

# What Can Developmental Defects of Enamel Reveal About Physiological Stress in Nonhuman Primates?

DEBBIE GUATELLI-STEINBERG

Because of their durability, teeth are among the most common fossils found.<sup>1</sup> Dental enamel, in particular, is the hardest tissue in the human body and is capable of withstanding great masticatory force.<sup>2</sup> In view of enamel's near invulnerability, it may seem surprising that the process of enamel formation can be rather easily perturbed, resulting in irregularities known as developmental defects of enamel. In fact, such defects are relatively common in many primates, including humans, and provide a permanent record of physiological stress during the dental ontogeny of the individual. Consequently, they are a valuable source of comparative information about the life histories of living and fossil primates.

Several different types of developmental defects occur in enamel,<sup>3,4</sup> including opacities, which are changes in enamel translucency; accentuated striae of Retzius,<sup>1</sup> which are dark microscopic bands that can be observed in thin sections of enamel; and enamel hypoplasias,<sup>1</sup> or macroscopic defects appearing on the enamel surface (see Glossary). Opacities are caused by hypomineralization.<sup>1,2</sup> The latter two types of defects result from disruptions to enamel-producing cells, known as ameloblasts, as they secrete the enamel matrix (Box 1). Periods of malnutrition,<sup>2,5,6</sup> systemic illness,<sup>2,7–10</sup> and even, as new evidence suggests, psychologically stressful situations<sup>11</sup> can disrupt ameloblast function. Because enamel

does not remodel once a tooth has formed, accentuated striae of Retzius and enamel hypoplasias provide indelible markers of such disturbances.<sup>2</sup>

Anthropological interest in accentuated striae of Retzius and enamel hypoplasias has traditionally concentrated on humans. Because nutritional and disease stress are common causes of enamel hypoplasias, and because these defects can be easily observed on the surfaces of teeth, the prevalence of enamel hypoplasia is often used as an indicator of the health status of human populations.<sup>2</sup> Bioarcheological applications have involved documenting an increase in the prevalence of enamel hypoplasia in the transition from hunting and gathering to agricultural subsistence systems in various regions of the globe<sup>12–14</sup> and reporting a high prevalence of enamel hypoplasia in slave populations.<sup>15,16</sup>

Although Colyer<sup>17,18</sup> published findings on nonhuman primate enamel hypoplasia in 1936 and 1947, it is only during the last 15 years that this topic has become a strong focus of research interest and activity.<sup>19–38</sup> There is growing recognition that if developmental defects in enamel can help answer questions about the conditions under which humans experience stress, then

they may also be valuable in answering these questions about nonhuman primates. Interpretation of the prevalence of developmental defects in enamel in nonhuman primates is more complex than it is in humans, in which data derive from within a single species. Across primate species, differences in the prevalence and expression of such defects are likely to reflect not only differences in physiological stress experience, but also species-specific aspects of enamel morphology and development that affect how episodes of stress are recorded in teeth. Comparison of the prevalence of developmental enamel defects in populations of a single primate species, however, can offer a window into physiological responses to varying environmental conditions. Analysis of such defects at the level of the individual can clarify the relationship between stressful events in the lives of primates and the formation of enamel defects. Methods for determining when defects form during the life of a primate and the time intervals represented by successive defects can help to identify patterns in the timing of stress episodes that primates experience.

To date, a growing body of research on developmental defects of enamel in nonhuman primates has addressed two important questions: What do these defects reveal about the nature and timing of physiological stress in primate lives? How and why do the prevalence and expression of these defects vary across and within primate taxa? After providing background information I will discuss these questions in turn, emphasizing what has been learned and what further re-

Debbie Guatelli-Steinberg is an Assistant Professor in the Department of Anthropology at The Ohio State University. Her research focuses primarily on the use of developmental defects of enamel to understand patterns of stress in human and nonhuman primates.

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### Glossary

**Accentuated striae**—In thin sections of enamel, dark lines that either lie between or coincide with the incremental striae of Retzius.

**Ameloblasts**—specialized cells that produce enamel.

**Amelogenesis**—the process of enamel formation (detailed in Box 1).

**Appositional or cuspal enamel**—on the cusp of a tooth, successive domes of enamel covering the striae of Retzius.<sup>1</sup>

**Cross-striations**—small increments that lie between adjacent striae of Retzius and are known to have a circadian periodicity.<sup>81</sup>

**Developmental defects of enamel**—defects in enamel tissue that form as a result of disruption of the normal course of enamel development. These include enamel hypoplasias and accentuated striae of

Retzius, defects that form while the enamel matrix is secreted.

**Enamel hypoplasia**—defects of enamel appearing on the enamel surface in which there is an area of deficient enamel thickness as a result of disruption of ameloblasts during the secretory stage of amelogenesis.

**Imbricational enamel**—enamel covering the lateral surfaces of teeth in which perikymata are visible.

**Linear enamel hypoplasia**—enamel hypoplasias that take the form of lines or grooves on the surface of a tooth crown.

**Localized hypoplasia of the primary canine**—An enamel defect on the deciduous canine that takes the form of an approximately circular area of deficient enamel thickness.

**Neonatal line**—an accentuated stria of Retzius that forms at birth and is often visible in the first permanent molar.

**Perikymata (singular, perikyma)**—tiny wave-like enamel features that are surface manifestations of underlying striae of Retzius. Distinctions are made between perikymata grooves and ridges that make up the overall pattern of the perikymata.<sup>1,40</sup>

**Striae of Retzius**—brown or dark lines in longitudinal crown sections viewed at low magnification.<sup>1</sup> In a three-dimensional crown, the striae are actually a series of planes layered over each other. Striae of Retzius form with a periodicity that varies across and within species but is constant within an individual's dentition.<sup>52</sup>

### Box 1. Amelogenesis: The Process of Enamel Formation (Based on Ten Cate<sup>81</sup>)

Amelogenesis consists of a secretory stage, during which the enamel matrix is formed and mineralization begins, and a maturation stage, in which mineralization is completed. The entire process is controlled by ameloblasts, specialized epithelial cells that produce enamel.

#### 1. Secretory Stage:

Ameloblasts secrete a matrix consisting of two classes of proteins, amelogenins and enamalins, as well calcium and alkaline phosphatase. Amelogenins make up 90% of the protein in the enamel matrix<sup>79</sup>; they control the growth and orientation of enamel crystallites.<sup>80</sup>

Mineralization begins when crystals of dentin act as nucleators of the hydroxyapatite crystals of the enamel, which form from mineral in the matrix. The crystals of this first-formed enamel are packed randomly, intermingling with the dentin crystals. Once this structureless enamel layer is formed, ameloblasts migrate away from the dentin-enamel junction. Conical projections known as Tomes processes organize the formation of structured enamel. As the

ameloblasts reach the enamel surface they lose their Tomes processes and once again secrete a layer of structureless enamel. By the end of the secretion stage, enamel is 30% mineralized.

#### 2. Maturation Stage:

During this stage, ameloblasts undergo structural changes associated with new functions. Ameloblasts are now involved in a cyclical process in which water and organic materials are removed while inorganic material is introduced. Ameloblasts alternate between being ruffled-ended and smooth-ended. When the ameloblasts have ruffled borders, they secrete minerals needed for calcification. When the ameloblasts have smooth borders, they are removing water and protein. This cycling between the secretion of mineral and the uptake of water and protein continues until the enamel tissue is 96% calcified. As Goodman and Rose<sup>2</sup> note, enamel, the hardest tissue in the human body, is "well suited to its role in mastication" (p. 61).

search is necessary to answer them more conclusively.

### ENAMEL DEFECTS: TYPES, ETIOLOGY, TIMING, AND ISSUES OF INTERPRETATION

Developmental defects in enamel vary in size from macroscopic to microscopic. Studies of macroscopic defects in the enamel of nonhuman pri-

mates focus on enamel hypoplasias, of which there are several forms. The form most commonly studied,<sup>22–27,32–36</sup> known as linear enamel hypoplasia, appears as one or more horizontal lines or grooves on the surface of a tooth crown (Figs. 1A, 1B). Clinical standards define linear enamel hyperplasia as a macroscopic defect,<sup>3,4</sup> as do studies of linear enamel hyperplasia in nonhuman primates. How-

ever, even very small or microscopic linear hyperplasias, if they can be matched across tooth classes, are likely to represent a particular episode of stress that affected all teeth forming when the stress episode occurred.<sup>39,40</sup> Other macroscopic enamel hypoplasias, such as enamel pitting,<sup>21,30</sup> vertical groove defects,<sup>20</sup> and localized hypoplasia of the primary canine,<sup>28</sup> have also been de-

scribed in nonhuman primates (Fig. 1C).

To date, most studies of microscopic developmental defects in the enamel of nonhuman primates have focused on accentuated striae of Retzius<sup>11,41–45</sup> (Fig. 1D). These are similar to features of the enamel known simply as brown striae of Retzius, first described by the Swedish anatomist Anders Retzius.<sup>46,47</sup> At low magnification, striae appear as brown or dark lines in longitudinal crown sections<sup>5</sup> (Fig. 1D). In a three-dimensional crown, the striae are actually a series of planes layered over each other, representing the past location of the developing enamel front<sup>40</sup> (Fig. 2). The striae result from brief, periodic interruptions in ameloblast matrix secretion.<sup>1</sup>

The periodic nature of the striae has been established by counting smaller increments, known as cross-striations, that lie between striae of Retzius. Cross-striations themselves have a circadian periodicity.<sup>48</sup> In humans, the number of cross-striations between striae ranges from seven to ten in different individuals, with an average of eight.<sup>1</sup> The enamel between adjacent striae of Retzius thus represents approximately one week's worth of enamel growth in humans. This near-weekly periodicity is known as a circaseptan rhythm.<sup>1</sup> The causes of this rhythm are not known, but near-weekly rhythms exist in other human physiological systems.<sup>49</sup> In nonhuman primates, striae of Retzius have periodicities ranging from two to ten days in different species.<sup>50</sup> Accentuated striae of Retzius are prominent striae that either coincide with or lie between the periodic Retzius lines.

The etiology of accentuated striae is not well known, but is assumed to be similar to that of enamel hypoplasia.<sup>1</sup> One type of accentuated stria, known as the neonatal line, seems to be caused by the trauma of birth. This defect often appears in deciduous teeth and first molars, marking the transition from the intrauterine to extrauterine environment.<sup>1</sup> The etiology of enamel hypoplasias is well established, with nutritional stress and disease being the most common systemic causes of these defects<sup>40</sup> (Box 2).

Accentuated striae and surface hy-

poplasias are, in fact, related to each other. In the portion of the enamel known as imbricational enamel, which covers the lateral surfaces of teeth, the edges of the Retzius planes are exposed in a series of tiny furrows known as perikymata grooves<sup>1</sup> (Fig. 2). Perikymata are thus surface manifestations of underlying striae of Retzius. Hillson and Bond<sup>44</sup> have shown that linear enamel hypoplasias result when a “wider margin” than normal of “each brown stria plane is exposed” at the tooth surface (p. 97). Linear enamel hypoplasias are therefore surface remnants of a disturbance that affected one, and usually several, adjacent Retzius planes, leaving a furrow in the enamel surface that is larger than a normal perikymata groove (Box 3). The association between accentuated striae and enamel hypoplasias has been known for some time: Condon<sup>51</sup> found that all of the hypoplastic defects he examined were underlain by one or more accentuated striae. On the other hand, not all accentuated striae of Retzius are observable as macroscopic enamel hypoplasias. A long accentuated stria can be more easily observed in a thin section than can a tiny enamel hypoplasia that is only one perikymata wide on the surface of a tooth.

Because of the periodic nature of the striae of Retzius, it is possible to determine the timing of accentuated striae formation. The number of cross-striations between striae of Retzius, known as striae periodicity, is constant within a tooth and within the teeth of an individual,<sup>52</sup> but varies from individual to individual and across species. The time intervals represented by successive accentuated striae can be determined by using histological techniques to ascertain the periodicity of the striae of Retzius in a particular specimen. Furthermore, it is possible to discover how old an animal was at the time an accentuated stria formed by “registering” accentuated striae in the tooth in question with the pattern of accentuated striae in the first molar,<sup>41</sup> which records the event of birth in the neonatal line (see Fig. 3).

Determining the timing of hypoplastic defects can be accomplished by similar methods. Intervals between

linear defects can be estimated by counting perikymata. Perikymata are not visible in the cuspal or appositional zone of enamel, where successive domes of enamel cover the striae of Retzius.<sup>1</sup> (Fig. 2). However, using either a stereomicroscope or a scanning electron microscope, perikymata can be counted in the imbricational zone.<sup>1</sup> Accurately ascertaining the age of a hypoplastic defect, however, requires more than a surface examination of the tooth. The age at onset of mineralization as well as the time to form cuspal enamel in a particular tooth must be determined by histological methods. The difficulty with determining the age of hypoplastic defects is that most researchers investigating linear hyperplasias do not incorporate histological methods in their studies, and only recently have begun to count perikymata on tooth surfaces. Until recently, studies attempting to determine the age of defects have divided tooth crown height by the average crown formation time, based on the assumption that equal parts of the crown height take equivalent times to form.<sup>53</sup> This assumption has been shown to be erroneous in humans<sup>40,53</sup> as well as the great apes.<sup>54</sup>

Several issues of interpretation must be addressed, the first of which is observer error. Attempts have been made to standardize the criteria used for recording the presence of enamel defects. For example, The Fédération Dentaire Internationale Developmental Defects of Enamel Index<sup>3,4</sup> offers a classification system for types of macroscopic surface defects, ranging from opacities to hypoplastic pitting to linear enamel hypoplasias, which has proven useful in anthropological and clinical studies.<sup>50,58,59</sup> This index has yielded 86.4% intra-observer agreement and 80.8% inter-observer agreement.<sup>2</sup> However, inter-observer error in recording the presence of enamel hypoplasias is likely to be more of a problem than the index suggests. Enamel hypoplasias are expressed along a continuum from microscopic to macroscopic, so that it often is difficult to determine exactly what minimum criterion different researchers have used for recording the presence of a defect.

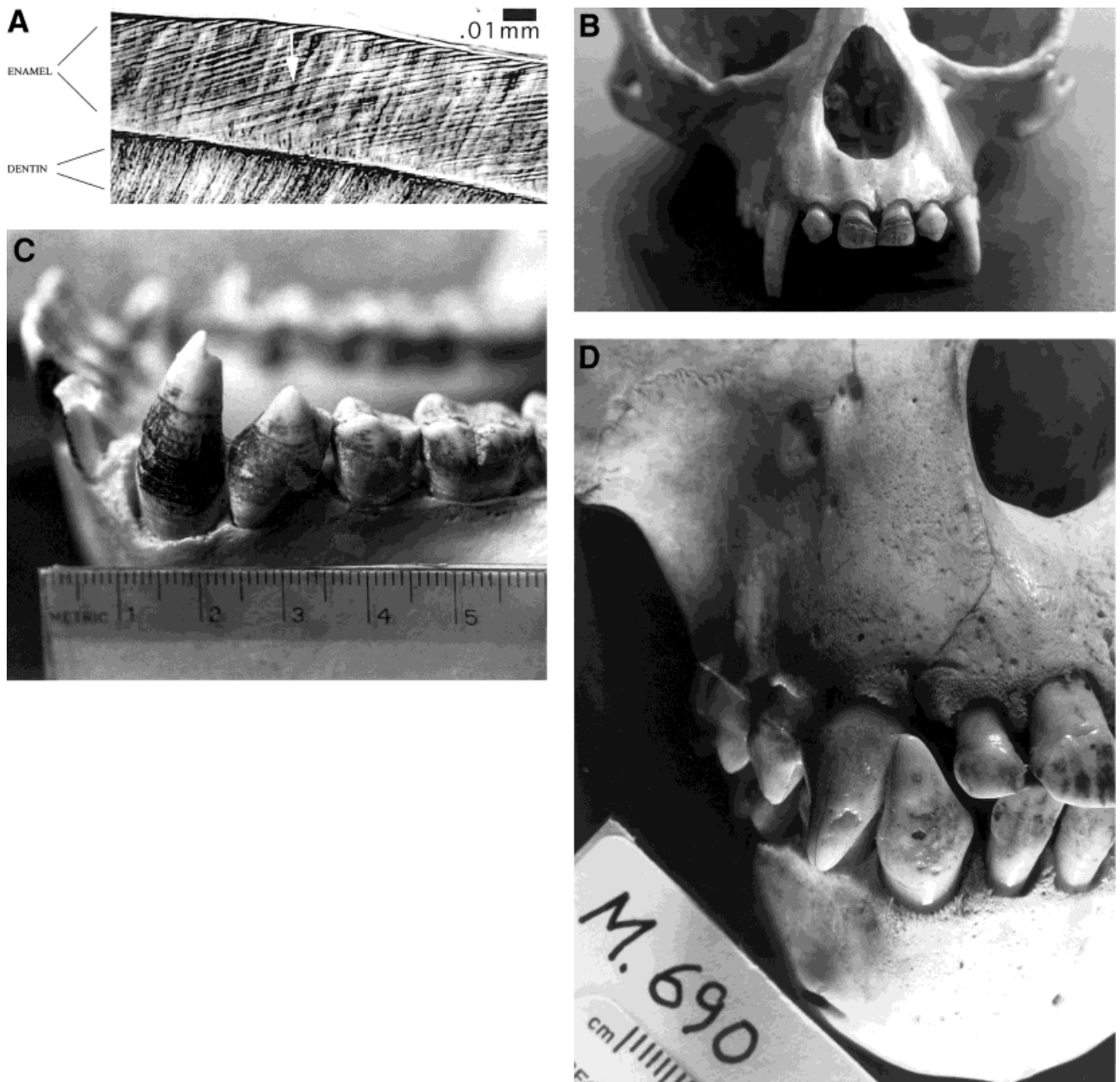


Figure 1. Developmental defects of enamel in primates. 1A: Paired LEH defects in the upper central incisors of a specimen of *Hylobates lar carpenteri* (Museum of Comparative Zoology specimen #41492; 1B: Eight LEH defects on the lower left canine of a specimen of *Pongo pygmaeus* (Museum of Comparative Zoology specimen #37358); 1C: Oblique  $\frac{3}{4}$  view of right deciduous canine teeth of a specimen of *Gorilla gorilla* (Powell-Cotton Museum specimen M-690; courtesy John Lukacs); 1D: Accentuated stria of Retzius in a canine section of *Semnopithecus entellus* (courtesy: Wendy Dirks).

A second issue of interpretation is the biological significance of hypoplastic defect dimensions. Suckling, Elliot, and Thurley<sup>9</sup> found that the amount of enamel missing in sheep enamel hypoplasias resulting from induced parasitism was related to the severity of the parasite load. Yet the relationship between the size of a de-

fect and the severity of the stressor, or even the duration of a stress episode, is not straightforward. Hillson and Bond<sup>40</sup> recently demonstrated that defect dimensions are related to variation in the spacing and form of perikymata in different parts of the tooth crown. According to these authors, because perikymata are spaced

more widely in the occlusal region a furrow that is 10 perikymata grooves wide in the occlusal part of the crown would be wider than one having a 10-PKG furrow in the cervical region. In addition, Hillson and Bond<sup>40</sup> suggest that only perikymata in the occlusal wall of a defect represent growth disruption; those in the cervical wall

### Box 2. The Etiology of Enamel Hypoplasia

Enamel hypoplasias have been linked to nutritional deficiency and infectious disease in humans<sup>5,6,10</sup> (see Hillson<sup>82</sup> and Goodman and Rose<sup>2</sup> for reviews) and other mammals.<sup>7,8,9,833</sup> Such growth disruptions cause ameloblasts prematurely to cease secreting the enamel matrix.<sup>40</sup> Kreshover<sup>7</sup> demonstrated that mice infected with tuberculosis have abnormal ameloblasts and hypoplastic teeth. Microscopic examination of the ameloblasts revealed irregular morphology, an excessive number of vacuoles, and displaced nuclei. Suckling and co-workers<sup>9</sup> found similar ameloblastic changes in association with enamel hypoplasia in sheep experimentally infected with nematode parasites.

Although growth disruptions such as nutritional deficiency and childhood fevers are the most common causes of enamel hypoplasias,<sup>40</sup> rare inherited conditions such as amelogenesis imperfecta, trisomy 21, and inborn errors of metabolism are also associated with hypoplastic defects.<sup>84,85</sup> In addition, local trauma to developing tooth germs as a result of physical injury (such as falls) or localized infection (such as periapical osteitis) may induce enamel hypoplasia.<sup>85</sup> Because of this variety of potential

causes, enamel hypoplasias are best seen as nonspecific indicators of physiological stress.<sup>2</sup> It is possible, however, to differentiate systemic from local causes. Defects matched across antimeres<sup>2</sup> (right and left teeth of the same type) and various tooth classes<sup>1</sup> are likely to result from systemic causes because these affect all teeth developing during a period of physiological stress.

Different types of enamel hypoplasias may have different etiologies. Localized hyperplasia of the primary canine, which, by definition, is restricted to primary canines, has been associated in humans with calcium deficiency, very low birth weight (<1,500 grams) and endotracheal intubation,<sup>86</sup> as well as nutritional disadvantage,<sup>87</sup> low sunlight,<sup>87</sup> and low levels of retinol.<sup>87</sup> The etiology of vertical enamel hypoplasias, which are rare in both human and nonhuman primates, is in large measure, unknown,<sup>20</sup> though these hyperplasias are associated with X-linked amelogenesis imperfecta in females.<sup>88</sup> Owing to differing potential etiologies, it is usually recommended that enamel hypoplasias be recorded and studied by type. In most recent studies, care has been taken to do so.

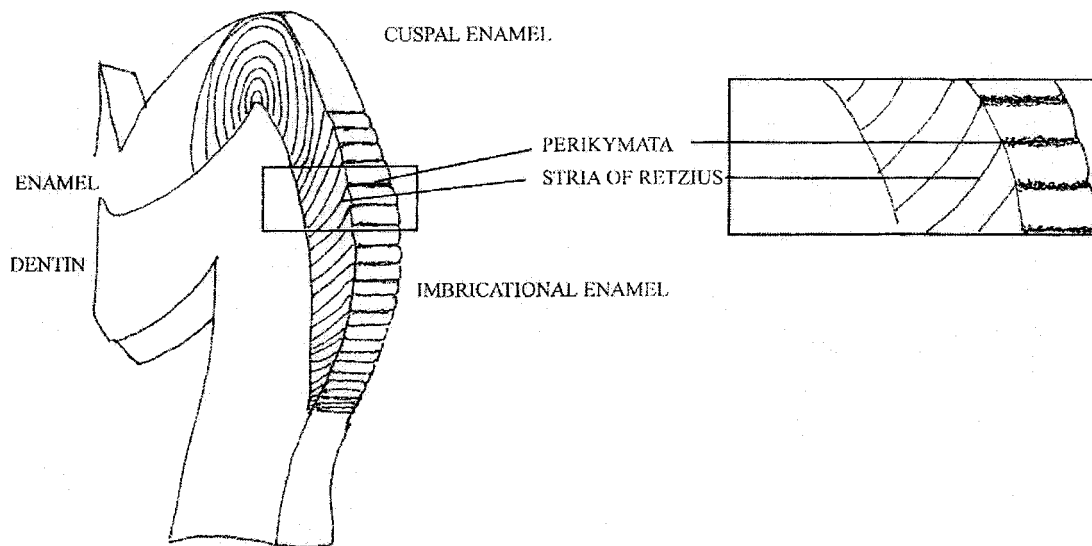


Figure 2. Diagram of tooth section, showing relationship of striae of Retzius to perikymata and the difference between appositional (or cuspal) enamel and imbricational enamel.

“seem to represent a recovery to the normal pattern of crown formation” (p. 97–98). Hillson and Bond’s<sup>40</sup> conclusions raise an important issue regarding the variability of perikymata spacing in primate species and the extent to which this variability may be related to taxonomic differences in defect dimensions.

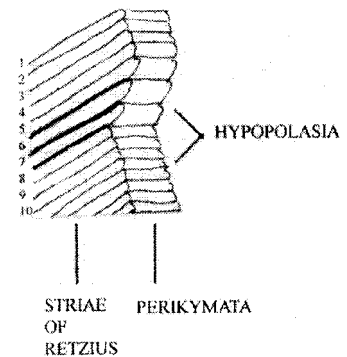
Another important issue of interpretation is the relationship between

the prevalence of enamel hypoplasia in a sample and the health status of a source population. Developmental enamel defects can only form during the period of enamel formation and thus provide a limited window on population health status. As Wood and coworkers<sup>60</sup> pointed out, there is no clear-cut connection between the stress recorded in the teeth of individuals who have survived the period of

enamel formation and aggregate health status. Under conditions of severe stress, a high prevalence of linear enamel hyperplasias will not occur if most individuals do not survive long enough to record stress in their teeth. A dental sample with a high prevalence of linear hyperplasia may therefore represent a population in which individuals experienced less severe stress than one with a lower preva-

### Box. 3. Hillson and Bond's Model for How an LEH Defect Forms

Compare this three-dimensional view of a portion of enamel with a hypoplastic groove to Figure 2, in which there are no hypoplastic grooves. This figure and associated explanation are based on Hillson and Bond H6 model of the formation of linear hyperplasia. The striae of Retzius are actually a series of planes that are layered over each other and are exposed on the enamel surface as perikymata. As a result of growth disruption, more of the surface of Retzius planes 5, 6, and 7 is exposed than it is in any of the other planes. Retzius planes 5, 6, and 7 form what Hillson and Bond refer to as the "occlusal wall" of the hypoplastic defect. Note accentuated striae underlying the occlusal wall of the defect.



lence of linear hyperplasia. Thus, differences in the prevalence of linear hyperplasia among samples may signify overall differences in stress experience among them. However, the nature of these differences, representing better or worse general health or differences in the timing of stress during dental development, can be determined only if additional health and demographic data are available.

#### WHAT DO DEVELOPMENTAL DEFECTS OF ENAMEL REVEAL ABOUT THE NATURE, TIMING, AND FREQUENCY OF PHYSIOLOGICAL STRESS IN PRIMATES?

Studies of developmental defects of enamel in captive primates with known life histories provide a valuable source of information about the kinds of events that can cause disruptions in enamel formation. In dental samples for which minimal life-history data are available, it remains possible, using the techniques described earlier, to determine when and how often disruptions occur. Studies focusing on the timing of defect formation suggest that there are regularities in the timing with which primates experience metabolic disruption in the wild.

Schwartz and coworkers,<sup>45</sup> determined the timing of accentuated striae in fourteen permanent teeth of a captive juvenile female western lowland gorilla that had been to a veteri-

nary clinic several times. These researchers found that accentuated striae identified in all teeth were associated with the exact days of this female's surgical procedures and follow-up clinic visits. It is important to note that the timing of accentuated striae was determined before this female's medical records were examined. The "blind" nature of this study makes the association between the veterinary clinic visits and accentuated striae particularly persuasive.

Bowman<sup>11</sup> was able to link life-history events with accentuated striae in captive female macaques. In one female, Indira, major accentuated striae coincided with three events: Indira's birth, the removal of all males from her social group, and the removal of Indira from her mother. These latter two associations suggest that psychological stress can disrupt enamel formation. This fascinating possibility requires further study.

Attempts have also been made to link enamel hypoplasias to stressful events or living conditions. For example, the prevalence of developmental enamel defects in specimens from captive environments has been compared to that in wild specimens.<sup>17,23,61</sup> The evidence of a difference in prevalence is not convincing, however, as it is based on small samples. Linear enamel hyperplasia has also been studied in the remains of Cayo Santiago rhesus monkeys.<sup>23,24</sup> Regular provisioning of this rhesus colony did not begin until 1956.<sup>23</sup> Individuals in which the teeth formed prior to regu-

lar provisioning had a greater number of stress events recorded in their teeth than did those in which the teeth formed after 1956.<sup>23</sup> However, the percentages of individuals with linear enamel hyperplasia did not differ significantly in the before-provisioning and provisioned groups.<sup>23</sup>

Several studies have focused on wild primates. Reid and Dirks<sup>43</sup> and Dirks and coworkers<sup>41</sup> studied the timing of accentuated striae in two female Awash baboons. Exact dates of death were known for these baboons, in which third molar crowns were still forming at death. The authors matched accentuated striae across teeth and determined the interval between the end of third molar formation and the preceding accentuated stria. They continued to work backwards, determining the intervals between accentuated striae until they reached the neonatal line, giving them both a date at birth and age at death. The authors then constructed a chart showing the developmental state of each tooth when each accentuated stria formed. Interestingly, the authors found that both baboons had accentuated striae that formed at birth (neonatal lines), at just over one year of age, and with greater frequency after four years of age. It is noteworthy that neither animal formed accentuated striae during the entire first year of life. The authors believe that the striae that formed just after one year of age may be related to the weaning process and acquisition of foraging independence. In the au-

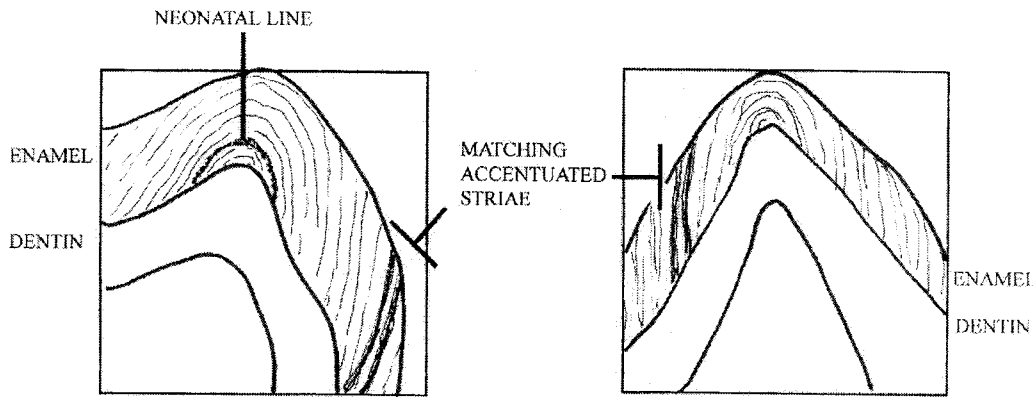


Figure 3. The figure at left shows a longitudinal section through a cusp of the first molar, while the figure at right shows a longitudinal section through a central incisor. Accentuated striae in the first molar can be aged with reference to the neonatal line. The accentuated striae in the first molar can be matched to accentuated striae that formed simultaneously in the central incisor.

thors' view, striae forming after four years of age may be related to physiological stresses associated with reproductive maturity. Both specimens also formed accentuated striae during a period of high rainfall in 1970 and a long drought from 1972 to 1973.

Estimates of defect timing have interesting applications to fossil samples. Recently, Kelley and Bulicek<sup>27</sup> demonstrated that in a sample of Middle Miocene hominoids from Pasalar, nine upper central incisors from seven or eight individuals had identical patterns of linear enamel hypoplasias representing two episodes of stress. The defects appeared at nearly identical locations on the crowns of these teeth. As determined by counting perikymata; The time between the two separate stress episodes was also essentially identical. On the basis of these findings, the authors conclude that they have identified a birth cohort that may imply birth seasonality in this sample.

One of the most intriguing aspects of studying the timing of developmental defects in the enamel of nonhuman primates is that defects appear to occur at regular time intervals in some samples. Macho and co-workers<sup>42</sup> demonstrated that the teeth of *Theropithecus oswaldi* from Koobi Fora (1.89 and 1.5 million years ago) and *Olororgesailie* (0.7 million years ago) exhibit an annual pattern in which accentuated striae divide each year into two long intervals (132 to 138 days) and one short interval (90 to 102 days). Noting that East African environments have been seasonal for the last two million years, the authors suggest that within each year there may have been three stressful periods:

the onset of each of two rainy seasons and the height of the dry season.

Linear enamel hypoplasias also may exhibit evidence of periodicity. Skinner<sup>33</sup> studied linear hyperplasias in the dental remains of 229 sympatric chimpanzees and gorillas from the Republic of Cameroon. He found multiple, regularly spaced linear hyperplasia grooves on mandibular canine

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teeth. Skinner, Dupras, and Mota-Sola<sup>34</sup> also found multiple, regularly spaced defects in *Dryopithecus crusafonti* and *D. laietanus* from Middle to Late Miocene sites in northeastern Spain. These investigators estimated the time intervals between hypoplastic grooves by calculating the ratio of the space between the grooves to the overall crown height, then multiply-

ing by an average crown formation time of 4.9 years. Using this method, they found that the average space between grooves represented 6.7 months worth of growth, with a standard deviation of 2.7 months. This approximately six-month periodicity coincides with the pattern of semi-annual rainy seasons in the Cameroon. Skinner<sup>33</sup> suggested that during rainy seasons animals might be more prone to respiratory illnesses. Skinner additionally hypothesized that the regular patterning in both modern and fossil great apes might be the result of semi-annual occurrences of malaria<sup>34</sup> or other parasitic infections<sup>25</sup> influenced by seasonality in rainfall.

At the time when the aforementioned two studies were conducted, much less was known about crown formation in the great apes than is known at present. For example, Reid, Hillson, and Dean,<sup>54</sup> have recently shown that the imbricational enamel in male chimpanzee canines takes an average of 6.34 years to form and that enamel formation does not proceed at a uniform rate down the sides of the crown. For this reason, researchers, including Skinner,<sup>62</sup> have begun to count perikymata between defects to arrive at a more accurate and precise method of determining time intervals represented by successive linear enamel hyperplasias. By doing so, Skinner<sup>62</sup> has found a "clear semi-annual pattern" (p. 283) in the intervals between linear hyperplasias in specimens of orangutans and bonobos. However, the consistency of this pattern in large samples of great apes, and the extent of variation about the

mean interval between hyperplasias, have yet to be determined.

Studies focusing on the causes, timing, and periodicity of developmental enamel defects therefore suggest that nonhuman primates experience metabolic disruption in captive situations in conjunction with surgical procedures and psychosocial disturbances. Both nutritional deficiency and the weaning process appear to cause disruptions in primate enamel formation. The patterning of defects in modern and fossil primates suggests that primates experience metabolic disruption with seasonal patterns of rainfall and drought. Studies by Dirks and coworkers<sup>41</sup> and by Kelley and Bulicek<sup>27</sup> indicate that individuals sharing environments and aspects of life history may also form enamel defects at similar or even identical ages. Taken together, these studies suggest that there are similarities in the kinds of stresses primates experience and in the patterns of timing of these stresses. However, more studies like these will be required to discover how consistent and widespread these patterns are.

#### HOW DO DEVELOPMENTAL DEFECTS OF ENAMEL VARY ACROSS AND WITHIN PRIMATE TAXA?

The second major focus of research on developmental defects in the enamel of nonhuman primates has been documenting taxonomic variation in defect prevalence. This is an important goal because description of this variation provides a context for comparison and interpretation. For example, a 40% frequency of linear enamel hyperplasias would be high for a sample of lemurs, but low for a sample of chimpanzees. Such a relatively high frequency in a lemur sample would suggest something unusual about the nature of individuals' stress experience during their tooth crown formation periods. Several apparent patterns in the prevalence of developmental defects of enamel both across and within primate taxa are emerging from recent studies.

Schuman and Sognaes<sup>44</sup> 1956 study remains the only large-scale comparative study of taxonomic vari-

ation in the presence of accentuated striae in nonhuman primates. These researchers reported that a majority of chimpanzee and some orangutan first and third molars had accentuated striae of Retzius, gorilla and gibbon molars had few, and rhesus monkey molars had hardly any.

Many researchers have found that extant great apes and large-bodied Miocene hominoids exhibit higher frequencies of enamel hypoplasia than do cercopithecoids and ceboids.<sup>30,34,37,38,44</sup> The prevalence of linear enamel hypoplasia tends to be very low in prosimians<sup>23,32</sup> and to progressively increase from monkey, to gibbon, to great ape grades<sup>22,23,32</sup> (Table 1, Fig. 4). This is consistent with the trend reported by Schuman and Sognaes<sup>44</sup> for accentuated striae. These results suggest that developmental timing influences the distribution of defects across the primate order, although several other factors may be involved.

Several recent studies have focused on variation in linear hyperplasia among great apes, finding that chimpanzees and orangutans usually have higher frequencies than gorillas do (Table 2). The studies summarized in Table 2 are ones in which a single observer has studied samples of two or more great ape species. Thus, frequencies of linear enamel hyperplasia in different species samples are not compared across the data sets of different observers, which would introduce inter-observer error, but within each observer's data set. As shown in the table, these studies, with the exception of one,<sup>33</sup> indicate that chimpanzee and orangutan samples have frequencies of linear hyperplasia that are higher than those of gorilla samples.

Lukacs<sup>28</sup> published the first study of localized hypoplasia of the primary canine in great apes, finding significantly higher frequencies of this defect in orangutans (88%) and gorillas (88.7%) than in chimpanzees (22%). It is not surprising that the distributional pattern of such hypoplasia in great ape species appears to differ from that for linear hyperplasia given the fact that these two types of enamel defects may have different causes (Box 2) and are formed at different times. Summarizing results from sev-

eral studies, Lukacs<sup>28</sup> notes that chimpanzee deciduous canines begin forming during the fifth fetal month and are completed by the fifth month of postnatal development. In contrast, chimpanzee permanent canine crown formation begins at about 0.5 years and is usually complete between 6 and 7 years of age.<sup>63</sup>

While these studies have demonstrated between-species in linear enamel hyperplasia and localized hyperplasia of the primary canine differences among great apes, most have not adequately addressed enamel hypoplasia variation within great ape species. Hannibal,<sup>26</sup> however, demonstrated a significant difference in the prevalence of linear hyperplasia in samples of mountain and lowland gorillas, with the former having much lower frequencies (Table 2). Hannibal found little difference in the frequencies of linear hyperplasia in orangutan subspecies. (Table 2). Interestingly, Tsukamoto<sup>64</sup> recently reported a 98% prevalence of linear enamel hyperplasia in a sample of bonobos. The significance of this figure is not yet clear because this study did not include frequencies of linear hyperplasia in other great ape species, which would have served as standards for comparison.

In sum, developmental defects of enamel exhibit the following taxonomic patterns: They increase in frequency from prosimian to monkey to ape grades and appear to be more frequent in chimpanzees and orangutans than they are in gorillas, especially mountain gorillas, and gibbons. Understanding these patterns is important because they provide a context for identifying atypical prevalence rates. Because monkeys generally exhibit little linear enamel hypoplasia, high prevalence rates may signify unusual circumstances. For example, a high frequency of linear enamel hyperplasia (54%) was reported for a sample of 24 Preuss's red colobus from Cameroon.<sup>25</sup> Lee, Thornback, and Bennet<sup>65</sup> list this subspecies as endangered. The finding of an elevated frequency of linear hyperplasia may be related to the habitat destruction and population decline of red colobus in this area, although detailed health and demographic information

on the source population would be necessary to examine this possibility fully.

### WHY DO DEVELOPMENTAL DEFECTS OF ENAMEL VARY ACROSS THE PRIMATE ORDER?

The question that remains to be answered is why developmental defects of enamel appear to have the patterns of distribution I have described. Do taxonomic differences in the prevalence of these defects reflect real differences in stress experience or are they simply consequences of taxonomic differences in enamel development and morphology? There has been much speculation about the causes of this taxonomic variation but little by way of research explicitly designed to elucidate this issue. The explanations offered for these taxonomic patterns include the possible influences of both intrinsic and environmental factors on taxonomic variation in linear enamel hyperplasia. Potentially important intrinsic factors include variation in crown size<sup>2</sup> or crown height,<sup>30</sup> the visibility and spacing of perikymata<sup>22</sup> (based on Hillson and Bond<sup>40</sup>), the length of crown formation periods,<sup>34</sup> and overall maturation time.<sup>32</sup> Among the environmental factors considered are dietary differences between monkeys and apes, as well as among great ape species.<sup>22,23,25,26,36</sup>

It has been suggested that differences in crown size or height between great apes and monkeys may be related to the differences in frequency of linear hyperplasia.<sup>2,30</sup> Larger teeth may contain ameloblasts that function over a long period of crown formation and perhaps are more sensitive to disruption as they become increasingly fatigued.<sup>2</sup> However, larger crown size does not necessarily imply longer periods of ameloblast function: The relevant variables in terms of ameloblast life span are the rate of enamel secretion and the thickness of the enamel an ameloblast must secrete. No influence of enamel thickness on the taxonomic distribution of linear enamel hyperplasia is currently clear because there is taxonomic overlap in primate enamel thickness. Shellis and co-workers<sup>66</sup> have shown that *Daubentonia*, *Cebus apella*, *Thero-*

*pithecus*, and *Homo* have similar enamel thickness relative to body weight. Despite this overlap, a potential relationship among the variables of enamel secretion rate, enamel thickness, and linear hyperplasia might be a worthwhile focus for future studies.

Another potential intrinsic factor is that the visibility and spacing of perikymata may affect the visibility of linear hyperplasias. Perikymata are easier to see and appear to be more widely spaced on the anterior teeth of great apes than they are on the anterior teeth of monkeys. These observa-

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**Do taxonomic differences in the prevalence of these defects reflect real differences in stress experience or are they simply consequences of taxonomic differences in enamel development and morphology? There has been much speculation about the causes of this taxonomic variation but little by way of research explicitly designed to elucidate this issue.**

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tions may mean that linear hyperplasias representing brief episodes of stress involving few perikymata will be more easily observed in great apes than in monkeys. Yet this explanation, which only addresses differences between monkeys and great apes in the number of surface defects observed, cannot account for Schuman and Sognaes'<sup>44</sup> observation that accentuated striae are more common in the molars of great apes than they are in the molars of monkeys. This raises the question of whether visibility issues

affected Schuman and Sognaes' results. This does seem possible, given that microscopic techniques are more advanced now than they were when these researchers published their study in 1956.

Skinner, Dupras, and Moya-Sola<sup>34</sup> noted that monkeys, in contrast to great apes, rarely show multiple defects on their teeth, a difference they attributed to the short period of crown formation in monkeys relative to that in great apes. Great ape mandibular canines form over several years and have a greater opportunity to record multiple episodes of stress. At present, the data suggest a minor influence of crown formation time on the number of stress events recorded. On the mandibular canines of gibbons, which have shorter crown formation spans than do the corresponding teeth in great apes,<sup>67</sup> fewer episodes of stress are recorded than are seen in the great apes. Specifically, a maximum of four episodes were observed in the gibbons, as opposed to eight in great apes.<sup>22</sup> However, some monkey species, such as *T. gelada*<sup>68</sup> and *M. nemestrina*<sup>69</sup> have mandibular canine crown formation times comparable to or exceeding those of gibbons, and yet these monkey species rarely exhibit multiple defects. Thus, factors other than the duration of crown formation must also influence differences in the expression of linear enamel hyperplasias across taxa.

Maturation length may also influence taxonomic patterns in the distribution of developmental enamel defects. Newell<sup>32</sup> found that in twenty nonhuman primate species, age at M1 eruption was highly correlated with the frequency of linear enamel hyperplasia ( $r = 0.87$ ) and explained 74.3% of the taxonomic variation in such hyperplasia. Newell<sup>32</sup> also found that other variables associated with maturation length, specifically adult brain mass and neonate body mass, are highly correlated with frequencies of linear hyperplasia across taxa. Newell<sup>32</sup> argued that the longer the period of immaturity, the more vulnerable a primate may be to nutritional stress. Recently weaned juvenile primates, especially during periods of food scarcity, may be more susceptible to malnutrition than adults are.<sup>70,71</sup> Enamel

TABLE 1. Prevalence of Linear Enamel Hyperplasia in Samples of Monkeys, Gibbons, and Great Apes

Species	Collection	Provenience	No. of Specimens	M/F/?	No. of Individuals With Matched Hyperplasias on a Pair of Antimeres (out of N)	Percentage of Individuals With Hyperplasia
<i>Cebus albifrons</i>	MVZ <sup>1</sup> LACMNH <sup>2</sup>	Columbia; Peru	17	11/4/2	6 (17)	35
<i>Alouatta caraya</i>	UO <sup>3</sup>	Argentina	12	8/4/0	0 (12)	0
<i>Saimiri sciureus</i>	MVZ <sup>1</sup> LACMNH <sup>2</sup>	Brazil; Columbia; Peru	14	13/1/0	2 (14)	14
<i>Presbytis cristata</i>	MCZ <sup>4</sup>	Borneo	23	11/12/0	2 (23)	9
<i>Nasalis larvatus</i>	MCZ <sup>4</sup>	Borneo	18	8/9/1	2 (18)	11
<i>Macaca fascicularis</i>	UO <sup>3</sup> MCZ <sup>4</sup>	Borneo; Celebes Island	60	18/39/3	8 (60)	13
<i>Macaca mulatta</i>	CPRC <sup>5</sup>	Cayo Santiago	360	179/181/0	61 (360)	17
<i>Papio anubis</i>	MVZ <sup>1</sup>	Niger Park; Kenya	18	10/8/0	3 (18)	17
<i>Hylobates lar</i>	MCZ <sup>4</sup>	Thailand	92	45/47	33 (92)	36
<i>Pongo pygmaeus</i>	MCZ <sup>4</sup>	Borneo; Sumatra	14	6/7/1	11 (14)	79
<i>Gorilla gorilla</i>	MCZ <sup>4</sup>	Cameroon	23	12/11/0	9 (23)	39
<i>Pan troglodytes</i>	LACMNH <sup>2</sup> MCZ <sup>4</sup>	West Africa; Uganda	26	8/7/11	22 (26)	85

<sup>1</sup> MVZ = Museum of Vertebrate Zoology (at UC Berkeley)

<sup>2</sup> LACMNH = Los Angeles County Museum of Natural History

<sup>3</sup> UO = University of Oregon (primate collection of the Anthropology Department)

<sup>4</sup> MCZ = Museum of Comparative Zoology (at Harvard University)

<sup>5</sup> CPRC = Caribbean Primate Research Center Museum

formed during juvenile periods might therefore be especially prone to disruption, so that a long period of immaturity coupled with an extended period of crown formation may provide conditions conducive to defect formation.

Finally, it is possible that environmental factors, especially those affecting physiological stress experience, may contribute to taxonomic patterns in the expression of linear hyperplasia. There is some evidence that great apes may be more prone to nutritional stress than monkeys are. Knott<sup>72</sup> has shown that orangutans experience nutritional stress, especially following “mast” fruiting events, when trees fruit in synchrony. Knott<sup>72</sup> found ketones in orangutan urine, indicating that orangutans were catabolizing fat stores. While such “mast” fruiting events are supra-annual, there is also annual variation in fruit availability, compelling orangutans to augment their diets with bark and pith during fruit-poor times.<sup>73</sup> Macaques that live

sympatrically with orangutans do not appear to experience seasonal nutritional fluctuations. *M. fascicularis* inhabits stream-side areas where trees are continuously distributed in space, providing abundant and rapidly renewing food supplies.<sup>74</sup> *M. nemestrina* has a diverse diet that includes fruits, seeds, young leaves, leaf stems, fungi, insects, spiders, termites and grasshoppers.<sup>75</sup> An analysis of the prevalence of linear hyperplasia in 97 specimens collected in the 1930s at what was then known as Camp Abai along the Kinabatangan River in Borneo revealed that orangutans were much more likely to have matching defects (on a pair of antimeres) than were sympatric gibbons, macaques, or colobines.<sup>23,25</sup>

There is evidence that chimpanzees also occasionally experience nutritional stress. Growing chimpanzees from Gabon do not gain weight from February to June during seasonal low productivity.<sup>76</sup> Wrangham and colleagues<sup>77</sup> found that chimpanzees in

Kibale National Forest, Uganda, experience seasonality in the availability of ripe fruit, relying on piths as their “fallback” food when ripe fruit is scarce. Blue monkeys, redtail monkeys, and grey-cheeked mangabeys, on the other hand, turn to unripe fruits and seeds, maintaining a “diverse” diets at all times. In a taxonomically mixed group of 115 West African primates, chimpanzees and gorillas were found to have much higher prevalence of linear hyperplasia than monkeys, with the exception of *Colobus badius*.<sup>25</sup>

Seasonal variation in fruit availability might explain differences between great apes and monkeys with respect to linear enamel hyperplasia. However, that explanation does not seem to account for the lower prevalence of linear hyperplasia in lowland gorillas relative to chimpanzees, for their diets are similar.<sup>78</sup> During wet seasons, lowland gorillas, like chimpanzees, include a large component of fruit in their diets, while in dry seasons they

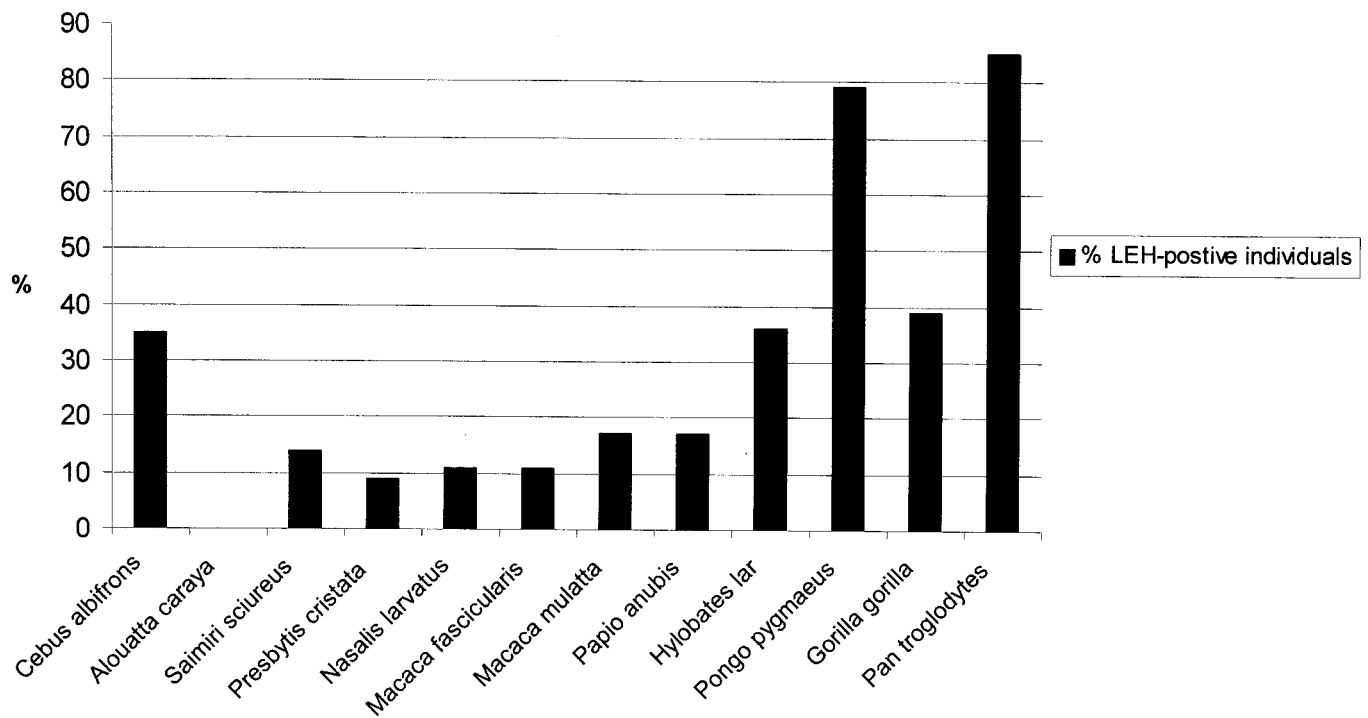


Figure 4. Histogram of frequencies (given in Table 1) of LEH-affected individuals.

TABLE 2. Prevalence of Linear Enamel Hyperplasia in Samples of Great Apes

Study	Species or Subspecies	Number of Specimens	Number of Individuals With Hyperplasia	Percentage of Individuals With Hyperplasia	Method of Determining Hyperplasia
Guatelli-Steinberg (2000) <sup>22</sup>	<i>Pongo pygmaeus</i>	14	11	79	If matched defects present on antimeric pair
	<i>Pan troglodytes</i>	26	22	85	
	<i>Gorilla gorilla gorilla</i>	23	9	39	
Hannibal (2000) <sup>26</sup>	<i>Pongo pygmaeus abelli</i>	13	11	84.6	If matched defects present on antimeric pair
	<i>Pongo pygmaeus pygmaeus</i>	57	47	82.5	
	<i>Pan troglodytes</i>	25	17	68.0	
	<i>Gorilla gorilla berengei</i>	18	1	5.3	
Newell (1998) <sup>32</sup>	<i>Gorilla gorilla gorilla</i>	22	11	50.0	Individuals with hyperplasia on any tooth
	<i>Pongo pygmaeus</i>	48	30	62.5	
	<i>Pan troglodytes</i>	79	41	51.9	
Skinner (1986) <sup>33</sup>	<i>Gorilla gorilla</i> (primarily <i>Gorilla gorilla gorilla</i> )	146	48	32.9	Individuals with hyperplasia on any tooth
	<i>Pan troglodytes</i>	110	64	58	
Stottlemire (1998) <sup>36</sup>	<i>Gorilla gorilla</i>	119	90	76	Individuals with hyperplasia on any tooth
	<i>Pan troglodytes</i>	98	79	80.6	
	<i>Gorilla gorilla</i>	229	63	27.5	

fall back on piths and bark.<sup>78</sup> It is possible, though, that lowland gorillas are somewhat better buffered than chimpanzees during the period of reliance on fall-back foods. As previously mentioned, Hannibal<sup>25</sup> found a significantly higher frequency of linear hyperplasia in a sample of lowland gorillas relative to a sample of moun-

tain gorillas. This difference may imply that mountain gorillas, which have a more folivorous diet,<sup>78</sup> are less likely to experience the nutritional stress that large-bodied frugivores do.

Of the explanations for the differences between monkeys and great apes that have been offered, two seem

to have the most merit in terms of plausibility and in accounting for differences in both surface hypoplasias and accentuated striae. The first of these is that the higher prevalence of developmental enamel defects in great apes results from the vulnerability of a long period of immaturity in conjunction with long periods of crown for-

mation during which metabolic disruptions can be recorded. The second is that the greater overall dietary flexibility of monkeys as compared to great apes may enable monkeys to maintain sufficient caloric intake during periods of low fruit availability when great apes, particularly the most frugivorous ones, cannot. These two explanations are not mutually exclusive. However, there is not sufficient evidence at this time to rule out other possibilities. The visibility of perikymata, enamel thickness, and enamel secretion rates may also be important influences. Comparative data on perikymata visibility and spacing in nonhuman primates is needed, as is confirmation or refutation of Schumann and Sognaes' observations on the prevalence of accentuated striae. Furthermore, it is possible that there are other relevant intrinsic aspects of enamel that have not yet been considered. One possibility, for example, is that there could be taxonomic variation in the sensitivity of ameloblasts to disruption. Much more work is clearly required before the causes of these patterns across and within primate taxa can be fully understood.

#### WHAT FURTHER INSIGHTS MAY DEVELOPMENTAL DEFECTS OF ENAMEL PROVIDE?

The greatest promise of developmental defects of enamel as tools for understanding physiological stress in nonhuman primates is in their use in elucidating the timing and frequency of stress episodes. Accentuated striae of Retzius are particularly informative as indicators of metabolic disruption for which ages at defect formation can be determined with precision and accuracy. Studies by Schwartz and colleagues and Bowman, in which known events in the lives of captive primates have been linked with accentuated striae, provide evidence that such striae are sensitive indicators of disruption. Examination of accentuated striae in the teeth of wild primates therefore has the potential to reveal a great deal about when, how often, and under what conditions primates experience metabolic disruption. It might be possible, for example, to determine if seasonal fluctuations

in fruit availability are reflected in the pattern of accentuated striae or if the most subordinate primates in a group have the most accentuated striae.

The ability to derive information about physiological stress from teeth is especially useful for studies of fossil primates. When fossilized teeth can be sectioned, the timing of stress episodes can be inferred from the record of accentuated striae. When teeth cannot be sectioned, linear enamel hypoplasias can provide information about the timing of stress episodes provided that perikymata have not been worn away from tooth surfaces.

While developmental enamel defects appear to increase in frequency from prosimian to monkey to ape grades, the meaning of this pattern is not currently clear. To understand the significance of this broad taxonomic pattern, answers to two questions are essential. First, to what extent are prevalence rates affected by intrinsic factors such as taxonomic variation in the visibility and spacing of perikymata, enamel thickness, and enamel secretion rates? Second, do different grades of primates vary in their experience of physiological stress in ways that could be reflected in their enamel? The answer to the first question could be investigated by a correlation study of these variables and the distribution of linear hyperplasia across the primate order. However, answering the second question would require not only comprehensive and extensive data on the health status of free-ranging primates but also empirical knowledge of the ways in which different physiological stresses and varying ages at death interact to produce frequencies of developmental enamel defects in given samples.

It is not likely, therefore, that the significance of the broad taxonomic pattern described here will be understood in the near future. However, because prosimians generally display fewer developmental defects in their enamel than monkeys do, and monkeys generally exhibit fewer such defects than apes do, a departure from this pattern can serve as red flag for population stress, as it might in the case of Preuss's red colobus from Cameroon.<sup>25</sup>

Enamel hypoplasias and accentu-

ated striae of Retzius are unique in that they provide permanent records of metabolic disruption during enamel formation. As such, they have long been a useful tool in health assessments of ancient as well as living humans.<sup>2</sup> The use of developmental defects of enamel to understand the nature, timing, and frequency of physiological stresses in nonhuman primates has, however, only recently begun to attract attention. Researchers are discovering the variety of environmental and social factors that can cause disruption and are ascertaining the timing of these disruptions in the lives of young primates. Continued work in this area of research promises to further our understanding of the stresses that affect nonhuman primates, and when and how often they experience them.

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