

Macroscopic and Microscopic Analyses of Linear Enamel Hypoplasia in Plio-Pleistocene South African Hominins With Respect to Aspects of Enamel Development and Morphology

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ABSTRACT This study uses macroscopic and microscopic methods to analyze the expression of linear enamel hypoplasia (LEH) in Plio-Pleistocene South African hominins. LEH is a developmental defect of enamel that is used in many anthropological contexts as a physiological stress indicator. Previous research has not settled the question as to whether differences in LEH expression exist between *Paranthropus* and *Australopithecus* and if they exist, to what extent these differences might be explained simply by taxonomic differences in enamel development and morphology rather than by differential stress experience. In this study, the analysis of LEH is conducted with respect to differences between *Paranthropus* and *Australopithecus* in aspects of enamel development and morphology that are thought to influence LEH expression. Two factors impacting LEH expression are considered: the duration of enamel formation, and the spacing of perikymata. It is predicted that if the first factor strongly influences the expression of LEH, then there should be fewer defects per tooth in *Paranthropus* because of its abbreviated crown formation spans (and fast extension

rates) relative to *Australopithecus*. It is also predicted that because *Australopithecus* has more densely packed perikymata in comparable regions of the crown than *Paranthropus*, this taxon should, on average, have narrower defects than *Paranthropus*. To address these questions, 200 *Australopithecus* and 137 *Paranthropus* teeth were examined for LEH, and the analysis of defect width with respect to perikymata spacing was conducted on tooth impressions examined under a scanning electron microscope using INCA (Oxford Instruments) measurement software. Data support the first prediction: *Australopithecus* does have significantly more defects per canine tooth than *Paranthropus*. Data do not support the second prediction in large part because several *Australopithecus* specimens have wide groove defects in which perikymata are not visible and enamel is irregular. Such wide grooves are not predicted by perikymata spacing such that alternative explanations, including taxonomic differences in ameloblast sensitivity and the duration/severity of disruptions to enamel growth, must be considered. *Am J Phys Anthropol* 120:309–322, 2003. © 2003 Wiley-Liss, Inc.

Linear enamel hypoplasia (LEH) is a developmental defect of enamel appearing as one or more horizontal lines or grooves on the surface of a tooth crown (Goodman and Rose, 1990, 1991). These defects form when systemic physiological stresses, usually episodes of febrile disease or periods of malnutrition, disrupt the activity of ameloblasts (enamel-producing cells) as they secrete the enamel matrix (Skinner and Goodman, 1992). Because enamel does not remodel, linear enamel hypoplasias provide an indelible record of systemic physiological stresses that have occurred during an individual's enamel formation period. LEH, however, can only be used as a nonspecific indicator of such stresses because the precise cause(s) of a defect cannot be ascertained by examination of its physical attributes (Skinner and Goodman, 1992).

Anthropological interest in LEH has traditionally concentrated on recent humans. Because nutritional and disease stress are common causes of these defects (Goodman and Rose, 1990; Jontell and Linde,

1986; May et al., 1993; Skinner and Goodman, 1992) and because they can be easily observed on the surfaces of teeth, their prevalence is often used as an indicator of human population health status (Goodman and Rose, 1990). Bioarchaeological applications have involved documenting an increase in LEH prevalence in the transition from hunting/gathering to agricultural subsistence systems in various re-

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gions of the globe (Cohen and Armelagos, 1984; Goodman et al., 1980; Lukacs, 1992) and reporting high LEH prevalence in slave populations (Blakey et al., 1994; Corruccini et al., 1982). Over the last 15 years, interest in enamel hypoplasia has extended to the nonhuman primates, and is helping to illuminate the timing and frequency of stress episodes primates experience (Eckhardt, 1992; Eckhardt and Protsch von Zeiten, 1993; Guatelli-Steinberg, 2000, 2001; Guatelli-Steinberg and Skinner, 2000; Hannibal, 2000; Kelley and Bulicek, 2000; Lukacs, 1999, 2001; Moggi-Cecchi and Crovella, 1991, 1992; Newell, 1998; Skinner, 1986; Skinner et al., 1995; Vitzthum and Wikander, 1988).

Whereas LEH is extensively used as a stress indicator in recent humans and is beginning to shed light on stress episodes in primate lives, it has received relatively little attention in studies of hominin teeth. This situation is surprising, given the usefulness of studying LEH in recent human and nonhuman primates. Individual cases of enamel hypoplasia have been described in Plio-Pleistocene hominins (e.g., Tobias 1991; Robinson, 1956); however, White (1978), Bombin (1990), and Moggi-Cecchi (2000) remain the only publications to date to systematically compare defect prevalence among early hominin samples from different species and localities.

Work by White (1978) on South African Plio-Pleistocene hominins encompassed several different types of enamel hypoplasia, including pitting and discoloration as well as linear defects. He found that the most common of these defects was pitting, and that the Swartkrans dental sample had a higher incidence of pitting (12.1%) than the Sterkfontein sample (8.4%). On the other hand, using an expanded sample that included more recently excavated Sterkfontein specimens unavailable to White (1978), Moggi-Cecchi (2000) found no difference between South African *Australopithecus* and *Paranthropus* in the total number of teeth affected by enamel hypoplasia. Because both White (1978) and Moggi-Cecchi (2000) combined several types of hypoplasias, their data do not address the issue of potential differences in linear enamel hypoplasia prevalence specifically. The various types of enamel hypoplasias (e.g., hypoplastic pits, furrows, or planes) result from differences in the manner in which disruptions affect groups of ameloblasts (Hillson and Bond, 1997), and may also have different etiologies (Seow, 1992; Skinner et al., 1994; Alve-salo, 1997), such that LEH cannot be assumed to follow the same pattern of distribution as composite hypoplasia prevalence. Bombin (1990), however, focused on linear enamel hypoplasias, specifically on permanent canines, finding an LEH frequency of 89.7% in gracile canines from Sterkfontein (*Homo* plus *Australopithecus*) and Makapansgat and a "negligible presence" of LEH in the canines of robust specimens from Swartkrans and Kromdrai.

Previous research, therefore, has not settled the question of whether there are differences in LEH prevalence and expression between South African *Paranthropus* and *Australopithecus*, and to what extent these differences, if they exist, are related to species differences in enamel development and morphology, sample biases, and/or stress experience. Blakey and Armelagos (1985), Ensor and Irish (1995), and Hutchinson and Larsen (1988) considered the relationship between the dimensions of a defect and the underlying stress that produced it. While these researchers made important strides, the use of LEH as a stress indicator can be further refined by analyzing how intrinsic aspects of enamel development and morphology affect the manifestation of defects. The purpose of this study is therefore twofold. The first objective is to use macroscopic as well as microscopic methods to determine if and in what ways South African *Paranthropus* and *Australopithecus* differ in LEH expression. The second objective is to analyze any observed LEH differences between these two genera with respect to attributes of enamel development and morphology that can affect defect expression.

One intrinsic attribute of enamel relevant to LEH expression is the duration of enamel formation. There is a progressive increase in LEH prevalence from prosimian to monkey to great ape grades that appears to be at least partly the result of progressive increases in the duration of enamel formation across these different grades (Guatelli-Steinberg, 2001; Newell, 1998; Skinner et al., 1995). This relationship between LEH and the length of enamel formation periods in nonhuman primates implies that developmental differences in enamel across taxa can affect the expression of LEH. All else being equal, because *Paranthropus* teeth take a shorter period of time to form than *Australopithecus* teeth (Beynon and Dean, 1988; Bromage and Dean, 1985; Dean and Reid, 2001), South African *Paranthropus* could be expected to have a lower prevalence of LEH and a smaller number of defects per tooth than South African *Australopithecus*.

It must be noted that the absolutely shorter period of crown formation in robust australopith teeth is partly accomplished by a fast enamel extension rate, the rate at which new cervical ameloblasts differentiate (Beynon and Dean, 1988). This faster extension rate means that relative to the teeth of *Australopithecus*, fewer perikymata (tiny wave-like growth increments) appear on the enamel surfaces of robust australopith teeth. Because linear enamel hypoplasias can only form in the region of the crown (the imbricational zone) where perikymata are present (Hillson and Bond, 1997), the rapid extension rate of robust hominin teeth itself suggests fewer potential defects. Rapid extension rates characterize fast-forming modern human deciduous teeth that rarely exhibit enamel hypoplasias (Goodman and Rose, 1990), perhaps in part because of their short crown formation times and fast extension rates (although

they may also be buffered against physiological disturbances when they are forming in utero).

Besides developmental timing, perikymata spacing is another intrinsic attribute of enamel that can affect LEH expression. Perikymata are surface manifestations of underlying striae of Retzius, dark lines visible in longitudinal crown sections that have a periodicity of 8–9 days in hominins (Dean and Reid, 2001). The spacing of perikymata has been hypothesized to affect the dimensions of hypoplasias in different tooth classes and in different parts of the crown: where perikymata are more widely spaced, hypoplastic “furrows” are also expected to be wider (Hillson and Bond, 1997). In equivalent divisions of crown height, perikymata are more widely spaced in robust hominins (as a result of rapid extension rates) than they are in *Australopithecus*. Thus, if there is no average difference in the duration or severity of growth disruptions in the teeth of South African *Australopithecus* and *Paranthropus*, the average width of defects should be greater in *Paranthropus* in comparable regions of the crown. If this is not the case, then a variety of potential causes, including differences in the duration and severity of growth disruptions in the enamel of South African *Australopithecus* vs. *Paranthropus*, must be considered.

MATERIALS AND METHODS

Samples

Only permanent teeth were selected for observation, as these teeth are more likely than deciduous teeth to display LEH (Goodman and Rose, 1990). The permanent teeth included in this study were those for which an estimated 50% or more of the crown height was present and those with perikymata that were visible under a magnification of 10× over much of the enamel surface. There were several reasons for this latter requirement. If perikymata have been worn away, then small linear defects might also have been worn away. In addition, to be able to accurately identify defects, it is useful to compare them to the pattern of normal growth (the perikymata pattern) that surrounds them. Finally, measurements of perikymata spacing were required for the analysis of defect width (see below).

The sample consists of 200 *Australopithecus* teeth and 137 *Paranthropus* teeth. The *Australopithecus* sample includes specimens from Sterkfontein (Brain, 1981) and Makapansgat, and excludes specimens such as STW 53, STW 95, and STW 75 that have been attributed to *Homo* (Clarke, 1985). The robust specimens are those listed as *Australopithecus robustus* from Swartkrans and Kromdrai (Brain, 1981) and specimens from the Swartkrans extension site ascribed to *Paranthropus* (Grine, 1993). Swartkrans specimens of *Homo* as listed by Brain (1981) or Grine (1993) are of course excluded. Teeth observed in this study are housed at the Transvaal Museum in Preto-

ria and the University of Witwatersrand in Johannesburg.

Macroscopic observations and analysis

LEH was observed following methods outlined by Goodman and Rose (1990) and Lukacs (1989). Teeth were observed under conditions of diffuse lighting, with a second (incandescent) light source oriented obliquely to the specimen. A 10× hand lens aided in identifying defects. Following Skinner (1986), the threshold for identifying an LEH defect was set deliberately low, so as not to exclude information about growth disturbance. Berti and Mahaney (1995, p. 313) found substantial concordance in the intertooth distribution of defects of varying size, implying that even very small defects are “variants of the same phenomenon, LEH.” Hillson and Bond (1997, p. 98), in a similar vein, explained that microscopic furrows that can be matched across simultaneously forming teeth are “just as much indicators of disturbance to growth as . . . large defects.” As Skinner et al. (1995) suggested, perikymata adjacent to the defect were identified and compared to hypoplastic lines to help reduce the chance of falsely identifying LEH. Thus, the lower limits of defects identified in this study were lines or grooves that appeared to be larger than adjacent perikymata grooves under 10× magnification. The upper limit was prominent lines or grooves of varying depth and width that were clearly visible without magnification.

While the conclusions of Hillson and Bond (1997) and Berti and Mahaney (1995) indicate that thorough analyses of LEH require detailed examination of tooth surfaces under magnifications greater than 10×, macroscopic observations were undertaken in this study for the explicit purpose of maximizing sample sizes for assessments of prevalence and defect counts per tooth. Silicon-rubber impressions were made of hypoplastic teeth for later examination under a scanning electron microscope (SEM), specifically for the purpose of quantifying defect width in comparison to the spacing of adjacent perikymata (below).

Hillson (1996) pointed out that if a systemic rather than localized stressor has caused a hypoplastic defect on a tooth, it should be possible to identify defects on all teeth that were forming at the time of the stress episode. He therefore argued that defects that cannot be matched on all teeth forming at the same time should not be counted in assessments of LEH prevalence. In this study, because of the fragmentary nature of the fossil record, it was not always possible to match defects on antimeres, on isomeres, or across tooth classes: a single individual might be represented by only one tooth. This problem is dealt with by reporting LEH prevalence in two different ways. Using the criterion of Hillson (1996), prevalence is first reported for a subsample of individuals represented by two or more anterior teeth, i.e., teeth that are often affected by LEH (Goodman and Rose, 1990) and that have overlap-

ping crown formation spans (Beynon and Dean, 1988). Prevalence is then reported in a less conservative fashion by using the entire sample to report the frequency of LEH-affected teeth for each tooth type.

Beyond simple presence or absence, the number of defects on each tooth was recorded. To test the hypothesis that because of their longer crown formation spans *Australopithecus* teeth should have more linear enamel hypoplasias than *Paranthropus* teeth, the number of defects on canines with an estimated 80% or more of their crown heights present was compared between these two groups. There were 23 *Paranthropus* canines with an estimated crown height of 80% or more present. The average crown height of these canines is 10.35 mm, SD = 1.51. Dean and Reid (2001) gave an average crown height of 11.7 mm (uppers) and 11.2 mm (lowers) for *Paranthropus* canines with an estimated 90% or more of their crown heights present, consistent with the average of 10.35 mm for this sample of robust canines with an estimated 80% or more of crown height present. There were 26 such *Australopithecus* canines, with an average crown height of 13.08 mm (SD = 2.28), which is close to the average of 13.0 mm (lowers) and 13.5 mm (uppers) given by Dean and Reid (2001) for *Australopithecus* canines with 90% or more of their crown heights present. These researchers' values for 90% or more estimated *Australopithecus* canine crown height are close to those given here for 80% or more of estimated crown height, most likely because their sample includes *A. afarensis* specimens as well. The requirement for 80% or more of crown height to be present is a stringent one, given that most defects occurred within 15–50% of the measured crown height in both *Australopithecus* and *Paranthropus* specimens (see below).

Canines were chosen to test the "crown formation" hypothesis because they are frequently affected by LEH in this sample (below) and because *Paranthropus* canines have shorter crown formation spans and faster extension rates than *Australopithecus* canines (Dean and Reid, 2001). Although *Australopithecus* upper canines appear to take only slightly longer to form than those of *Paranthropus*, the lower canines of *Australopithecus* may take about 1 year longer to form than those of *Paranthropus* (Dean and Reid, 2001). The sample of canines examined here is evenly distributed between lower and upper canines within each taxonomic group. Comparisons of frequency differences in LEH involved either chi-square tests or Fisher's exact tests, the latter used for expected cell counts less than five (SPSS, Inc., 2000).

Macroscopic measurements and analysis

Mitutoyo needle-point digital calipers (providing measurements to 0.01 mm) were used to measure crown height and the distances of defects from the cemento-enamel junction (CEJ) in order to deter-

mine 1) if defects on the crowns of LEH-affected individuals might have formed at the same time, and 2) the position of defects relative to the overall crown height.

For the purpose of determining whether or not defects might have formed at the same time on anterior teeth, the chronology by Beynon and Dean (1988) of dental formation for *Australopithecus* and *Paranthropus* was used. Crown heights and distances from the CEJ were measured on the mesial-buccal surfaces of the canines and along the midline of the labial surfaces of incisors. Care was taken in placing the calipers in the same positions on the teeth of each class. Note that crown height and CEJ measurements are linear measurements taken between two points, and are not "true" measurements of distances along the slightly curved surfaces of teeth. Dean and Reid (2001), however, noted that hominin anterior teeth are relatively flat on their anterior surfaces, with the exception of the last 10% of crown height in the cervical region of canines.

Lastly, an analysis of defect position relative to overall crown height was necessary in order to determine if there was a significant difference in the location of defects on the crowns of the gracile and robust samples: if such a discrepancy existed, then comparison of defect widths would become more complicated because perikymata spacing varies over the height of the crown. That is, while perikymata spacing is known to be greater in robust australopiths than in gracile australopiths in equivalent fractions of crown height (Dean and Reid, 2001), perikymata spacing near the cusps of *Australopithecus* teeth is actually greater than perikymata spacing near the cervix of *Paranthropus* teeth. To accomplish this comparison between samples, canine teeth with 80% or more of their crown heights present were used. A *t*-test was then performed for *Australopithecus* vs. *Paranthropus* on the logged value of the ratio of the defect distance from the CEJ to the measured crown height.

Microscopic methods, measurements, and analysis

A scanning electron microscope study was conducted to determine the width of defects relative to adjacent perikymata spacing. Microscopic observations and measurements were made on impressions of the buccal surfaces of canine teeth exhibiting one or more linear enamel defects. A silicon-rubber impression material made by Struers, with a company-reported resolution of 0.1 μm , was employed. Because this impression material is SEM-stable, it was possible to image the impressions themselves without the added standard step (Hillson, 1992) of making an epoxy replica. The impression is then the "negative" of the actual tooth surface, in that lines or grooves on the tooth surface are actually ridges on the impression. These ridges appear as darker areas, with sloping walls (one wall lighter, reflecting more electrons, and the other in apparent shadow)

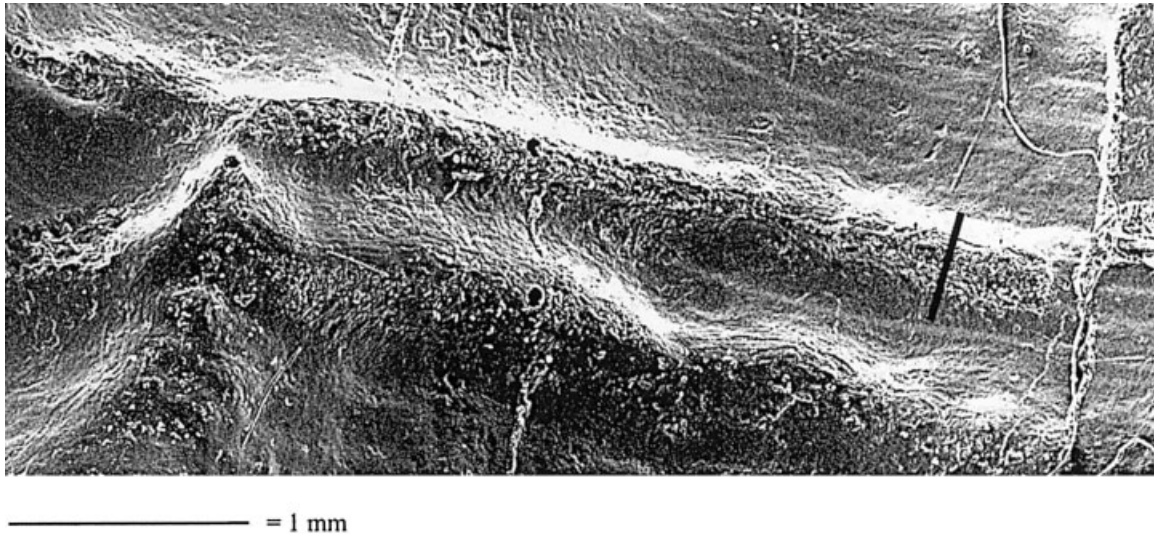


Fig. 1. LEH grooves in STW 213d (LRC) appear as “ridges” in impressions. Because defect width varies (as seen in this image), three width measurements, perpendicular to the defect, were taken along the length of each defect. Black line indicates how these width measurements were taken: from the point where the top wall of the ridge emerges from the impression surface to the point where the bottom wall of the ridge meets the impression surface. This is a montage of three micrographs taken at a magnification of $\times 50$. Note that portions of some perikymata can be seen along the sloping walls of the defect, but they are not visible in other regions of the defect.

on the SEM image relative to the surrounding surface (Fig. 1). As can be seen in Figure 1, the entire width of the defect extends from where the top wall emerges from the impression surface to where the bottom wall meets the surface.

All impressions were prepared by gold-palladium coating, and were imaged in a JEOL SEM using an accelerating voltage of 12 KeV, a working distance of 20 mm, and with a tilt of 30° . Specimens were entered into the chamber and rotated into identical orientations: with their perikymata oriented vertically on the screen, and the apex of the tooth at the left of the screen. The tilt was necessary to enhance the contrast of surface topography. INCA measurement software (Oxford, Inc.) provided electronic “calipers” which were used to measure linear distances. All measurements were made at a magnification of $50\times$. Measurements using INCA calipers assume an untilted surface such that, according to Oxford Instruments (personal communication), measurements taken at a tilt of 30° must be multiplied by the secant of 30° (1.15) to obtain the true linear distance. The author observed that in some areas of the impression where measurements were taken, the degree of foreshortening was slightly greater or less than that indicated by Oxford Instruments for a tilt of 30° . To correct for this problem, in the region of the impression where measurements were taken, distances between specific features on the impression surface were measured, first at 0° and then at 30° of tilt, and the ratio between the two measurements were used as a correction factor. Also, because the curvature varies over the surface of each specimen, these distance measurements are not actual ones, but are linear measurements between two points.

As can be noted in Figure 1, defect widths vary along their lengths. Therefore, three width measurements perpendicular to the defect were taken along the course of each defect and averaged to obtain an average defect width. The three measurements were made at what appeared to be the widest and narrowest points, and at a point that appeared intermediate in width between the two extremes. Using the electronic calipers, a line equivalent to 1 mm at 0° of tilt was drawn perpendicular to the adjacent perikymata incisal to each defect. Where perikymata were clearly visible over this distance, they were counted in these regions for the purpose of obtaining a measure of perikymata spacing prior to the formation of the defect.

While it would have been useful in every case to count perikymata within each defect and to therefore isolate the component of defect width that is caused by the duration of the growth disruption, perikymata were often not visible within the defect (e.g., as seen in Fig. 1). Where perikymata were visible within the defect, they were counted, are reported here, and are incorporated into statistical analyses; however, perikymata in the defect’s occlusal wall, which Hillson and Bond (1997) argued most closely represent the length of disruption, were difficult to identify, and so all perikymata encompassed within the defect were counted if they could be clearly observed.

A *t*-test was used to compare defect widths (logged values) between *Paranthropus* and *Australopithecus* samples. A linear regression of defect width on the number of perikymata within the defect was then performed. A linear regression was also performed of defect width on perikymata spacing (measured here as the number of perikymata per millimeter

adjacent and incisal to each defect) in order to examine the potential relationship between perikymata spacing and defect width. Lastly, a forward-stepping multiple linear regression was performed on a subset of defects for which perikymata could be counted within the defect, to ascertain which variables are the best predictors of defect width.

Intraobserver reliability

Observer reliability for macroscopic observations was assessed on a sample of hominin teeth from East Africa housed at the Kenya National Museum. Fifty teeth that were scored for the presence of 0, 1, or 2 or more defects were rescored 2 weeks later. The kappa statistic was used to analyze the results of the intraobserver reliability test. Kappa reflects the degree of agreement between observations in excess of that expected by chance (Cohen, 1960). The guidelines by Landis and Koch (1977) for the interpretation of kappa were employed. The value of kappa for these 50 repeated observations across the three categories of 0, 1, or 2 or more defects was 0.81. This value is the lower limit of the "almost perfect" category of Landis and Koch (1977) of agreement between observations (kappa values of 0.81–1.00).

To assess observer reliability for perikymata counts, a line equivalent to 1 mm at 0° of tilt was drawn and perikymata were counted for 20 cases (9 *Paranthropus*, 11 *Australopithecus*) on two separate occasions. Percent error was calculated using the method of Calcagno (1989, p. 13) in which, first, the absolute value of the difference in each measurement pair is expressed as a proportion of the first measurement. Then these values are summed, divided by the number of measurement pairs, and multiplied by 100 to give the percent error. Here, the sum of these values is 0.48, the number of measurement pairs is 20, and the percent error is 2.4%.

RESULTS

By individual

Tables 1 and 2 list specimens for which two or more anterior teeth were present in *Paranthropus* and *Australopithecus* samples, respectively. To be included in Tables 1 and 2, anterior teeth were required to have 50% or greater of their crown heights present and perikymata visible over most of their surfaces. Incisors and canines begin forming at about the same time in early hominins (Beynon and Dean, 1988), but their canines may take from one-half (Beynon and Dean, 1988) to 1 year longer (Dean and Reid, 2001) to complete their formation. Roughly, then, only defects in the cervical region of the canine may not be matched by defects in incisors. Specimen numbers, teeth, crown heights, numbers of defects present, and defect distances from the CEJ are recorded for each tooth.

In the *Australopithecus* sample, 13 individuals are represented, of which 4 (MLD 18, STW 213, STW 498, and TM 1512) can be considered to have

TABLE 1. *Australopithecus* sample of individuals with two or more anterior teeth meeting scoring criteria

Specimen	Tooth	Crown height (mm)	Defect count	Distance from CEJ (mm)
STS 24	LLI1	12.49	0	
STS 24	LRI1	12.56	0	
STS 24	LLI2	11.50	0	
MLD 18	LLI2	5.26	1	2.98
MLD 18	LRI2	5.29	1	2.85
MLD 11	URI2	10.16	1	5.46
MLD 11	ULC	14.12	0	
STW 151	ULI1	13.83	0	
STW 151	URI1	13.07	0	
STW 151	ULI2	9.57	0	
STW 151	URI2	9.20	0	
STW 151	ULC	13.75	0	
STW 151	LLI1	10.81	0	
STW 151	LRI1	10.48	0	
STW 151	LLI2	11.62	0	
STW 151	LRI2	11.54	0	
STW 151	LLC	12.12	0	
STW 151	LRC	12.06	0	
STW 213	LLC	12.82	1	3.03
STW 213	LRC	11.93	2	3.15, 3.77
STW 537	LI	12.17	0	
STW 537	LI	12.36	0	
STW 537	LRC	13.84	0	
STW 537	LLC	14.15	0	
STW 491	LLI1	8.00	0	
STW 491	LRC	13.90	0	
STW 498	ULI1	11.12	1	3.96
STW 498	URI1	11.60	1	4.40
STW 498	ULI2	8.84	1	3.71
STW 498	ULC	14.66	3	2.72, 6.74, 7.25
STW 498	URC	13.20	4	3.10, 4.50, 6.56, 7.49
STW 498	LLI1	8.18	1	2.97
STW 498	LRI1	8.95	1	3.42
STW 498	LLC	13.85	3	3.12, 6.52, 8.11
STW 116	LLI1	11.84	0	
STW 116	LRI1	13.31	0	
STW 116	LRI2	10.27	0	
STW 116	LRC	14.44	1	2.31
STW 252	ULI1	10.60	0	
STW 252	ULI2	11.24	0	
STW 252	URI2	11.06	0	
STW 252	ULC	14.79	0	
STW 252	URC	14.52	0	
STW 332	LLI1	11.59	0	
STW 332	LRI2	13.18	0	
TM 1512	URI2	8.19	2	2.68, 4.46
TM 1512	URC	8.69	2	1.35, 4.62

"matching" defects in that defects appear at similar distances from the CEJ in antimeres and in similar regions of the crown in incisors and canines. However, STW 116 has defects in the cervical region of its canines that may have formed after incisor crown formation was completed. Thus, the frequency of individuals with matching defects is 4/12 (33.3%), or 4/11 (36.4%) if STW 116 is omitted. In the *Paranthropus* sample, 10 individuals are represented, of which one, SK 65, can be considered to have matching defects. SK 23 appears to have matching defects on its lower lateral incisors and canines, but none were observed on the lower central incisors. Thus, the frequency of individuals with matching defects is 1/10 (10%) or 2/10 (20%), depending on whether SK 23 is counted.

With such small sample sizes and uncertainties with respect to exact crown formation times, the anal-

TABLE 2. *Paranthropus* sample of individuals with two or more anterior teeth meeting scoring criteria

Specimen	Tooth	Crown height (mm)	Defect count	Distance from CEJ (mm)
KB 5223	LLI1	Not measured ¹	0	
KB 5223	LRI1	Not measured ¹	0	
KB 5223	LLI2	Not measured ¹	0	
KB 5223	LRI2	Not measured ¹	0	
KB 5223	LLC	Not measured ¹	0	
SK 23	LLI1	7.53	0	
SK 23	LRI1	7.40	0	
SK 23	LLI2	8.91	1	1.90
SK 23	LRI2	7.82	1	2.30
SK 23	LLC	9.66	1	2.83
SK 23	LRC	9.17	1	2.89
SK 34	LRI1	8.61	0	
SK 34	LRI2	7.34	0	
SK 52	ULI2	7.98	0	
SK 52	URI2	8.95	0	
SK 55	ULI1	9.62	0	
SK 55	URI1	9.36	0	
SK 55	ULI2	7.94	0	
SK 55	URI2	7.99	0	
SK 55	ULC	11.57	0	
SK 55	URC	11.33	0	
SK 93	ULC	12.42	0	
SK 93	URC	12.49	0	
SKW 8	ULC	6.10	0	
SKW 8	URC	5.85	0	
SK 585	LLI1	7.07	0	
SK 585	LRI1	7.58	0	
SK 585	LRI2	8.86	0	
SK 585	LRC	8.78	0	
SK 65	ULI1	6.86	1	4.21
SK 65	URI1	6.84	1	3.44
SK 65	ULC	8.93	1	3.71
SK 65	URC	8.93	1	3.78
SKX 242	ULI1	11.09	0	
SKX 242	URI1	10.85	0	

¹ Not measured because cervical margin was broken off.

ysis by individual is not particularly informative. However, the analysis does suggest a slightly lower frequency of LEH in the *Paranthropus* sample and also indicates a difference with respect to the number of growth disruptions recorded in the enamel: in neither SK 65 nor SK 23 is more than one disruptive event indicated. However, in STW 498, three episodes are indicated by matched defects in the canine teeth, and two may be indicated in TM 1512.

By tooth

As assessed by either chi-square or Fisher's exact tests, the frequency of affected teeth does not differ significantly (at the 0.05 level) between robust and gracile hominins for any tooth type, when isomeres are either separated or combined. The sample size of each tooth type and the percentage of teeth for each type affected by LEH are shown in Table 3 and Figure 2. The histogram shows that there is a slight trend for the intertooth distribution of LEH in *Paranthropus* to be shifted toward later-forming teeth (premolars, and second and third molars) relative to *Australopithecus*. This difference is not statistically significant, however (df = 1, chi-square = 3.209, $P = 0.073$).

There is a statistically significant difference ($P = 0.005$, Fisher's exact test) between the *Paranthropus* and *Australopithecus* samples with respect to the number of defects recorded on canine teeth with 80% or more of their crown heights present. All 9 of the LEH-affected *Paranthropus* canines (5 lowers, 4 uppers) had only one defect, whereas 8 of 12 LEH-affected *Australopithecus* canines (6 lowers, 5 uppers) had two or more defects. These data are shown in Table 4 and Figure 3, respectively. This result is consistent with the expectation that more defects would be recorded in *Australopithecus* canines because their crown formation spans are longer than those of *Paranthropus*.

In the *Paranthropus* sample, relative to the average canine crown height of 10.38 mm (SD = 1.51; $n = 23$ canines), the average defect distance from the CEJ occurs at 31.1% of the measured crown height, with a standard deviation of 15.0%. In the *Australopithecus* sample, relative to the average canine crown height of 13.08 mm (SD = 2.28, $n = 26$ canines), the average defect distance from the CEJ occurs at 34.6% of the measured crown height, with a standard deviation of 14.6%.

Thus, even though most of the canine crown height is present, defects cluster between about 15–50% of the measured crown height in both samples. A t -test of the logged ratios of defect distances from the CEJ relative to measured canine crown height revealed no significant difference between the *Paranthropus* and *Australopithecus* samples ($P = 0.915$, separate variances). Because defects in both samples cluster in similar regions of the crown, the comparison of defect width between the two samples (below) is conducted in comparable crown regions.

Defect width with respect to adjacent perikymata spacing and number of perikymata within the defect

Tables 5 and 6 present the data on defect width and perikymata spacing for the *Australopithecus* and *Paranthropus* samples, respectively. While the average width of the *Australopithecus* defects at 504 μm is greater than the average width of the *Paranthropus* defects at 345 μm , this difference is not statistically significant. A t -test performed on the *Australopithecus* and *Paranthropus* difference in defect width on the logged values of all 21 data points had a P -value of 0.197 (separate variances). Despite this lack of significance, there appears to be a qualitative differences in the way in which enamel has been disturbed in these two genera. Figure 4 compares one of the widest defects of the *Australopithecus* specimens with that of one of the widest *Paranthropus* defects. With the exception of one, the *Paranthropus* defects were minor furrows (appearing as narrow ridges on the impressions), similar to those shown in Figure 4A,B, in which perikymata could actually be counted. On the other hand, while some of the gracile defects had this form, others were similar to the defects pictured in Figure 4C,D

TABLE 3. Percentage of teeth with LEH

	I1		I2		C		P1		P2		M1		M2		M3	
	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N
<i>Paranthropus</i>	18.2	11	22.2	9	32.1	28	36.8	19	22.2	18	0.0	19	9.5	21	8.3	12
<i>Australopithecus</i>	16.7	30	28.0	25	50.0	28	31.6	19	19.2	26	0.0	32	0.0	24	0.0	16

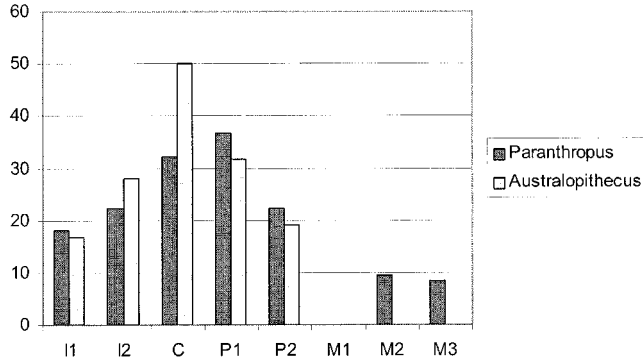


Fig. 2. Percentage of teeth with LEH by tooth class.

TABLE 4. Percentage of canine teeth with one vs. multiple (two or more) defects

	Canines with one defect	Canines with multiple defects
<i>Paranthropus</i>	100.0 (9)	0.0 (0)
<i>Australopithecus</i>	33.3 (4)	66.7 (8)

or Figure 1: large ridges (grooves in the original tooth) in which perikymata were not observable. Indeed, in Figure 4C,D, the enamel within the more incisal defect appears to have been strongly disturbed.

The linear regression of defect width on perikymata spacing for these 21 defects reveals no relationship between these two variables ($P = 0.286$). Figure 5 presents a graph of defect width vs. number of perikymata per millimeter for these 21 data points. Notice that for *Paranthropus*, the slope is negative, in accordance with predictions based on the model by Hillson and Bond (1997): where there are fewer perikymata per millimeter (i.e., where they are more widely spaced), LEH defects are wider. When the linear regression is run for just the *Paranthropus* defects, the regression coefficient is -0.624 , and the P -value is 0.098. The lack of statistical significance is likely related to the fact that there is no control in this linear regression model for the number of perikymata within the defect. On the other hand, the slope is slightly positive for *Australopithecus*, and when the linear regression is run with just the *Australopithecus* defects, the regression coefficient is 0.158, with a P -value of 0.606. This positive coefficient and large P -value are influenced by those *Australopithecus* defects that were very wide and seem to be of a different character than the minor furrows of either taxon.

Lastly, a forward-stepping multiple linear regression to determine the best predictors of defect width

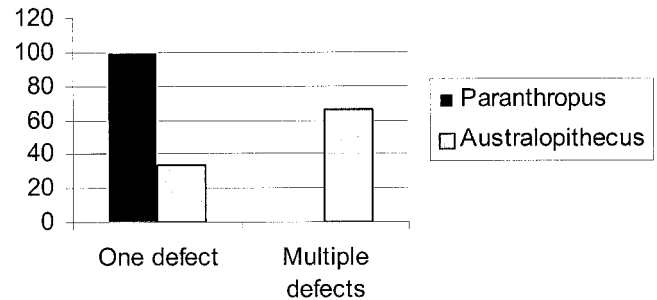


Fig. 3. Percentage of canines with one or multiple (two or more) defects.

was performed involving data points for which both the number of perikymata within the defect and the perikymata spacing were known. This analysis is therefore limited to the minor "furrow-form" defects (sensu Hillson and Bond, 1997), and does not include the wide grooves in which perikymata could not be counted. Variables examined are taxon (*Paranthropus* vs. *Australopithecus*), perikymata spacing, and number of perikymata within each defect. Interactions examined are taxon by perikymata spacing, taxon by number of perikymata within the defect, and taxon by perikymata spacing by number of perikymata within the defect.

On this subset of the data ($n = 15$; 8 *Australopithecus* defects and 7 *Paranthropus* defects), only the number of perikymata within the defect is significant by itself as a predictor ($P = 0.003$), accounting for 51% of the variance in defect width. As would be expected, the number of perikymata within the defect is positively associated with defect width (Fig. 6).

In the next step of the multiple linear regression, the number of perikymata within the defect is controlled, such that perikymata spacing now becomes a highly significant predictor of defect width ($P = 0.000$); however, taxon is not significant ($P = 0.135$), perhaps because of within-taxon variation in perikymata spacing. As anticipated by the model of Hillson and Bond (1997), the number of perikymata per millimeter is negatively correlated with defect width. With both the number of perikymata within the defect and perikymata spacing included in the model, 89% of the variance in defect width is explained with no other predictors (either taxon or any interactions among variables) significant. Tolerance values for the two predictors indicate that they are not highly correlated with any other predictors. Thus, for the minor furrow-form defects, most of the variance in defect width can be explained by these two variables: there is no significant difference be-

TABLE 5. Defect width in *Australopithecus canines* relative to perikymata spacing adjacent and incisal to defect, and number of perikymata within defect

Specimen	Defects (numbered from occlusal to cervical)	Defect width (μm)	Perikymata per mm cervical and adjacent to defect	Number of perikymata within defect
MLD 18	Defect 1	220	20	
MLD 42	Defect 1	357	20	7
STS 51	Defect 1	1,124	16	
STS 51	Defect 2	963	18	
STW 287	Defect 1	305	10	3
STW 287	Defect 2	307	11	3
STW 369	Defect 1	395	13	5
STW 369	Defect 2	435	14	6
STW 369	Defect 3	387	14	5
STW 410	Defect 1	1,011	14	
STW 446	Defect 1	572	10	
STW 498	Defect 1	255	14	4
STW 498	Defect 2	215	13	4
Averages		504	14.4	4.6

TABLE 6. Defect width in *Paranthropus canines* relative to perikymata spacing adjacent and incisal to defect, and number of perikymata within defect

Specimen	Defects (numbered from occlusal to cervical)	Defect width (μm)	Perikymata per mm cervical and adjacent to defect	Perikymata within defect
SK 65	Defect 1	392	10.0	4
SK 23	Defect 1	460	8.0	5
SK 38	Defect 1	278	9.5	3
SK 86	Defect 1	381	9.5	4
SK 87	Defect 1	340	10.0	
SK 96	Defect 1	189	14.0	3
SK 6013	Defect 1	505	9.0	6
SK 95	Defect 1	218	9.0	2
Averages		345	9.9	3.9

tween *Australopithecus* and *Paranthropus* defect widths when the number of perikymata within the defect is controlled or when both the number of perikymata and perikymata spacing are controlled.

DISCUSSION

This research was undertaken for the purpose of comparing LEH in a South African *Australopithecus* sample with that of a South African *Paranthropus* sample, evaluating any observed differences in light of taxonomic variation in aspects of enamel development and morphology that might impact LEH expression. In terms of LEH prevalence, there is little difference between *Australopithecus* and *Paranthropus*, either when individuals or teeth are the units of analysis. However, there is a statistically significant difference in the number of defects recorded in canines with 80% or more of their crown heights present: as observed macroscopically, none of the LEH-affected *Paranthropus* canines had more than one defect, whereas the majority of LEH-affected *Australopithecus* canines were affected with two or more defects. This difference was expected, given differences in the canine crown formation spans and extension rates of *Australopithecus* and *Paranthropus* (Dean and Reid, 2001). Thus, there is no need to invoke differences in the frequency of stress episodes experienced by *Australopithecus* vs. *Paranthropus* individuals as an additional explanation for this result.

Australopithecus and *Paranthropus* were found to differ little in the relative position of defects on their canine teeth, with most defects occurring within 15–50% of the measured crown height, even though 80% or more of the crown height was present in these samples. Defects in the upper 50% of the measured canine crown height were infrequent. In fact, it was often the case that in the incisal half of the crown, perikymata were spaced regularly, only to become disrupted in the lower half of the crown, sometimes a number of times in the gracile specimens. Figure 7 shows one example (STW 446) in which this was the case: note how even growth appears to have been in the incisal half of the crown.

This delineation between an initial period of undisturbed growth followed by a period of disrupted growth common to both *Australopithecus* and *Paranthropus* could represent a common cause in their life histories, such as the onset of the weaning process. On the other hand, as Katzenberg et al. (1996) pointed out, this pattern of disruption might result from intrinsic features of enamel (although the authors did not specify what these are). One feature common to *Australopithecus* and *Paranthropus* enamel formation that could produce this pattern is that enamel is formed more quickly in the incisal half of the crown (Dean and Reid, 2001). Prospective studies on human and nonhuman primates could help to discriminate between these two possible explanations. If the weaning process is related to this pattern, then the slight

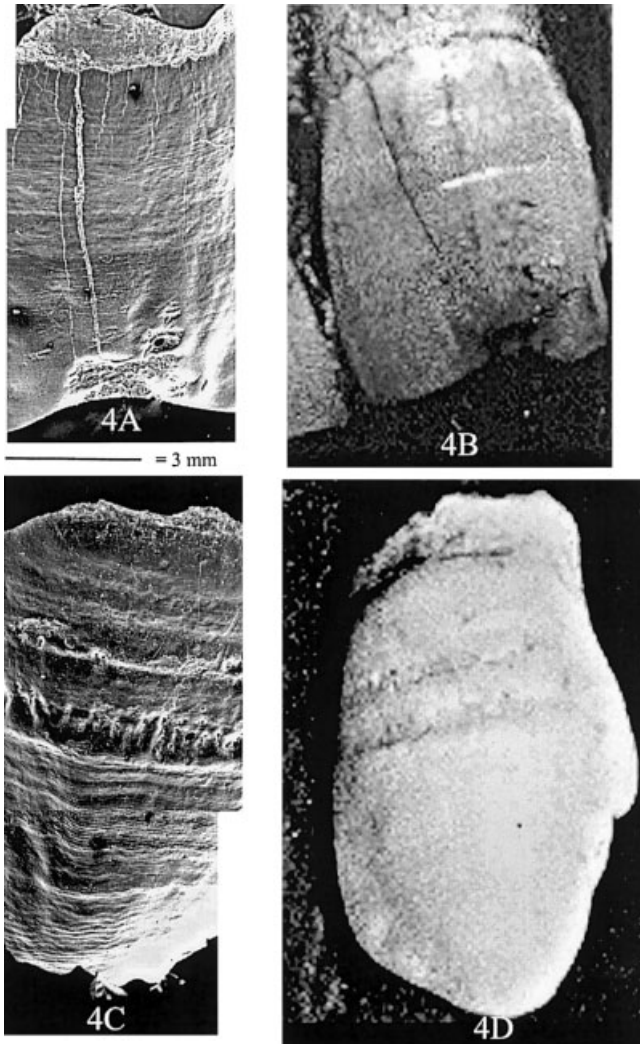


Fig. 4. A, B: SK 65 exhibits one of the widest *Paranthropus* defects. **A:** Micrograph of impression. **B:** Photograph of tooth. The SK 65 tooth was scored as having only one defect (the most prominent one) in the macroscopic scoring. Under SEM, here at a magnification of $\times 20$, a more minor furrow is visible cervical to the larger defect. C, D: STS 51 has one of the widest *Australopithecus* defects. **C:** Micrograph of impression. **D:** Photograph of tooth.

trend for later-forming teeth to be more affected in *Paranthropus* relative to *Australopithecus* may indicate that *Paranthropus* was more dentally advanced at weaning age. Godfrey et al. (2001) showed that folivorous primates have greater dental “endowment at weaning” (a measure of the postcanine occlusal area present at weaning relative to that of an adult) than do comparably sized frugivores. The authors argue that this greater endowment in folivores is necessary for processing fibrous leafy material and/or seeds. It seems plausible that South African *Paranthropus* would be more dentally “endowed” at weaning, owing to its numerous cranio-dental adaptations to heavy masticatory stress.

In this study, defect widths were also compared between *Australopithecus* and *Paranthropus*, with

the expectation that the closer spacing of perikymata in the former would result in narrower defects. This result was not obtained, most likely because of the very large groove defects seen in several of the *Australopithecus* specimens. Analysis of a small subsample for which both perikymata spacing and the number of perikymata within the defect could be determined revealed no difference between *Australopithecus* and *Paranthropus* in terms of defect width, even when the number of perikymata within the defect was controlled, perhaps because of overlap in the perikymata spacing of *Australopithecus* and *Paranthropus*. The best predictors of defect width in this subsample were the number of perikymata within the defect and the number of perikymata per millimeter adjacent to the defect, together accounting for 89% of the variance in defect width.

Given the difference in mean number of perikymata per millimeter adjacent to defects in *Australopithecus* vs. *Paranthropus* observed in this study (see Tables 5 and 6), the lack of wider defects in *Paranthropus* is surprising. The results may indicate that stress episodes in *Australopithecus* were more severe, as the large groove defects in *Australopithecus* entail what appear to be disturbed enamel in which perikymata are not observable. There is no significant difference in the average number of perikymata within the minor furrows of *Australopithecus* and *Paranthropus*, such that for these minor furrows, a taxonomic difference in the duration of stress episodes is not indicated.

There are several reasons why defect widths can vary, as is shown in Figure 8. In Figure 8, the perikymata in the occlusal wall of the defect, most representative of the period of disturbed enamel formation, are shown between two horizontal dotted lines. The defect in Figure 8B is wider than that in Figure 8A simply because the perikymata are spaced more widely. The defect pictured in Figure 8C is wider because there are more perikymata within the defect, reflecting a stress episode of longer duration. A third factor, however, is the amount of each Retzius plane that is exposed at the enamel surface: with larger percentages of planes exposed, the defect becomes wider because the Retzius planes are not perpendicular to the enamel surface. The defect in Figure 8D demonstrates this situation that presumably results when a disturbance affects a larger group of ameloblasts on the enamel-forming front. This latter situation reaches an extreme in what have been termed “plane-form” defects in which an entire Retzius plane may be exposed (Hillson and Bond, 1997). This may be what is happening in the *Australopithecus* groove defects, though it is difficult to demonstrate that this is the case. In some of these grooves, it was possible to discern perikymata along the sloping walls of the defect, while they were not discernible over the greatest part of the defect’s width, possibly because a large portion of a single or a few Retzius planes may have been exposed. If this is the case, the in-

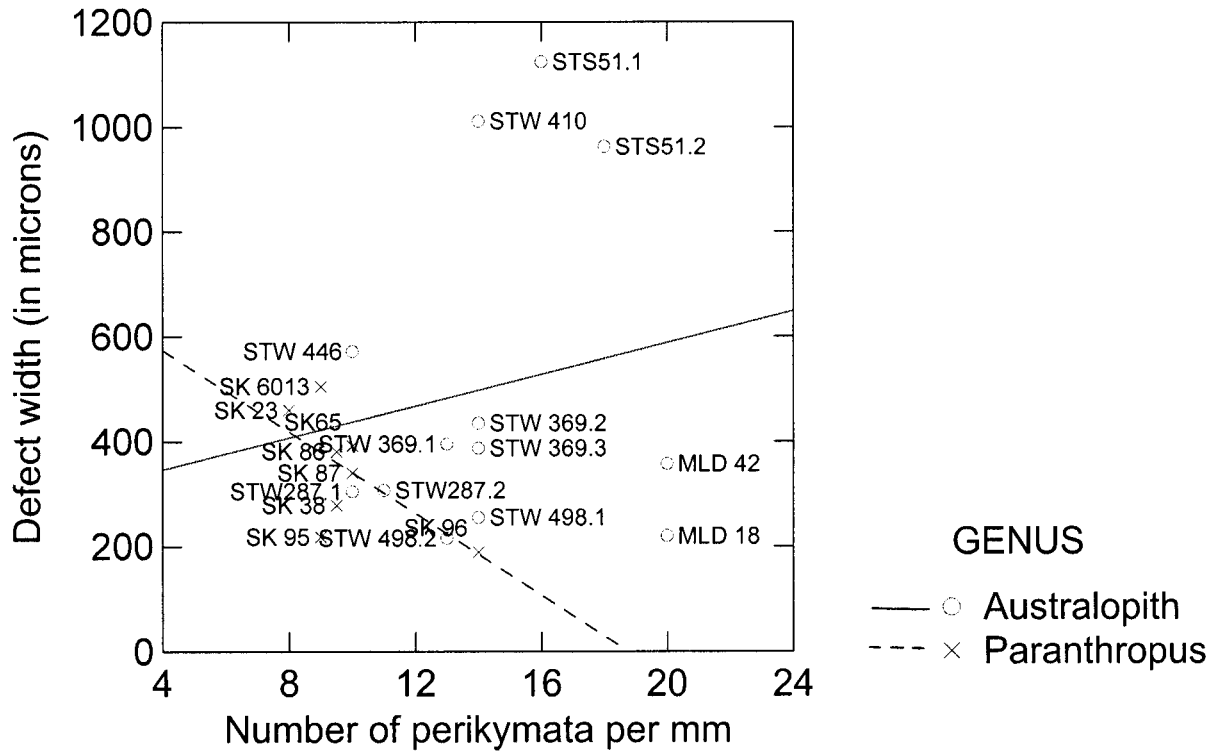


Fig. 5. Relationship between defect width and perikymata spacing (measured as number of perikymata per millimeter adjacent and incisal to each defect). Note that for *Paranthropus*, the relationship is negative, as expected: as perikymata become more closely spaced, defects are more narrow in width. The relationship is slightly positive for *Australopithecus* because many specimens had wide groove defects, for which the spacing of perikymata may have been irrelevant: no perikymata were observed within these wide groove defects.

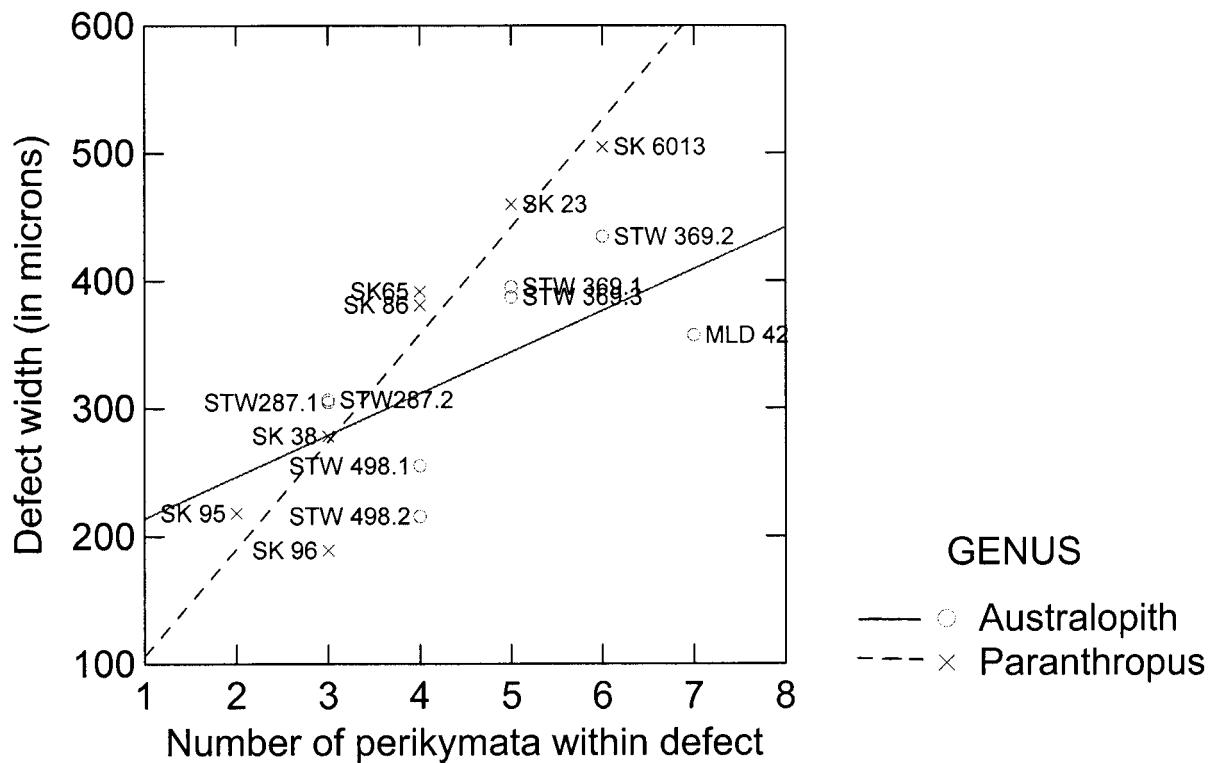


Fig. 6. Relationship between defect width and number of perikymata within defect on a small subsample of minor furrows (n = 15 defects) for which perikymata could be counted within the defect. Note that the relationship is positive for both *Australopithecus* and *Paranthropus*.

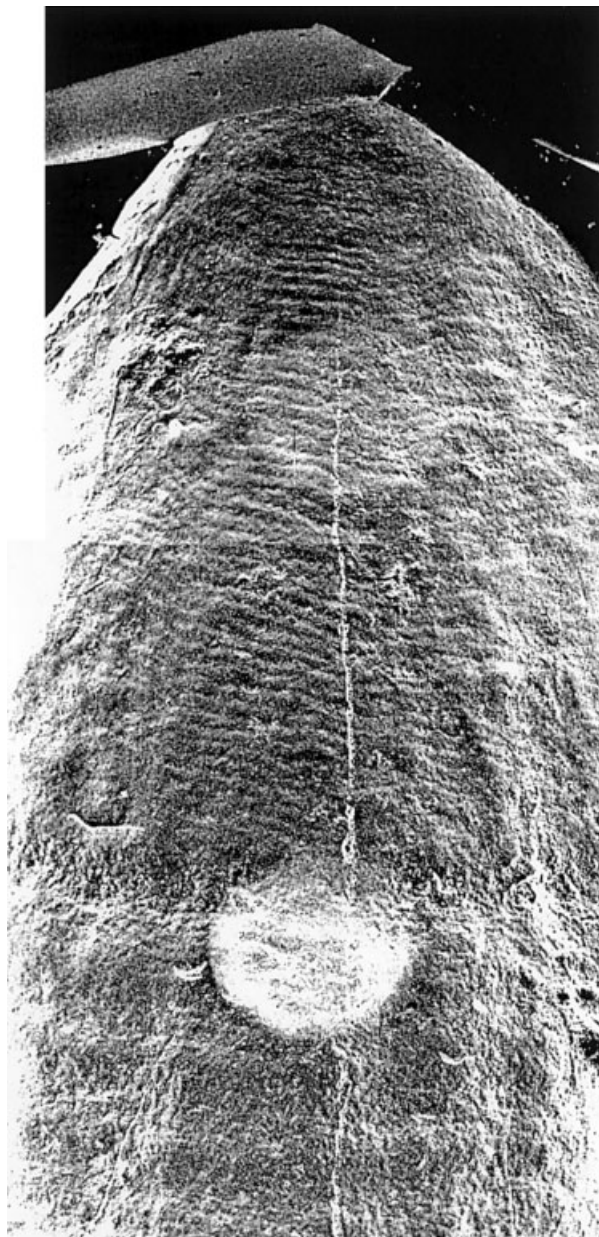


Fig. 7. STW 446, showing patterning of perikymata on upper 50% of crown height, with multiple disruptions occurring in lower half of crown. Part of lower portion of tooth is missing from micrograph, and two white circular artifacts are present.

significant positive relationship between perikymata spacing and defect width in *Australopithecus* can be more easily understood as the result of width being influenced more by the amount of exposed Retzius planes than by perikymata spacing.

This interpretation of how wide grooves are formed is consistent with the experimental work by Suckling (1989) on inducing hypoplastic lesions in sheep through parasitic infection. Suckling (1989) found that there was no relationship between the duration of the stressor (period of infection) and the

amount of missing enamel, and on this basis, refutes the assertion of Sarnat and Schour (1941) that a "wide groove" takes several months to form. According to the experiments of Suckling (1989, p. 91), the "severity of the stressor," not the duration of the stress event, determines the "extent of the lesion" in wide groove defects. The parallel between wide grooves in hominins and hypoplastic lesions in sheep must be interpreted cautiously, however, because of differences in the way sheep and hominins deposit enamel (Don Reid, personal communication).

If the groove defects in *Australopithecus* indeed represent more significant disruptions than the minor furrows, then the question is why such defects are nearly all observed in *Australopithecus* as opposed to *Paranthropus*. One possibility is that the ameloblasts of *Australopithecus* may be more sensitive to disruption than those of *Paranthropus*, indicating that tooth growth was more canalized in *Paranthropus*. A second possibility is that sample bias is at work: that owing to the "osteological paradox" (Wood et al., 1992), severely stressed robust australopiths did not survive to record defects in their teeth. Although this is possible, it is not clear why *Australopithecus* and not *Paranthropus* would have survived to record these potentially more severe disruptions. A third possibility involves dietary differences between the robust and gracile South African australopiths. While Grine and Kay (1988) provided microwear evidence that *A. africanus* subsisted on fruits and leaves (as do modern chimpanzees), Sponheimer and Lee-Thorp (1999, p. 368) found that *A. africanus* also ate ^{13}C -enriched foods such as "grasses and sedges or animals that ate these plants, or both." The authors argue that *A. africanus* and *P. robustus* may have eaten similar food items, but that the latter may have processed these foods more efficiently. If this were the case, then the difference in groove defects between *Australopithecus* and *Paranthropus* observed in this study may be related to differences in food-processing efficiency.

In conclusion, this study suggests that both intrinsic differences in the enamel of South African robust and gracile australopiths, as well as differences in the severity of stress episodes recorded in their teeth, contribute to observed differences in LEH expression between them. Longer crown formation spans in *Australopithecus* may result in their larger number of defects on canine teeth relative to *Paranthropus*. However, the influence of perikymata spacing on defect width is not in the expected direction, such that *Paranthropus* defects are not wider than those of *Australopithecus*. The most likely explanations for this latter result are taxonomic differences in ameloblast sensitivity and differences in the severity of stress episodes experienced by *Paranthropus* and *Australopithecus*. Both possibilities suggest that there was a difference between *Paranthropus* and *Australopithecus* in the nature of their adaptations to stresses affecting enamel growth.

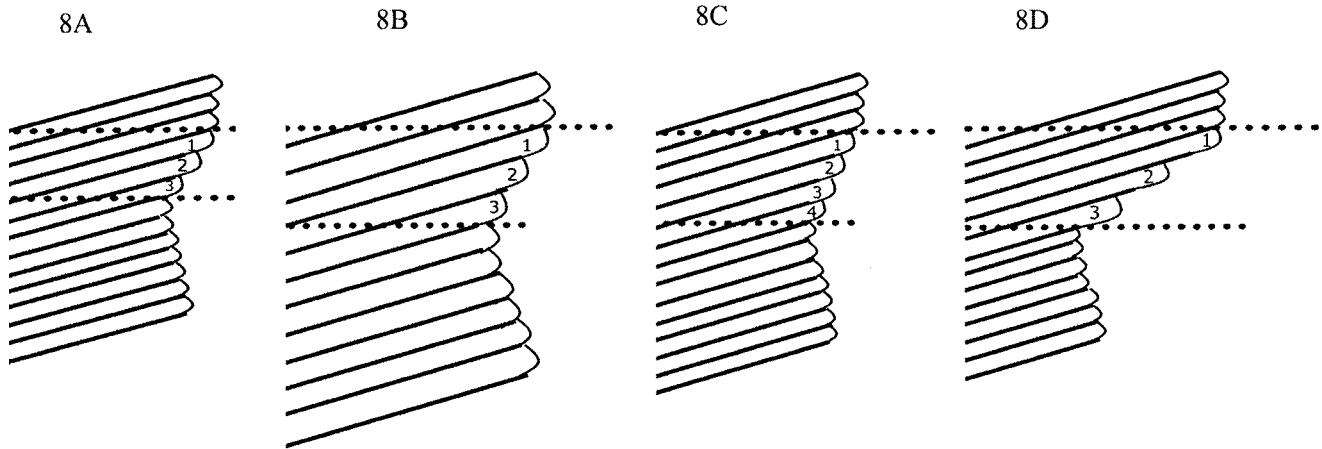


Fig. 8. There are several reasons why defect widths can vary. Shown here is a cross section of tooth enamel in which layers represent Retzius planes, and curved ends of the planes represent perikymata, where planes “outcrop” onto enamel surface. Perikymata in the occlusal wall of the defect, most representative of the period of disturbed enamel formation, are shown between two horizontal dotted lines. **A:** Diagram is based on model of Hillson and Bond (1997) for defect formation. **B:** Defect is wider than in A simply because perikymata are spaced more widely. **C:** Defect is wider because there are more perikymata within the defect, reflecting a stress episode of longer duration. **D:** A third factor, however, is the amount of each Retzius plane that is exposed at the enamel surface; with larger percentages of planes exposed, the defect becomes wider because the Retzius planes are not perpendicular to the enamel surface.

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