in Jablonski's analysis (1981), while Cook (1981) used polarized light to identify striae of Retzius and abnormal prism structures.

Rose (1983) used 23 different combinations of microscopes, optics, and specimen preparation techniques to identify microdefects on the same thin section. Little of the enamel prism morphology can be observed with Kohler illumination and standard transmitted light optics, with or without a coverslip. Using optics designed for use without coverslips improves resolution considerably. Nomarski reflected light optics did not aid in the identification of abnormal prism structure because the translucency of the enamel diffuses the light and obscures the surface detail. Coating the surface with carbon or gold, as done in the preparation of specimens for use with scanning electron microscopes, improves the resolution dramatically and highlights the abnormal prism structure; however polarized light is no better than Kohler illumination in resolving prism morphology, and the multiple color patterns can obscure some of the details. It is clear that the appearance and definition of a Wilson (pathological) band is dependent upon the specific procedures and technology utilized. Specimen preparation and the optical configuration of the microscope must be included as part of the definition. Our experience, however, suggests that the technique described by Condon (1981) is the simplest, most reliable, and least time consuming.

The second problem encountered by those using Wilson bands to study childhood stress is establishing the age in the individual's life when the band was formed. Following the standard of Massler and co-workers (1941), Rose (1973; Rose et al., 1978) divided the DEJ of mandibular canines into eight equal ½ year age intervals. This method was also adopted by Clarke (1978), Jablonski (1981), and Wright (1987, 1990).

Cook (1981), however, notes that big teeth would have more enamel in a ½ year unit than small teeth and the Wilson band chronologies for small and large teeth would not be well correlated. Cook's alternative was to compute the frequency of microdefects per millimeter of DEJ. Rudney's (1981) method is similar as he converts the distance from the cusp tip to the Wilson band into a percentage of the total DEJ length. Condon (1981) used the timing of crown development, determined from radiographic studies (Moorrees et al., 1963), and the median sample DEJ lengths to construct a chart for converting distances from the cervical end of the DEJ to the Wilson band into an age at formation. This method adjusts the growth chronology to the mean tooth size of the sample and, also, assumes a uniform rate of DEJ extension. While Condon's method seemed to work well, cross matching of Wilson bands on canines and first premolars demonstrated that the radiographic ages of the two teeth were not in good correspondence, probably because of the difficulty of observing canine development from a radiograph. Additionally, because radiographic studies are based on calcification and not matrix formation, it was determined that radiographic standards would underestimate the age of defect formation.

None of the aforementioned studies offered a clear resolution to the problem of integrating the assumption of constant rate of DEJ extension, variation in the crown heights of teeth, and populational variation in the ages of crown initiation

and completion into a usable system for determining the age distribution of Wilson bands within a sample. For example, if the conversions of Wilson band to cervical DEJ distances to age of formation are adjusted to the sample crown heights then the assumption of uniform DEJ extension is violated. If one either employs the frequency of Wilson bands per mm segment of DEJ (Cook, 1981) or converts the distance between the band and the cervical DEJ to a percentage of total DEJ length (Rudney, 1981), then a distribution of Wilson bands by developmental age does not follow.

Recent work on growth rates determined from clinically extracted teeth suggests that the assumption of a constant rate of DEJ extension may be invalid for permanent teeth (Gohdo, 1982; Shellis, 1984). Shellis indicates that the rate of DEJ extension falls from a high level at the occlusal DEJ to a constant rate as the tooth grows cervically. On bigger teeth, the occlusal rates of extension are higher and fall to their constant rate over a longer distance than on teeth with smaller cusps (Shellis 1984). This suggests that a uniform chronology of dental growth can be used as long as the conversion of Wilson band to cervical DEJ distances are adjusted for total DEJ length of the sample.

Regarding the problem of mortality selection, Cook (1981), Rose et al. (1978), and Rudney (1981) all reported that individuals with microdefects have a lower mean age at death than those without or with fewer of these microdefects. This implies that individuals with enamel defects are more susceptible to stress and are more likely to enter the skeletal sample at an earlier age. Consequently, Wilson band rates and age distributions could differ significantly between two skeletal samples because of differences in their mortality experience. In other words, the sample with the largest proportion of old individuals will have the lower rate independently of differences between childhood stress levels.

Rose et al. (1978) solved this problem by demonstrating the absence of significant differences in age structure among his three samples, and used only the teeth of individuals dying after 15 years of age (mortality selection is highest for subadults). Others have used analysis of variance to test for the influence of age at death on the frequency and distribution of Wilson bands (Rudney, 1981) or have compared all frequencies between age-specific groups (i.e., birth to 2 years; 3 to 11 years, etc.) (Cook, 1981). These studies all demonstrate modest solutions to the problem of mortality selection.

A fourth problem not mentioned in the literature is the differential susceptibility of the teeth to Wilson band formation. Each researcher has employed a single tooth for all samples investigated, and no one has yet compared the frequencies among teeth. This is unfortunate because Condon (1981), using mandibular canine and first premolar pairs from the same individual, demonstrated that the canine was more susceptible to metabolic disturbance than the premolar. During the same period of development, the canine always had more frequent and more severe enamel microdefects than the premolar. Because Wilson bands and enamel hypoplasias show similar patterns of inter-tooth susceptibility, we recommended that Wilson band data be collected from permanent maxillary central incisors and mandibular canines.

## Enamel surface defects in skeletal populations

Whether enamel hypoplasia patterns provide evidence for changing morbidity during the economic transition from gathering-hunting to agriculture has been addressed in many recent studies of enamel hypoplasias. Of the case studies in Cohen and Armelagos's (1984) book, at least ten include clear and specific information on the prevalence of enamel hypoplasias in permanent teeth: Angel (1984) for the Eastern Mediterranean, Smith et al. (1984) for the Levant, Martin et al. (1984) for Nubia, Cook (1984) for the Lower Illinois Valley, Goodman et al. (1984b) for Dickson Mounds, Cassidy (1984) for the central Ohio Valley, Perzigian et al. (1984) for the Ohio River Valley, Dickel et al. (1984) for California, Ubelaker

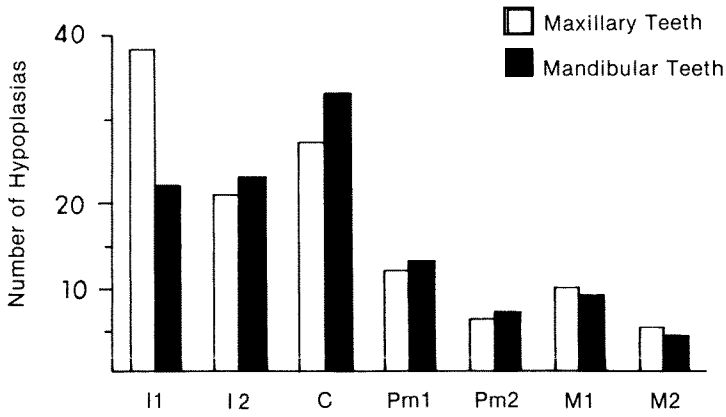


Fig. 17. Comparison of frequencies of linear enamel hypoplasia by different tooth types in a sample of 30 individuals from Dickson Mounds (from Goodman and Armelagos, 1985a).

(1984) for Ecuador, and Allison (1984) for Peru and Chile. All ten of these studies find some increase in enamel hypoplasia rates with agricultural intensification.

A number of other interesting epidemiological applications of enamel hypoplasia data have been published. Corruccini and co-workers (1985) examined the frequency and chronological distribution of linear enamel hypoplasias for a slave population from Barbados. They found a peak frequency of defects around 3 years of age and attribute this peak to post-weaning stresses, an assertion supported by ethnohistorical accounts. Other studies of slave populations and free blacks in colonial United States suggest a high degree of developmental stress (Blakey, 1987; Rathbun, 1987; Kelley and Angel, 1987; Rose, 1985). Lunz (1987) similarly found a high prevalence of LEH in skeletons from South African blacks and Chinese laborers. These studies suggest that the analysis of LEH frequencies might provide an objective evaluation of the hardships inherent in the conditions of slavery and apartheid.

The age of individuals at development of enamel hypoplasias in the Hammon-Todd skeletal series was studied by Goodman (1988), who was interested in whether the Hammon-Todd chronology was more similar to that proposed as universal by Sarnat and Schour (1941) or to chronologies found in archeological and prehistoric groups (such as Goodman et al., 1984a, Swärdstedt, 1966; Schulz and McHenry, 1975). Goodman (1988) found a peak frequency of defects around 2 to 3 years of age, more similar to the peak observed in prehistoric series, and further suggesting that the Sarnat and Schour chronology is not universal.

A series of studies of enamel developmental defects were performed on the Dickson Mounds skeletal series. Goodman et al. (1980, 1984a) studied permanent teeth and found an increased frequency of enamel hypoplasias in the agriculturally more intensive group. They also found some statistical evidence for an annual cycle of stress (1980) and a peak period of hypoplasias between about 2 and 3 years of age (1984a). Blakey and Armelagos (1985) examined deciduous teeth and found a high proportion of defects occurring prenatally.

On a methodological level, Goodman and Armelagos (1985a,b) focused on the intra- and inter-tooth pattern of susceptibility to enamel hypoplasias. They found that most defects occurred on the maxillary central incisors and mandibular canines. Anterior teeth were more hypoplastic than posterior teeth and maxillary teeth were more hypoplastic than mandibular teeth. Within developmental fields, the more polar teeth were the most hypoplastic (Fig. 17). These general patterns have been observed in other studies (Tables 1, 2). A clear epidemiological implication of these findings is that the prevalence of hypoplasias varies with the tooth chosen for study.

*TABLE 2. Comparison of proportion (%) of combined developmental defects of enamel by tooth type in different contemporary populations*[1]

| | I1 | I2 | C | Pm1 | Pm2 | M1 | M2 |
|---|---|---|---|---|---|---|---|
| Maxilla | | | | | | | |
| Murray and Shaw (1979) | 30 | 20 | 8 | 12 | 15 | 27 | 15 |
| Suckling et al. (1976) | 16 | 10 | 7 | 6 | 6 | 9 | 0 |
| Richards et al. (1967) | 9 | 7 | 5 | 2 | 1 | 2 | 1 |
| Young (1973) | 19 | 9 | 3 | 5 | 4 | 8 | 3 |
| Smith (1979) | 27 | 6 | 6 | 8 | 10 | 13 | 6 |
| Mean (unweighted) | 20.2 | 10.4 | 5.8 | 6.6 | 7.2 | 11.2 | 5.0 |
| Mandible | | | | | | | |
| Murray and Shaw (1979) | 5 | 4 | 5 | 10 | 7 | 23 | 16 |
| Suckling et al. (1976) | 10 | 7 | 6 | 7 | 6 | 10 | 0 |
| Richards et al. (1967) | 2 | 1 | 1 | 1 | 1 | 2 | 2 |
| Young (1973) | 7 | 6 | 3 | 5 | 4 | 8 | 3 |
| Smith (1979) | 12 | 6 | 7 | 10 | 10 | 11 | 2 |
| Mean (unweighted) | 8.0 | 4.8 | 4.4 | 6.6 | 5.6 | 10.8 | 4.6 |

[1]Modified from Cutress and Suckling (1982).

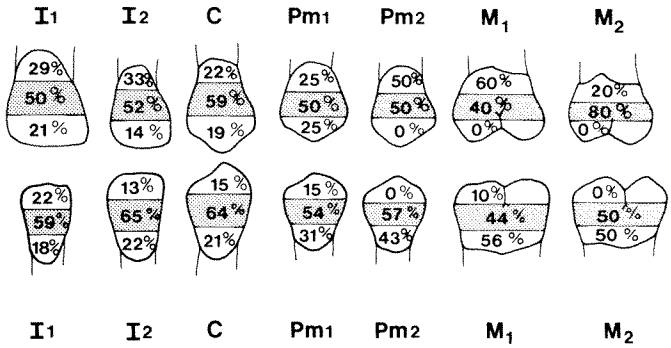


Fig. 18. Distribution of linear enamel hypoplasia within teeth from Dickson Mounds (from Goodman and Armelagos, 1985a).

Goodman and Armelagos (1985a) also found that the middle third of teeth were invariably the most hypoplastic (Fig. 18), followed by the incisal/cuspal and cervical thirds. It is also clear from our observations that labial sides of anterior teeth are more hypoplastic than lingual sides. Finally, Schuman and Sognnaes (1956) long ago had observed that the prevalence of defects differs widely across primate species. Our unpublished observations and the recently published results of Cecchi and co-workers (1988), Skinner (1986), and Vitzthum and Wikander (1988) suggest that defects are highly prevalent in the great apes and relatively rare in monkeys. All of these results have implications for understanding the meaning of epidemiological patterns of enamel defects. These consistent differences in "susceptibility" to enamel disruption may provide a key to understanding normal enamel development.

## Epidemiological studies in developing regions

Epidemiological studies of enamel defects continued in developing countries during the early 1980s (Alcorn and Goodman, 1985; Sawyer and Nwoku, 1985; Schamschula et al., 1980). Schamschula and co-workers (1980) found a 2.5 times greater risk of an enamel defect in Native Australian schoolchildren, versus "Caucasian" schoolchildren from New South Wales, Australia. In Nigeria, there were no severe hypoplasias in well-nourished individuals, compared to a prevalence of 15% among malnourished children (Sawyer and Nwoku, 1985). In South Africa, whites have the lowest prevalence of defects on both the permanent and deciduous teeth (Har-

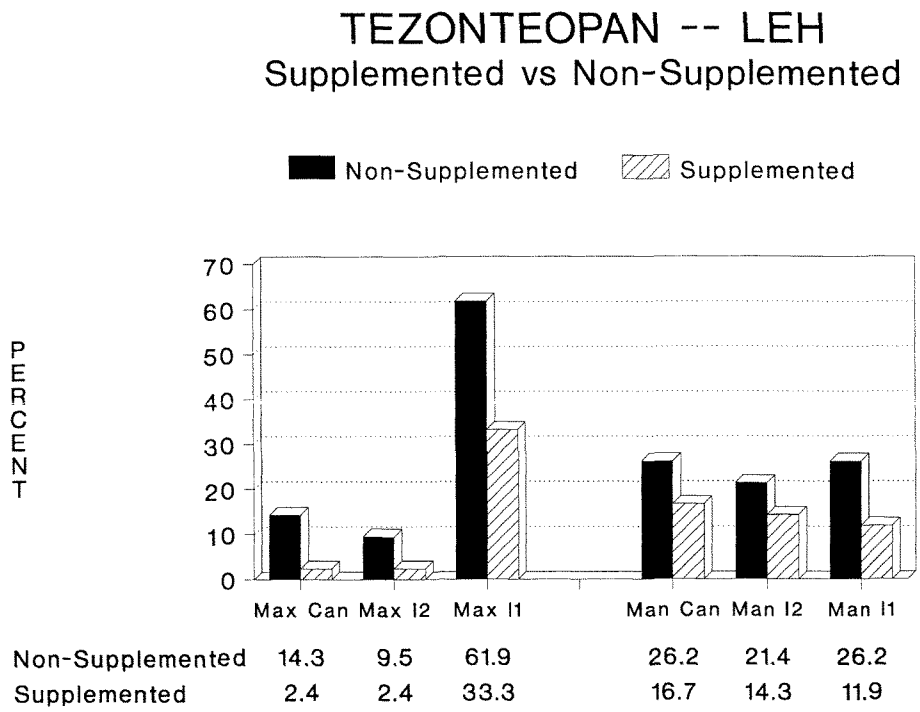


Fig. 19. Differences in linear enamel hypoplasia between nutritionally supplemented (n = 42) and control children (n = 42) from Tezonteopan, Mexico. Only defects which were observed on both antimeres were scored as true hypoplasias.

greaves et al., 1989a,b). This study updates Lunz's (1987) study from earlier in the century.

Concerned with the potential use of enamel defects as surveillance indicators of undernutrition, Goodman et al. (1987) examined 300 children from five villages in the Solis Valley of highland Mexico. Anterior teeth displayed a high prevalence of defects, with 46.7% of children having one or more hypoplasias. Most defects appear to develop between 18 and 30 months, or right after the documented mean age at weaning for these communities (Allen et al., 1987). Individuals with enamel hypoplasias tend to be from lower socioeconomic status families and to have a lower weight-for-age and height-for-age percentile (Goodman et al., 1988a). However, opacities did not display this epidemiological pattern. This cross-sectional study suggests that there is a general relationship between living conditions and the occurrence of enamel hypoplasias, but not enamel hypocalcifications.

A prospective research design has been employed with a sample of adolescents and young adults from Tezonteopan, Mexico. Half of this group of 84 received daily nutritional supplements since birth, while the matched controls did not. These two groups differ in nutritional status, as well as the prevalence of disease (Chavez and Martinez, 1982). Goodman and colleagues (1989) found that the control group did not have an excess frequency of pits (FDI type 3) or opacities (FDI types 1 and 2). However, the control group had nearly twice the frequency of LEH as the supplemented group (Fig. 19). Studies now in progress are considering the role of respiratory and diarrheal diseases on the development of enamel defects in this community. Ongoing studies in Cameroon (Maunders et al., 1990) and Mexico

(Goodman et al., 1987, 1989), among others, should further our understanding of the specificity and sensitivity of enamel defects.

## FUNDAMENTAL ISSUES AND DIRECTIONS
### *Identification and analysis of defects*

#### Choice of teeth

Because teeth exhibit differential susceptibility to defects, it is extremely important that the same tooth type be selected for study. When time is limited, as it often is, analysis can focus on a few key teeth without losing much information (Goodman et al., 1980; Goodman and Armelagos, 1985a). For both the permanent and deciduous human teeth, the maxillary central incisor and the mandibular canine are generally the most hypoplastic (Table 1) and frequently studied (Goodman and Armelagos, 1985a). Cecchi and co-worker's (1988) and Skinner's (1986) data also support the notion that these teeth are frequently hypoplastic in nonhuman primates.

We suggest that all studies of permanent and deciduous teeth include data on the maxillary central incisor. Lower canines, and to a slightly lesser degree upper canines, are also highly susceptible to disruption and provide information on a longer period of development, hence they are useful second teeth for study. All anterior teeth are easily studied in living individuals and Goodman and coworkers (1987, 1989) have recorded defects on all 12 anterior teeth. A dozen teeth can be studied relatively rapidly. The variety is useful for confirming the systemic nature of a disruption and providing a range of different levels of susceptibilities, which can yield insights into the severity of a disruption (see Fig. 19).

This last point is illustrated by comparison of enamel defects on maxillary teeth (incisor to first molar) from seven Cameroonian groups (Maunders et al., 1990). Teeth which most positively distinguish different ethnic and socioeconomic groups were often the least hypoplastic. In the comparison of supplemented and nonsupplemented Mexican children, Goodman et al. (1989) also found that the relative risk of hypoplasias was greatest for the least susceptible teeth (Fig. 19).

If LEH is to be used as a surveillance indicator then it will be necessary to streamline its method of identification. A surveillance study might include data on only the upper central incisor teeth. However, if other researchers find that the less susceptible teeth are better able to differentiate levels of stress, then this would certainly suggest including one or more of these less susceptible teeth. A suggestion for further research is to determine the best single tooth or combination of teeth for a basic surveillance study.

We have already noted that Condon's (1981) histological study of mandibular canine and first premolar pairs is the only one that addresses the issue of differential susceptibility of teeth to Wilson band formation. Condon (1981) demonstrated that during the same period of development, the canine always had more frequent and more severe enamel microdefects than the first premolar. There is a great need to extend this research to a comparison of Wilson bands on all tooth classes. In the meantime and in accord with our recommendations for surface defects, we suggest that epidemiological studies of enamel microdefect should focus on the most sensitive teeth—maxillary central incisors and mandibular canines.

#### Preparation and recording

Teeth from skeletal remains should be cleaned in a nonabrasive fashion to remove excess dirt and other obscuring objects. If a substance such as calculus cannot be removed and it obscures the tooth enamel then the concealed section needs to be recorded as unobserved. Similar procedures need to be taken with living individual and non-human primates. In studies of primate dental microwear, Teaford and Oyen (1989) suggest the use of an oral irrigation device such as the "Water Pick." In addition to cleaning, drying the teeth helps to study defects, especially opacities,

in living individuals (King, 1989). Any sort of directed air will facilitate drying. One to 2 minutes of directed air is sufficient for drying teeth of primates (Teaford and Oyen, 1989) and our experience suggests that even less time is required for observation of the labial surfaces of human anterior teeth.

The recording of DDEs should be performed with proper lighting and the location and type of defect should always be noted. A fundamental extant issue concerns the delineation of "minimal criteria" for an LEH and other defect types, and the meaning of defect dimensions such as LEH width and breadth. The observation of surface defects is aided by having a strong source of diffuse light, and a second mobile source that can be directed to provide oblique lighting. Our experience suggests that incandescent lighting is considerably better than fluorescent lighting. In work in the field it is frequently impossible to find strong artificial lighting. However, we have had good success working outside on days that are not overly sunny and working inside near a window on sunnier days.

In studies of skeletal materials, a streomicroscope and a small hand-held magnifying glass has proved useful. However, these devices should only be used for confirmation of a defect. If a defect can be seen only under magnification then it is likely to be too small to reliably record and is probably not a true hypoplasia.

Defect widths and breadths are characteristics about which there has been frequent speculation. For example, Guita (1984) asserts that the width of a band is a function of the duration of the causal stress and Shklar and McCarthy (1976) suggest that the depth of a defect is a function of the severity of the stress. As the severity and duration of the causative agent are often important epidemiological questions, one might automatically assume that one should record depth and breadth. However, it is difficult to do so accurately and defect dimensions may not be as important as has been assumed. Breadth is the more easily determined of the two measures and has been studied (Blakey and Armelagos, 1985; Hutchinson and Larsen, 1988). Hillson and Jones (1989) have recently tested equipment that might provide an on paper representation of tooth surface variation from which both defect breadth and depth might be easily measured. We suggest that one should not assume that defect dimensions are equal to dimensions of the underlying stress. However, these are hypotheses that should and can be tested experimentally or epidemiologically.

Rose (1983) could not find any effect of slightly varying preparation techniques on the histological observability of Wilson bands. The dental sections must be thin enough to allow light to pass through the enamel, although a specific thickness is not required, and polished to remove all scratches. The sections are then etched and observed *without* a coverslip, which permits the observer to focus upon the etched enamel surface. Rose (1977) found that etching sections with a 1 N solution of hydrochloric acid for 15 seconds worked best. Etching for less time does not sufficiently differentiate the enamel prisms and longer times begin to obscure the details. Wright (1987), however, found that etching in hydrochloric acid produced a precipitate on the enamel surface, so she chose instead to etch with 0.074 M phosphoric acid (0.05% by volume of 85% $H_3PO_4$ stock solution). Both methods produce clearly observed enamel prisms.

The instrumentation used to observe microdefects is an integral part of the definition of a Wilson band. The most practical microscope set up is transmitted white light with objectives designed for use without coverslips, although standard objectives are suitable. Nomarski reflected light optical systems produce the desired results only when the translucency of the enamel has been eliminated by sputter coating with carbon or gold. When using a transmitted light microscope, the substage microscope condenser should be off-centered and the high-power condenser tilted to direct the light into the thin section at an angle to the line of view through the optics.

These conditions result in Wilson bands appearing at low magnifications (e.g., 40–200×) as three-dimensional troughs or ridges, an optical pattern which can be

reversed by turning the slide 180 degrees. Conversely, most normal striae of Retzius will be obliterated, and those that remain are easily distinguished from Wilson bands by their absence of a three-dimensional image. The fact that these optical phenomena are produced by both abnormal prism morphology and an abrupt change in prism direction has been verified by observing Condon's (1981) Wilson bands with a scanning electron microscope (Marks 1988a). Marks (1988b) found that Wilson band identification is comparable with both light (as described above) and scanning electron microscopy.

Condon (1981) established three criteria for recording Wilson bands. First, the band must be observed on both the labial (buccal) and lingual enamel portions of the crown. Second, the band must be continuous from the DEJ to the enamel surface. Third, at high magnification (400× or greater) the prisms along the band must be observed to bend into the section or stop. We suggest some modifications to these criteria based upon more recent studies. First, the Wilson band must appear as a continuous trough or ridge extending from the enamel surface to three-fourths of the distance to the DEJ. The differential susceptibility of ameloblasts is such that those just beginning to secrete matrix are remarkably resistant to stress, and many well-defined Wilson bands do not extend into the region adjacent to the DEJ. Additionally, as the three-dimensional appearance produced by the oblique lighting is sufficient to identify this characteristic and distinguish them from striae of Retzius, it is not necessary to observe the prisms bending into or stopping at the band margin. With these modifications of Condon's definition, Wilson bands can be easily and unambiguously identified.

Both Rudney (1983) and Cook (1981) report two grades of microdefects and imply that there is a gradient of severity which must be considered. Conversely, our interpretation of Wilson band formation indicates that there can be no severity gradient because either the ameloblasts respond to stress and secrete abnormally structured matrix (Wilson band) or they do not respond (normal enamel). We suggest that the observed variation in Wilson band configuration is due to differential susceptibility of the ameloblasts. Any survey of enamel will locate small groups of abnormal enamel prisms which were produced by equally small groups of ameloblasts having physiological difficulties. It is only when all or almost all of the ameloblasts active at one time experience physiological perturbation that the resulting defect is considered a Wilson band. There are neither severity grades nor subtypes of Wilson bands.

Ameloblasts, however, do vary in their susceptibility within and between teeth, and thus the Wilson bands will vary slightly in their configuration. Ameloblasts are very resistant to physiological perturbation when they first begin to secrete enamel matrix and thus Wilson bands seldom extend to the DEJ. As well, ameloblasts in the most occlusal portion of the enamel are very resistant to stress, while those forming the most cervical enamel are more susceptible. Thus, Wilson bands in the more occlusal enamel seldom extend to the DEJ and the band of deformed prisms is very narrow, while more cervical bands almost always extend to the DEJ and often the band width appears thicker. In fact, the most cervical labial ameloblasts of the permanent canines appear to be so susceptible that it is not uncommon to see five or six clearly demarcated Wilson bands in a small portion of enamel. In addition, the enamel is so thin in this area that Wilson bands and striae are difficult to distinguish. We suggest that Wilson bands not be recorded in the terminal 1 to 2 mm of the labial enamel of permanent canines.

There is another microdefect type which should also be recorded and analyzed separately from Wilson bands. This defect consists of a Wilson band like stria (three-dimensional trough or ridge image) that extends from the enamel surface to one-half or less of the enamel width. Such bands appear to correspond to Condon's (1981) "band hypoplasia defect." However, because they almost always appear in clusters the term "Cluster bands" might be more suitable. Future research should be conducted to determine their epidemiological pattern and significance.

## Maintaining and testing for reliability

*Reliability* comprises two related concepts—repeatability and interobserver reliability. *Repeatability,* or intraobserver reliability, is a measure of agreement in observation and measurement of a phenomenon by the same observer at different occasions, while *interobserver reliability* is a measure of the extent of agreement between two different measurers (Cohen, 1960; Landis and Koch, 1977; Zerfas et al., 1985). The general effect of not collecting data reliably is that individuals with legitimate defects will be classified as not having defects, and those without defects will be classified as having defects. Randomness increases as the prevalence of these errors of classification increases, and randomness ultimately makes it much more difficult to uncover real trends and differences (Lilienfeld, 1976).

In general, poor reliability does not engender a directional bias. However, if one neither has a standard of comparison nor performs a repeatability test then it is possible to "drift," as data collection continues, and become more or less stringent in one's criteria for judging a DDE over time. For example, an inexperienced observer may become increasingly able to recognize an enamel defect on successive data collection days, or one might recognize more defects when one is most alert during a certain time of day.

Before commencing study one should test for reliability. This can be achieved with a sample of about 30 teeth which can be studied on different days (repeatability) and, if more than one observer is involved, by the different observers. Excellent guidelines for analysis of nominal scale data are provided by Fleiss (1973) and Cohen (1960), and by Landis and Koch (1977) for categorical data.

The DDE Index (FDI, 1982) has greatly increased comparability and reliability of surface defects. Murray et al. (1984), for example, report 86.4% intraobserver agreement in England and Goodman et al. (1987) report 80.8% interobserver agreement in Solis, Mexico. Once a acceptable degree of reliability has been obtained, one should typically repeat every tenth individual.

Interested researchers could make a number of important contributions by performing different repeat observation tests under varying conditions. For example, questions concerning the importance of differences in lighting or of drying teeth might be more formally answered by a repeat measurement cross-over design. Similarly, it would be useful to compare the relative reliability of defects of various sizes.

The same general guidelines should apply to the study of histological defects. With the methods outlined above, one should also be able to reliably score Wilson and Cluster bands. At present, however, we are unaware of any formal studies of repeatability or interobserver reliability of histological defects. Such studies should be carried out and reported.

## Permanent records: casts and photographs

For a number of reasons it is often desireable to cast and/or photograph teeth in order to obtain a permanent record of surface phenomena. Goodman et al. (1989) and Dobney (Dobney and Goodman, 1990) have had success using a polysiloxane impression material such as Coltene's or 3M's "Express" impression material in children from Mexico and England, respectively. Positives of the impressions may be made using a super hard 4 to 1 resin (Tap Plastics, Dublin, CA 94568). This method, which is similar to that used in studies of dental microwear (Teaford and Oyen, 1989), provides a very-high-quality replica. A photographic record of enamel defects in contemporary Jordanians was tried by Alcorn and Goodman (1985). Black and white prints were taken with a macro lens and ring flash. This standard method of oral photography was not very effective. Subsequently, much clearer reproduction has been achieved using color slide film, a 100mm macro lens with a bellows and flash attached to a rotating bracket, mounted at the end of the lens (Freehe, 1983).

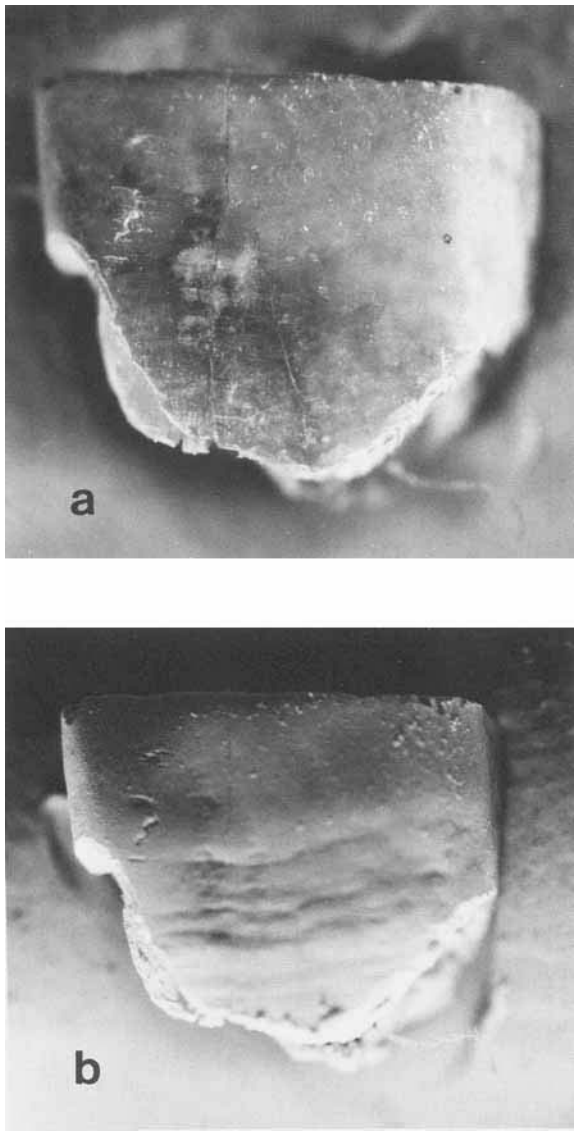


Fig. 20. Photographs of a broken right maxillary central incisor before (**a**) and after (**b**) coating with ammonium chloride. Note the increased surface detail after coating.

Because the enamel is translucent and reflects back diffuse light, teeth have always been difficult to photograph. The solution of Kraus and co-workers (1969) was to make casts of the teeth and photograph the casts. This has frequently been the accepted procedure among dental anthropologists, despite the fact that it is time consuming and relatively expensive. An effective solution is to make the enamel opaque and give the surface a matte finish.

An easy method for accomplishing this is to heat ammonium chloride (granular form) in a test tube over a bunsen burner and pass the tooth repeatedly through the resulting vapor until it is uniformly coated. Before (Fig. 20a) and after (Fig. 20b) photographs of a broken right maxillary central incisor demonstrates the in-

creased detail. This is especially important in the recording of hypoplasias. The enhanced texture and loss of translucency makes even the smallest surface details stand out under oblique incandescent lighting.

### *Individual age at defect formation*

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 defects in enamel matrix formation is that, unlike nearly any other possible indicator, one can infer the time of their formation. In this section we provide an assessment of some of the possible errors involved in the age determination process, and then suggest directions for future research.

We have already noted that Sarnat and Schour (1941) were the first to focus on the chronometric potential of enamel hypoplasias. Locations of enamel hypoplasias were compared to the standard of enamel crown development of Massler and coworkers (1941). They even suggested that each .1 mm of defect width corresponds to 1 month's duration (1941). Swärdstedt (1966) refined the method of Sarnat and Schour (1941) and relied on a number of assumptions in developing a chronology of enamel hypoplasias. We focus on the main assumptions and issues in developing a method for estimation of the time of a defect.

#### Choice of developmental standard

All current research rests on the standard established by Massler and coworkers (1941). This standard has historical precedence, which alone is a poor justification for its continued use. Although comparability is also increased by use of the same standard, one should consider whether it is most suitable for each specific population.

Massler et al.'s (1941) data are not optimal. The actual sample size, presumably about 1,000 teeth, as well as its relationship to the prior study of Logan and Kronfeld (1933), are points of contention. As well, because they were from autopsies, a possible bias toward delayed development exists. Finally, estimates of variation in development are not provided, nor of possible differences between boys and girls.

Interpretation of this standard has also varied. All that was unambiguously provided were ages at the beginnings and ends of main events such as crown formation. Swärdstedt (1966) apparently interpreted diagrams provided by Massler et al. (1941) to show intratooth variation in the rate of enamel formation. Although Goodman et al. (1980) initially followed Swärdstedt's interpretation, Goodman subsequently chose to adopt an assumption of constant velocity (Goodman et al., 1987, 1989; 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 enamel extension rates, it might be best to continue with a "null hypothesis" of constant velocity of enamel development (Goodman and Rose, 1990).

There are at least two main advantages in using the Massler standard in comparison to standards based on more recent and methodologically more sound studies. First, the Massler et al. (1941) chronology provides data on all teeth, deciduous and permanent, at all developmental stages, thus allowing comparability across teeth. Second, this standard is based on the direct observation of enamel; it is a matrix formation standard rather than a maturation/calcification standard (contra Moorrees et al., 1963, and others). More recent developmental studies, by comparison, tend to rely on the radiographic appearance of calcification. Accordingly, they introduce a systemic error because calcification occurs at an unknown length of time after matrix formation (matrix formation cannot be seen radiographically). Furthermore, these studies tend to commence around the ages of 3 and 4 years, thus eliminating data on all deciduous crown development and most of the development of the permanent crowns (e.g., Demirjian and Levesque, 1980; Haavikko, 1974; Moorrees et al., 1963). Finally, the populations upon which these standards

are based, typically middle and upper class North Americans or Europeans, are likely to be a poor choice for application to most anthropological groups.

More research is needed on the pattern of dental development before one can estimate how precisely the enamel hypoplasia chronologies reflect individuals' actual ages at the time of defect formation. 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 it does not share the scientific qualities of many newer standards, it has been commonly used and it is in substantial agreement with other standards (see Cameron and Sims, 1974; Gustavson and Koch, 1974) (see Table 4).

### Methods for converting defect locations to developmental ages

There are a number of methods by which one can translate locations of defects on teeth to an individual's age at the time of defect formation. Most researchers divide enamel development into half-year or yearly developmental periods (e.g., Schulz and McHenry, 1975; Swärdstedt, 1966). It should be stressed that these data are *estimates of ages at development*, with an error of unknown magnitude. The larger the developmental periods, the more likely a defect which was empirically assigned to the period actually developed during that period. However, larger zones provide a less-fine-grained estimate of ages at stress, and may make it more difficult to uncover meaningful phenomena such as annual cycles of stress.

With agreement on the underlying chronology, a number of methods are available for calculation of the individual age at time of defect formation. Following Swärdstedt (1966), most researchers have developed some form of graphic method for converting the distance of a defect from the CEJ to an age at time of development (Goodman et al., 1980, 1984a; Rose et al., 1985). With increased access to calculators and computers, we now feel it is easier to use a series of regression equations for converting a location of an enamel defect to an age at formation. Murray and Murray (1989) have recently developed a computer program, based on regression equations developed by Walker (1990). The program converts the distance between a LEH and the CEJ to an age at LEH formation, and saves the results as an ASCII file.

If one is to adopt the assumptions of constant velocity then the regression will be a simple linear one. The only data that are needed for constructing a regression equation are the ages at beginning and end of crown formation and the total crown height. Age at formation then equals the negative of the rate of enamel development (in the inverse of mm per year) multiplied by the distance of the defect from the CEJ, plus the intercept (the age at crown completion). The equations provided in Table 3 are based on the crown heights used by Swärdstedt (1966) and the ages at beginning and end of crown formation of Massler et al. (1941).

The main advantage of the regression equation procedure is that one can easily test the effect of changing an assumption by simply changing the parameters of the equations. For example, Hodges and Wilkinson (1988), for permanent teeth, and Blakey and Armelagos (1985), for deciduous teeth, have estimated the error involved in estimating age at formation using different mean tooth crown heights. Using an assumption that smaller teeth take just as long to develop as larger teeth, both studies reported that in populations where tooth size variation is relatively small, as might be expected in most anthropological populations, the error involved is minimal, usually less than a half-year. However, Hodges and Wilkinson (1988) suggest that this error might be quite substantial in a mixed series in which tooth size variation is substantial.

In general, differences in tooth size will have a negligible effect on the estimation of the age at development of a defect for later-developing defects (those near the CEJ) because the required velocity adjustment operates on a relatively short height of enamel. Conversely, if a velocity adjustment of 20% or greater is required, then this may introduce a difference of around 1 year in the estimated age at development of a defect near the occlusal surface of a permanent tooth.

*TABLE 3. Regression Equations for estimation of age at linear enamel hypoplasia formation*[1]

| Tooth | Formulae[2] |
|---|---|
| Maxillary Teeth | |
| I1 | Age = −(.454 × Ht) + 4.5 |
| I2 | Age = −(.402 × Ht) + 4.5 |
| C | Age = −(.625 × Ht) + 6.0 |
| Pm1 | Age = −(.494 × Ht) + 6.0 |
| Pm2 | Age = −(.467 × Ht) + 6.0 |
| M1 | Age = −(.448 × Ht) + 3.5 |
| M2 | Age = −(.625 × Ht) + 7.5 |
| Mandibular Teeth | |
| I1 | Age = −(.460 × Ht) + 4.0 |
| I2 | Age = −(.417 × Ht) + 4.0 |
| C | Age = −(.588 × Ht) + 6.5 |
| Pm1 | Age = −(.641 × Ht) + 6.0 |
| Pm2 | Age = −(.641 × Ht) + 7.0 |
| M1 | Age = −(.449 × Ht) + 3.5 |
| M2 | Age = −(.580 × Ht) + 7.0 |

[1]Age = age in years; Ht = distance of the LEH in mm from CEJ.
[2]Regression equations are based on mean crown heights of Swärdstedt (1966) and the crown formation standard of Massler et al. (1941).

Another parameter that could be varied is the estimate of times of matrix formation. Most developmental standards are rather consistent in their mean ages at development for early stages of crown development (Gustafson and Koch, 1974) and have relatively small measures of variation for the earlier stages (e.g., Moorrees et al., 1963). Therefore, a change in the regression equation based on a change in standard is likely to have a greater effect on the latter stages of crown development, the defects nearer the CEJ. As an illustrative example, the mean age at completion of mandibular canine crown calcification reported by Moorrees et al. (1963) is about 4.0 years for both males and females, a full 2.5 years earlier than that estimated by Massler et al. (1941). If Moorrees and co-workers' (1963) estimation is correct, then this would introduce a substantial correction, of up to 2.5 years, for defects near the CEJ. However, the new change in slope (rate of crown development) compensates for the change in intercept for earlier-developing defects.

Contrary to the above example, most standards are not radically different from that of Massler et al. (1941). Table 4 is a comparison of a summarized average of about 20 studies compiled by Gustavson and Koch (1974) compared to the original estimates of Massler and co-workers (1941). The two estimates of beginning crown calcification, for maxillary and mandibular central incisors to second molars, are all within .75 years, and all but the lower second molar are within .5 years. The estimates for the age at crown completion are in similar strong agreement. All estimates are within .8 years of, and all but one, the second upper premolar, is within .5 years.

Another potential error in estimating age at development of surface hypoplasias involves the length of time between initial matrix formation and when striae of Retzius reach the outer enamel. When a tooth begins matrix formation, the initial striae form partial circles which begin and end in the inner enamel (Fig. 1). Only after an unknown length of time do the striae of Retzius end near the surface. Bromage and Dean (1985) estimate this period of time to be approximately ½ year for permanent incisor teeth, an estimate which may be a bit liberal. Regardless, it would be highly useful to devise a means to measure the time involved from initial cusp tip amelogenesis to the time that the first stria reaches the enamel surface.

Much of the above discussion applies equally to histological defects. Condon (1981) made a chart for converting the distance of a Wilson band from the CEJ (measured along the DEJ) to an age at time of formation (canine chart reprinted in

*TABLE 4. Comparison of permanent tooth crown formation times from Massler and co-workers (1941) and Gustavson and Koch (1974)*

| | Crown formation begins | | | Crown formation ends | | |
|---|---|---|---|---|---|---|
| | Massler[1] | G & K[2] | Difference[3] | Massler[1] | G & K[2] | Difference[3] |
| Maxillary teeth | | | | | | |
| Central incisor | 0.0 | 0.25 | −.25 | 4.5 | 4.5 | 0.0 |
| Lateral incisor | 1.0 | 0.88 | .12 | 4.5 | 5.0 | 0.5 |
| Cuspid | 0.0 | 0.37 | .37 | 6.0 | 6.0 | 0.0 |
| First bicuspid | 2.0 | 1.75 | −.25 | 6.0 | 6.0 | 0.0 |
| Second bicuspid | 2.5 | 2.0 | −.50 | 6.0 | 6.8 | 0.8 |
| First molar | 0.0 | 0.0 | .00 | 3.5 | 3.0 | −.5 |
| Second molar | 3.0 | 2.9 | .10 | 7.5 | 7.5 | 0.0 |
| Mandibular teeth | | | | | | |
| Central incisor | 0.0 | 0.25 | −.25 | 4.0 | 4.1 | 0.1 |
| Lateral incisor | 0.0 | 0.25 | −.25 | 4.0 | 4.5 | 0.5 |
| Canine | 0.5 | 0.37 | .13 | 6.5 | 6.0 | −.5 |
| First premolar | 1.0 | 1.75 | −.75 | 6.0 | 6.0 | 0.0 |
| Second premolar | 2.0 | 2.1 | −.10 | 7.0 | 6.8 | 0.2 |
| First molar | 0.0 | 0.0 | .00 | 3.5 | 3.0 | 0.5 |
| Second molar | 3.0 | 2.9 | −.10 | 7.5 | 7.2 | 0.3 |

[1]From chronology of Massler et al. (1941).
[2]From chronology of Gustavson and Koch (1974).
[3]Difference between chronologies.

Rose et al., 1985), but this chart will not provide suitable ages for teeth bigger or smaller than his sample. In order to better relate microdefect timing to surface defects timing we suggest measuring microdefect distance from the CEJ along the outer enamel, rather than the DEJ. If this is done, then the same formulae can be used for both histological and surface defects (Table 3) and the distributions of Wilson bands and hypoplasias can be compared.

Perhaps the most important advice that can be given is that the raw measurement data (locations of enamel defects from CEJ) should always be saved and published when possible. If this is done, model parameters can be changed and chronologies can be reconstructed when other standards are developed and clearer estimates of bias are developed.

## Individual and population differences in crown formation times

Even with a standard that meets stringent criteria, one will find variation in matrix formation 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; Demirjian and Levesque, 1980). If one continues to use a standard that does not provide separate developmental times for males and females, then it might be reasonable to provide a correction factor. Generally speaking, however, this correction will be small; frequently less than 1 month for permanent tooth crown and even less for deciduous tooth crowns (Haavikko, 1970, 1974; Moorrees et al., 1963; Demirjian and Levesque, 1980; Trodden, 1982). In fact, Demirjian and Levesque (1980) suggest that there are no differences in timing of dental development between boys and girls before the age of 5 to 6 years.

The relationship between tooth development and tooth size is extremely interesting and not fully explored. The central question is whether a larger tooth develops more rapidly (per unit of enamel) or requires more time to develop (Blakey and Armelagos, 1985). 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, as has been assumed by Hodges and Wilkinson (1988).

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 sta-

tus influences the rate of tooth eruption (Alvarez et al., 1988; Alvarez and Navia, 1989). It may, therefore, be assumed that matrix formation might also be so affected. However, we presently do not have good information on the degree to which undernutrition effects crown matrix formation.

Populations and individuals within populations may also 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 consideration of environmental conditions (Garn et al., 1973a,b).

Working with the standards and information at hand, it would be helpful to obtain a better estimate of confidence interval size and systemic biases. We are close to being able to estimate the affect of sex on formation times, and the delay from initial matrix formation to the time stria of Retzius reach the outer enamel surface. On the other hand, we are further away in estimating the role of nutritional differences within and among communities and how to account for variation in tooth size. Basic research into these developmental factors will profoundly aid interpretations of epidemiological studies.

### *Patterns of differential susceptibility to disruption*

One of the most exciting developments in the study of enamel defects has been the increased realization that ameloblasts that form the enamel on different teeth are not equally sensitive to developmental disruption. Although differential susceptibility may seem to be a barrier to comparing results and to ease of epidemiological analysis, these patterns may offer a number of useful insights. These include an opportunity to understand how ameloblasts function at a fundamental level, and may also provide a means for analysis of the severity of the underlying stress.

We have noted a number of susceptibility patterns within the human permanent dentition, but less is known of nonhuman mammals. Gorillas display a high frequency of hypoplasias and the tooth specific pattern is similar to the human pattern (Skinner, 1986). Dobney (1983) has studied hypoplasias in archaeological pigs and Wilson (1988) has studied bison hypoplasia. Hypoplasias have been noted in ruminants, carnivores, and other animal groups. However, monkeys are infrequently hypoplastic (Schuman and Sognnaes, 1956; Vitzthum and Wikander, 1988). Free-living great apes, on the other hand, appear to have an extremely high prevalence of defects (Cecchi et al., 1988; Skinner, 1986). Understanding these patterns may provide some keys to understanding the cause of the differential susceptibility gradients and may have either phylogenetic or functional implications. For example, knowing whether or not Miocene primate enamel displays a pattern of disruption consistent with apes or monkeys could contribute to understanding phylogenetic relationships.

Human deciduous teeth are far less hypoplastic and have far fewer Wilson bands than their permanent successors. While the low frequency of defects on deciduous teeth has most often been attributed to their developing during a protected period, they may also have a differential susceptibility. Interestingly, the same pattern of susceptibility seems to hold true for opacities. Gedalia and Shapira (1989) note that the infrequency of opacities in the deciduous teeth is usually thought to result from the difficulty of transporting fluoride across the placenta. However, research since the 1970s clearly shows that fluoride readily passes across the placenta from the maternal to the fetal blood (Gedalia and Shapira, 1989).

We are not certain what explains all these differential patterns of susceptibility. They could be due to a similar underlying cause, or to some combination of factors. The differences between morphologically similar teeth which develop at about the same time may be due to genetic canalization. Polar teeth may be more canalized and subsequently more genetically stable (Waddington, 1957). Canalization may also be a factor in explaining the high rate of enamel defects on functionally key teeth such as the gorilla's sectorial complex (maxillary canine and mandibular

third molar). Ameloblasts that function for a longer period of time may become increasingly susceptible to disruption. This may explain the general trend to find a high proportion of defects on bigger teeth, such as gorilla canines, and to find few defects on rapidly developing small teeth, such as in the human deciduous dentition and in monkey dentitions. Additionally, a number of potentially significant phenomena tend to be coincident, including 1) angle of prisms, 2) curvatures of the striae of Retzius, 3) "age" of the secretory ameloblasts, 4) rates of enamel secretion, and 5) lengths of prism. Of all these factors we suggest that the age of the ameloblast might be most significant. When enamel secretion is disrupted the more recently activated ameloblasts, those that are most cervical, produce the least disrupted prisms. Physiologically, it is plausible to suggest that older ameloblasts might somehow become fatigued.

Finally, we need to be cognizant of whether differential susceptibility truly refers to differences at the cellular level, or only relates to differences in surface manifestation of developmental disruption. Condon's work clearly shows that the pattern of differential susceptibility seen on the surface (Goodman and Armelagos, 1985a,b) is also observed in section (Condon, 1981). However, the angle of the striae of Retzius may be an additional factor in determining the outward expression of a disruption. As has been previously shown, a defect near the CEJ often displays a number of defective striae, while having only a shallow surface disruption.

There is a variety of fascinating patterns of susceptibility of enamel to developmental disruption. These range from intratooth patterns to differences across tooth classes, dentitions, and taxa. Research is especially needed on patterns of enamel defects in non-human primates and other taxa. These patterns should help elucidate the pattern of stress in primate groups and provide a mechanism for testing the cause of differential susceptibility patterns.

### *Sensitivity and specificity: the meaning of enamel defects*

A wealth of data from clinical, epidemiological, and experimental studies now exists to suggest that enamel hypoplasias are generally the result of a wide variety of stressors acting during amelogenesis (Cutress and Suckling, 1982; Goodman and Rose, 1990; Jontell and Linde, 1986; Pindborg, 1982; Rose et al., 1985; Suckling, 1989). We suggest that no effort to relate enamel defects to a specific cause, such as hypocalcemia (Nikiforuk and Fraser, 1981), has been successful. But then, what does cause an enamel defect? How important are nutritional versus non-nutritional factors? Are there certain nutrients that are most important? What length of disruption and severity is needed to form a hypoplasia? We start this section, purposefully saved for the end of this paper, holding a strong belief in the general relationship between stress during amelogenesis and enamel hypoplasias. But, how do researchers move beyond this general relationship? How sensitive and specific a measure of general stress are hypoplasias? What research might help to further our understanding of the sensitivity and specificity of this indicator?

#### Experimental research

The experimental research of Suckling et al. (1983, 1986) has greatly contributed to our understanding of the relationship between parasitism and enamel disruption. However, there have not been any similar studies with an appropriate animal model (pig, sheep, monkey) on the relationship between LEH and specific nutrient deficiencies, or on interactions between nutrients and disease.

Designing an experiment to show the relative effect of a specific nutrient deficiency is relatively straightforward. It is also very important to evaluate the effect of combinations of nutrient deficiencies and how specific nutrient deficiencies might interact with other common stressors, such as parasitism, in the etiology of enamel defects. While animal research is not likely to provide the final word on either sensitivity or specificity, it certainly can provide a variety of useful insights, especially regarding the issue of specificity.

### Clinical and epidemiological research

The majority of future contributions to understanding the meaning of enamel hypoplasias and microdefects is most likely to come from clinical and epidemiological studies in which health and nutrition conditions are well documented. A number of clinical studies have recently been reported on neonatal conditions and enamel defects (Bhat and Nelson, 1989). Clinical case-control studies, especially where the time of development of a condition is known, are one relatively facile means of further assessing the sensitivity and specificity of enamel defects. Hall (1989) recently reported a surprisingly high prevalence of enamel defects in prematurely born (56.5%) children and children with a history of rubella embryopathy (81.8%), compared to a control group prevalence of 9.3%. The high sensitivity of enamel defects to rubella embryopathy is only somewhat surprising. Clearly more studies are needed of children with clinical histories of infection and nutritional deficiencies.

An important handful of longitudinal studies of undernutrition and associated health conditions have been undertaken in developing countries. Children in these studies were typically followed for a period of time, with repeat measures of such factors as disease episodes, nutritional status, and food intake. If one is able to locate the previously studied children and examine their erupted teeth, then an assessment of the cause of DDEs could be obtained.

While we are optimistic about the potential contribution of epidemiological, clinical, and experimental studies toward understanding the cause of DDEs, we are less certain about the contributions that might be made from paleopathological studies. Paleoepidemiological studies will continue to be useful in providing a time depth for evaluating long term patterns and trends. However, we suggest that skeletal biologists who wish to ascertain validity will do better to work with living humans or with experimental animals in a situation in which causal factors are more precisely known.

Trying to predict how precisely we might understand the cause and meaning of DDEs and microdefects in the future is a highly uncertain undertaking. One possibility that needs to be anticipated is that we will not know much more than we now know regarding the precise cause of these developmental disruptions in most human groups. Animal experimental research is limited in its ability to determine sensitivity in humans, while epidemiological studies are confounded by a host of coincident and corelated factors. The association of disease and nutrient deficiencies in the real world makes it extremely difficult to understand the precise cause of enamel developmental defects.

This scenario is not as unsatisfactory as it might appear. A nonspecific indicator of stress is highly useful to studies of the functional and adaptive consequences of stress. Most anthropometric measures of nutritional status, for example, are highly nonspecific and astonishingly useful (Sutphen, 1985; Zerfas et al., 1985).

If LEH is highly sensitive but nonspecific, as the current research suggests, then they will be useful in future studies. There will be a place for them in the bioanthropologists' toolkit. The fact that these defects might also provide time-specific information is a particularly useful and unique additional feature (Goodman and Rose, 1990).

## CONCLUSIONS

This review has documented the long history of advancements in the study of enamel hypoplasias and associated histological structures. The study of these defects is maturing and gaining momentum. A great deal is now known about the etiology of these defects, enough in fact that they may now be usefully applied to a number of epidemiological questions. LEH and Wilson bands are particularly useful in determining the chronology of stress during periods of tooth crown formation and in providing a biologically meaningful, retrospective assessment of

stresses. Enamel is nearly unique among biological tissues in its ability to fossilize past physiological perturbations. On the other hand, a great deal of uncertainty exists concerning the actual meaning and interpretation of these defects and how they might be best studied.

What excites us is the realization that most of the problems before us are definable and amenable to study. The study of enamel defects will greatly change in the approaching years due to the combined efforts of nonanthropologists and anthropologists.

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