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Developmental Defects of the Dental Crown in Chimpanzees From the Taï National Park, Côte D'Ivoire: Coronal Waisting

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KEY WORDS *Pan troglodytes verus*; weaning process; dentin; stress; infancy

ABSTRACT We describe a developmental defect that manifests as a mild constricted "waist" in anterior teeth from seven of nine chimpanzee individuals from Taï National Park, Côte D'Ivoire. The sample consists of 21 canine teeth and one incisor, imaged in profile with a digital microscope. Twelve teeth are affected. The waist develops during tooth formation as an external, encircling depression in the contour of the outer enamel surface, more easily seen labially. It is not a thinning of enamel per se, but rather a slight decrement in dentinal crown volume, shown in microCT scans as a change in contour of the enamel-dentin junction, spanning between 3 and 6 years of age, varying among individuals, with maximum expression at about age 4.3 years. The timing and duration of coronal waisting are consistent with descriptions of the weaning process at Taï and other chimpanzee study sites. We propose that coronal waisting records variation in the individual infant chimpanzee's physiological experiences during the process of attaining independence, increased foraging efficiency, and lactational weaning. Am J Phys Anthropol 149:272–282, 2012. ©2012 Wiley Periodicals, Inc.

Defects of enamel and dentin preserve an almost permanent record of an immature mammal's developmental experiences (Dobney and Ervynck, 1998; Dobney and Ervynck, 2000; Franz-Odendaal, 2004; Niven et al., 2004; Kierdorf and Kierdorf, 2007). We report the existence of a new developmental defect of anterior crown formation among chimpanzees from the Taï Forest whose expression will have to be differentiated from familiar forms of enamel hypoplasia. There is general acceptance that dental development provides a useful understanding of differential morbidity and mortality as influenced by birth, maternal nutrition, sexual and social differences, and so on. Human studies of enamel defects focus primarily on the interaction of the weaning process, malnutrition and disease (Goodman and Rose, 1990; Skinner and Goodman, 1992; Blakey et al., 1994; Hillson and Bond, 1997; Goodman and Song, 1999; Lukacs, 1999b; Guatelli-Steinberg et al., 2004). Non-human primate studies of enamel surfaces have emphasized ecological differences among species (Moggi-Cecchi and Crovella, 1991; Eckhardt, 1992; Eckhardt and Protch von Zieten, 1993; Guatelli-Steinberg, 1998; Newell, 1998; Guatelli-Steinberg and Lukacs, 1999; Lukacs, 1999a; Guatelli-Steinberg, 2000; Lukacs, 2001; Skinner and Newell. 2003; Hannibal and Guatelli-Steinberg. 2005; Lukacs. 2009) and effects of seasonality (Skinner. 1986; Guatelli-Steinberg and Skinner. 2000; Skinner and Hopwood. 2004) on enamel formation. There is an emerging literature on enamel hypoplasia among fossil hominins and apes whose patterning can be compared directly to that of extant species (Moggi-Cecchi and Crovella. 1992; Skinner et al., 1995; Skinner and Roksandic, 1995; Guatelli-Steinberg et al., 2004; Guatelli-Steinberg, 2008).

Here we report preliminary observations of a newly recognized developmental defect of the Taï chimpanzee anterior dental crown that we term "coronal waisting." We define coronal waisting as a macroscopic constriction of the normal profile of the mid-canine crown involving both dentin and enamel (Figs. 1 and 2). It is more obvious on the labial side but can encircle the crown. The condition is not restricted to the canine but is more obvious on this tooth type. We have not examined cheek teeth for waisting. It should be emphasized that our study is preliminary and was not designed to elucidate this adventitious observation. MicroCT scans of affected teeth show that the contour of the underlying dentin is affected; this in turn contributes to an abnormal depression in the contour of the outer enamel surface (Fig. 1). We consider this departure from normal to be a marker of physiological stress. Based on the reconstructed onset and duration of coronal waisting, we infer that the cause

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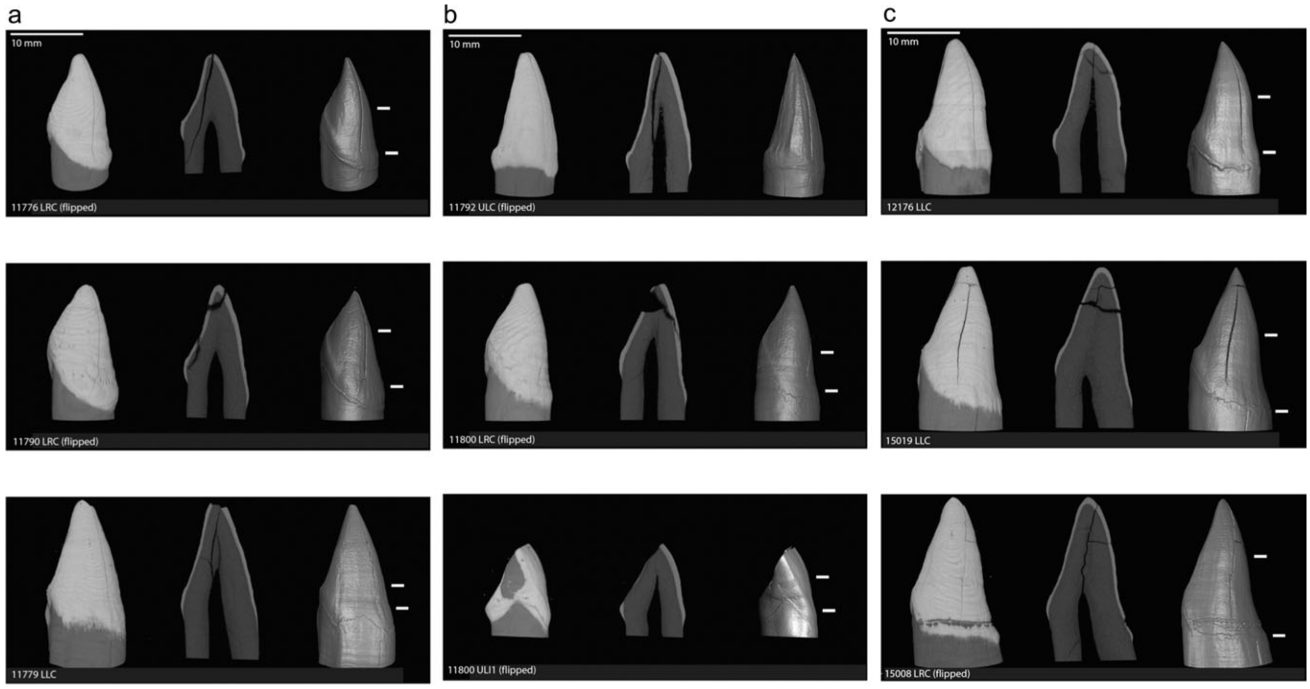

Fig. 1. **A–C:** Taï chimpanzee teeth. Images left hand panel is a volume rendered surface model, middle panel is a slice showing EDJ contour and enamel thickness, right hand panel is the dentin crown with enamel removed digitally. Horizontal white bars show location of waisting. First L, lower; U, upper; second L, left; R, right; C, canine; I, incisor. All teeth shown with apex up and lingual side to the left, to aid comparison); see text for further details. All teeth show waisting except for tooth 11792 in 1B.

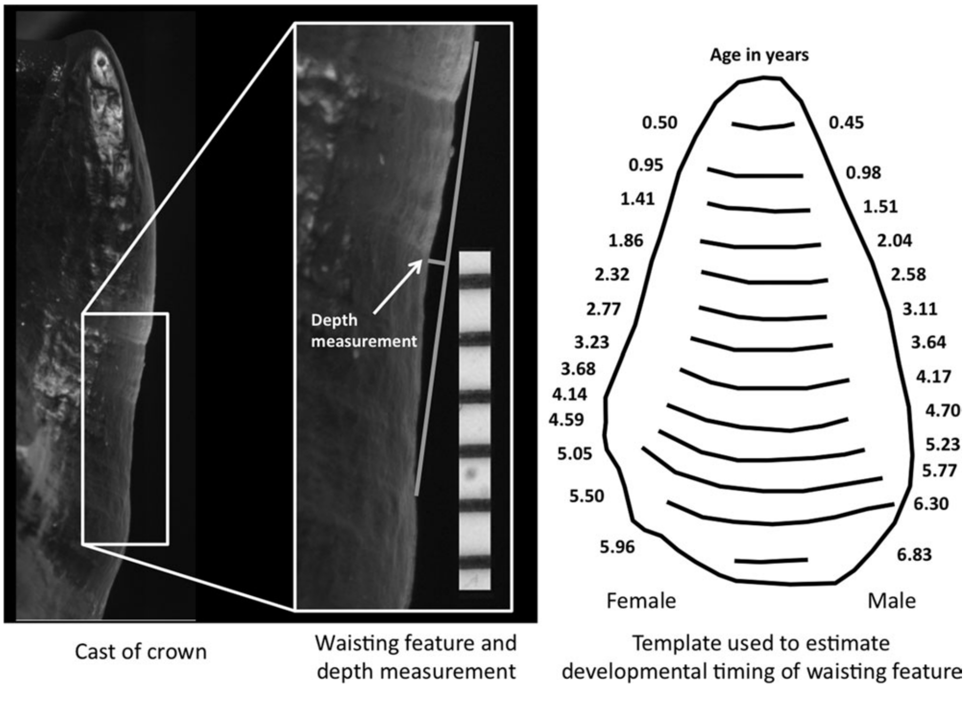

Fig. 2. **A**: Example of measurement of waist depth on lower left canine (12176) (same tooth as in Fig. 1C). This waist is considered Marked (depth = 260 μm) (ruled scale is mm). Note that the depth measurement deliberately excludes the cingulum. **B**: Reconstructed approximate timing of canine crown formation in chimpanzee males and females in 20 perikymata increments (ca. 6 months). Replicability of perikymata counts was tested on Zerlina's upper left canine with counts varying by only one perikyma (<0.5%).

of the defect is growth faltering and recovery during a protracted process of growing infant independence and lactational weaning. We use the phrase 'weaning process' in the broad sense provided by Martin (1984) : "... the whole array of behavioural, nutritional, morphological and physiological changes that constitute the transition

TABLE 1. Study subjects and coronal waisting

Animal	Sex	Age[a]	Number	Tooth (FDI)[b]	Waist	
					Severity	Depth (μm)
Arianne	Female	12.3	11776	2–3	none	–
				1–3	None	–
				4–3	Marked	200
Clyde	Male	12.6	11779	2–3	Mild	80
				3–3	Moderate[c]	150
				4–3	Mild	60 (occlusal only)
Tina	Female	9.6	11790	1–3	None	–
				3–3	Moderate	150
				4–3	Moderate	180 (occlusal too)[f]
Zerlina	Female	12.3	11792	2–3	None	–
Kiri	Female	23	11800	2–3	None	–
				1–3	None	–
				4–3	Moderate	180
				2–1[d]	Moderate	140
No name	Male	–	12176	3–3	Marked	260
				4–3	Marked	280
Nerone	Male	13	15008	4–3	Marked	300
Leo	Male	19	15012	4–3	Mild	100
Aramis	Male	adlt	15019	2–3	None	–
				1–3	None	–
				3–3	Uncertain[e]	360
				43	Uncertain[e]	350

[a] (Smith and Boesch, 2011).

[b] Federation Dentaire Internationale nomenclature.

[c] Unnaturally broad cingulum indicating recovery from stress, so counted and measured.

[d] Incisor.

[e] Bulge of cingulum may be exaggerating waist, so not counted.

[f] Two waists, of which the more cervical was measured and counted.

to an independent adult existence; a major transformation which may span an extensive period in ontogeny" (p. 1257).

MATERIALS

Observation of coronal waisting was incidental to other studies of repetitive episodes of linear enamel hypoplasia (rLEH) underway on casts of canine and incisor teeth from chimpanzees (*Pan troglodytes verus*) from Taï National Park, Côte D'Ivoire (Skinner et al., in prep.). Consequently, the study sample includes teeth chosen on the basis of showing countable perikymata between two or more macroscopically visible episodes of linear enamel hypoplasia, a phenomenon that we think reflects seasonality. The site (5°45'N and 7°7'W) is described as lowland rain forest with a major dry season from November to February and a minor dry season in July and August (Doran, 1997). There are two wet seasons: April–June and September–October (short wet season) and there is an annual fruiting trough in May (Anderson et al., 2005). The collection is curated at the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany. In this study, teeth were collected from animals that died between 1984 and 2007 (Table 1).

The study sample includes 21 canine teeth (nine maxillary and 12 mandibular) and one maxillary incisor tooth, chosen for comparative purposes, from four females and five males. Animals in this study have relatively less worn labial enamel; consequently, this is a young mortality cohort. While it is possible that waisting is peculiar to this particular subspecies of chimpanzee (*P.t. verus*), the phenomenon is likely to be more ubiquitous as we think it occurs in pigs as well (Witzel et al., 2006) (see below).

METHODS

Imaging the outer enamel surface

Molds of affected enamel surfaces were taken using Coltene President Plus Jet impression material. Casts were later made in Araldite MY 753 epoxy resin with XD 716 hardener (Ciba-Geigy). Casts were examined under oblique light at low magnification for episodes of linear enamel hypoplasia (LEH). Photographic images, covering the whole crown surface were recorded at high magnifications ranging typically from 50 to 100X with a VHX-100 Keyence digital microscope. Separate images were combined with Adobe Photoshop Elements 6 to form a photomontage of the enamel surface; these could be examined at very high magnifications allowing each episode of linear enamel hypoplasia to be identified and homologized among teeth from one individual. All depth measurements of LEH were taken using a Keyence VHX-100 digital microscope at 300× magnification using the depth-to-defocus function. On a machined block of grooved aluminum, replicated depth measures averaged 3.1% different (regardless of sign) from the standard.

Our examination revealed the presence on some teeth of much wider troughs of depressed enamel-the subject of this article. These areas were further imaged with the digital microscope to provide profile pictures of measured depth of depressed outer enamel surface (Fig. 2A). Repeated measures of depth on the casts varied by an average of 5%.

Imaging the dentin crown

Nine teeth were examined with micro-computed tomography to determine whether the dentin crown was

TABLE 2. Lower canine crown formation in years from birth among chimpanzees (sources indicated)

	Male		Female	
Stage	Kuykendall[a] (range)	T. Smith[b]	Kuykendall (range)	T. Smith
1 (init. miner.)	0.45	–	0.49	0.74 (Ophelia)
2	1.18	1.77–2.13 (Leonardo/Bambou)	1.18	–
3 (crown ca. ½)	4.16	5.24 (Oreste)	3.22	3.76 (Pimente)
4 (crown compl)	7.28 (6.37–8.20)	7.6 (Lefkas)	6.45 5.(60–7.69)	6.45(Goshu)

[a] (Kuykendall, 1996).
[b] (Smith et al., 2010)

involved (Fig. 1). Each tooth was scanned (SkyScan 1172, Kontich, Belgium) at an isometric voxel resolution of ~ 17 μm (100 kV, 94 μA, 2.0 mm aluminum and copper filter, 0.12 rotation step, 360° of rotation, 2 frame averaging). Raw projections were converted into TIFF image stacks using NRecon (parameters: ring artifact correction = 10; beam hardening = 30%). To facilitate tissue segmentation, the complete image stack for each tooth was filtered using a three-dimensional median filter (kernel size of 1) followed by a mean of least variance filter (kernel size of 1), implemented as a computer-programmed macro (Schulze and Pearce, 1994). This filtering process resulted in more homogenous tissue classes (i.e., enamel vs. dentin) and allocated pixels with intermediate gray-scale values at tissue interfaces (i.e., air-enamel, enamel-dentin, air-dentin) to the appropriate tissue. Segmentation of the filtered image stacks was completed semiautomatically and surface models of the enamel and dentin surfaces were created using Avizo 6.3 (www.vsg3d.com).

Determining the age at occurrence of waisting

Currently, reconstruction of the timing of developmental defects of teeth requires either histological sections of individual teeth, which yield exquisite precision and accuracy (e.g., Schwartz et al., 2006; Smith and Tafforeau, 2010) and can serve to validate timing based on perikymata counts; e.g. (Guatelli-Steinberg, 2008); or, when population patterns are being sought and destructive techniques are not justifiable, as in this study, one must turn to radiographic standards of dental crown formation. Recently, there has been debate whether chimpanzees in the wild mature significantly slower than those in captivity (Zihlman et al., 2004, 2007). In terms of *dental eruption*, H. Smith has concluded that there is indeed a mild wild-effect amounting to about 1 year (Smith and Boesch, 2011). T. Smith et al. (2010) have re-evaluated dental formation among known age or histologically aged Taï chimpanzees. They conclude that Kuykendall's (1996) radiographic standards of tooth formation among captive chimpanzees remain appropriate for estimating the timing of dental formation in wild chimpanzees. Table 2 summarizes canine crown formation in chimpanzees.

It can be calculated from Table 2 that average canine crown formation spans about 6.83 years in males and 5.96 years (Kuykendall, 1996) to 5.71 years (Smith et al., 2010) in females. These values can be compared to 6.81 ± 0.56 (range, 5.91–7.58) in males and 5.85 ± 0.51 (range, 5.28–6.49) in females reported by Schwartz and Dean (2001). If cuspal enamel in *Pan* takes 7.5% of crown formation span (Reid et al., 1998), then lateral/imbricational enamel (that which potentially records linear enamel hypoplasia) forms over a span of about 6.5 years in males and 5.6 years in females.

With the approximate time span for imbricational enamel derived, the next step is to render crown formation intervals into finer increments than those shown in Table 2. This can be accomplished by recourse to perikymata which are deposited with an individually invariant periodicity [ranging from 5 to 10 days in hominoids (Dean and Beynon, 1991; Dean and Reid, 2001; Smith et al., 2007)]. Typically, perikymata are more widely spaced at the cusp tip becoming more finely packed cervically (Dean and Reid, 2001). In only one tooth in this series— a female's (Zerlina) upper left canine—were all perikymata visible from cusp tip to cervix, amounting in this case to 244. To some this may seem a low number, as it suggests a high periodicity tooth with a long count on the order of 8–10 days to form one perikyma; however, it is quite in keeping with the few other published accounts of actual counts of perikymata in African ape canine imbricational enamel. Beynon et al. report (1991) perikymata counts on incomplete juvenile gorilla canine teeth: Upper canine >235, Lower canine >227. Dean and Beynon (1991) describe a male chimpanzee complete canine with 284 perikymata and a female with 306. A *Pan paniscus* male canine (90% visible crown surface) had 272 perikymata and a female, 228 in female (Guatelli-Steinberg et al.. 2012). The precise number of perikymata is immaterial except in so far as their distribution (packing density) along the canine crown allows us to reconstruct the timing of waist formation with greater precision than that afforded by published standards of crown formation. The simplifying assumption is made here that this count would apply to all canine teeth. In other words, the imbricational enamel of an average chimpanzee canine tooth would form in about 244 equal period increments spread over 6.475 and 5.55 years for male and female canine teeth, respectively. This approach allows for the fact that crown formation slows slightly in a cervical direction and provides a means of estimating the timing of events during crown formation with an uncertainty of about ±6 months (1 SD in Schwartz and Dean, 2001). Figure 2B illustrates this reconstructed "yardstick" in 20 perikymata increments that was used to estimate the age of onset and duration of waisting for all affected teeth by overlaying a transparency of Figure 2 over an image of the tooth.

RESULTS

Prevalence of waisting phenomenon

Remembering that these teeth were chosen for a purpose other than to illustrate waisting, in a sample of 22 teeth, waisting was observed in 12 teeth (11 canines and one incisor) from seven (of nine) animals. Only two of 10 upper teeth were affected, while 10 of 12 lower teeth showed waisting. In six animals (three affected) for whom both isomeres were present and scorable (six left/right

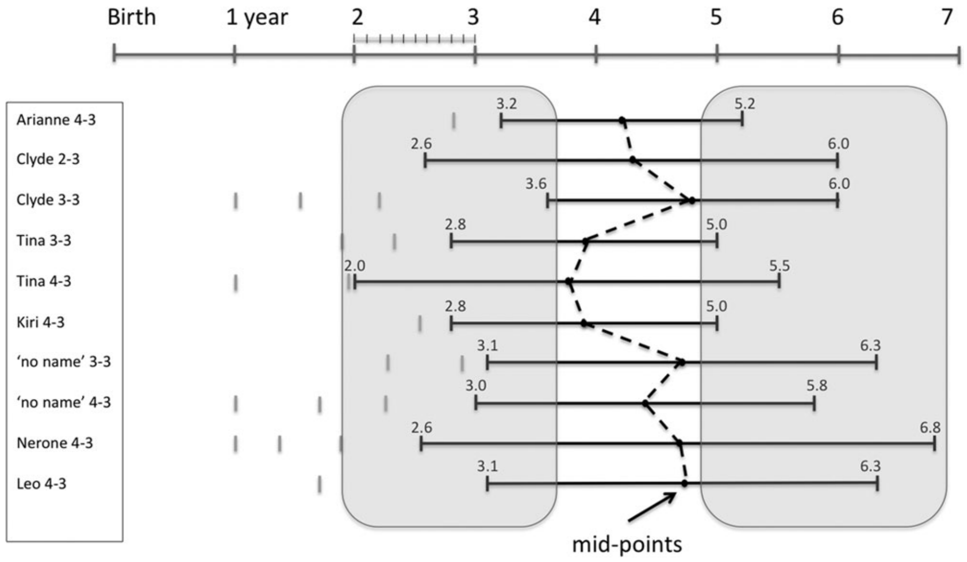

Fig. 3. Reconstructed variation in the approximate timing (years after birth) of the onsets, spans, and cessation of waisting on Taï chimpanzee canine crowns. Vertical isolated bars indicate episodes of prewaisting linear enamel hypoplasia; dotted line connects the mathematical mid-point of each span (see text for further details).

pairs), there was complete symmetry in all (Table 1). The difference between jaws may reflect normal occlusal relationships in the mouth which protect labial enamel of lower canines from wear more so than for uppers.

Onset and duration of waisting

Our knowledge of the timing of formation of the teeth from these subjects is imprecise since it was estimated from average rates among captive animals; consequently, we feel that an uncertainty of at least six months on either side of our estimated ages of onset and duration of waisting is appropriate. Infancy in Taï chimpanzees lasts up to 5 years of age; juveniles are aged 5–10 years and still in constant association with the mother (Boesch and Boesch-Achermann, 2000). Canine tooth formation embraces the full span of infancy including completed lactational weaning, up to age six or so (Kuykendall, 1996). As shown in Figure 3, waisting commences at about age 3 years and lasts almost 3 years on average. Ages of onset and cessation among individuals both range over 18 months. Ages of onset vary between 2 and 3.6 years (mean = 2.9 years) and ages of cessation between 5.0 and 6.8 years (mean = 5.8). We think variation in the timing of waisting is important and must reflect variation in aspects of its posited etiology (see later).

Degree of waisting

Degree of waisting was assessed two ways: a subjective assignment to a scale varying from None to Mild, Moderate, and Marked. Then, each "waist" was measured, with a Keyence digital microscope (calibrated for each measurement) at maximum depth between bordering high points on the outer enamel contour, drawn as a chord (straight line) avoiding any cingulum; this approach is quite replicable but provides only a minimum depth (Fig. 2A) as it ignores natural crown convexity. The results are provided in Table 1. As shown in Figure 2A, the waisting effect tends to reach a maximum depth at about mid-point, averaging 4.3 years but with notable variation (Fig. 3); that is, the inferred stressor is being experienced for about a year and half then, just as slowly, there is recovery from the stress (see Discussion). Some waists are wider than others. How to interpret this is unclear. It may be that wide defects are due to longer lasting stress; or, alternatively, the stress may be more severe, producing a deeper defect, since there is a period of reattainment of coronal enamel contour. Arguably, "duration" equals half the observed width of the defect excluding the recovery phase. Equally one could argue, using a medical analogy, that an animal is poorly until it is well. Pending further research we accept defect width as expressing stress duration.

Relationship between waisting and repetitive linear enamel hypoplasia (rLEH)

Figure 3 includes an indication of those episodes of linear enamel hypoplasia that predate the onset of waisting; this was done so as to illustrate that the two phenomena are separate. A year or more passes in the life of the Taï chimpanzee infant, before waisting commences, during which time there are brief intervals of stress sufficient to affect enamel formation.

Figure 4, which illustrates (in exaggerated vertical scale) the full range of severity of waisting, shows that the outer enamel surface is far from even; rather there are times, separated by about a year or occasionally 6 months, when the enamel thickens slightly. Our current research on perikymata counts between episodes of repetitive linear enamel hypoplasia in this sample indicates that undulations in the enamel contour are seasonally timed (Skinner et al., in prep.). We do not see evidence for corresponding undulations in the enamel-dentin junction (Fig. 1) and consider these to be separate phenomena. Analysis (ANOVA) of the relationship between degree of waisting and the depths of LEH events on affected teeth [Table 3 shows the association is not statistically significant ($F = 1.285$, $P = 0.285$)].

To summarize the results of this study of developmental defects of Taï chimpanzee anterior teeth, we report a slight decrement in dentinal crown volume at mid-

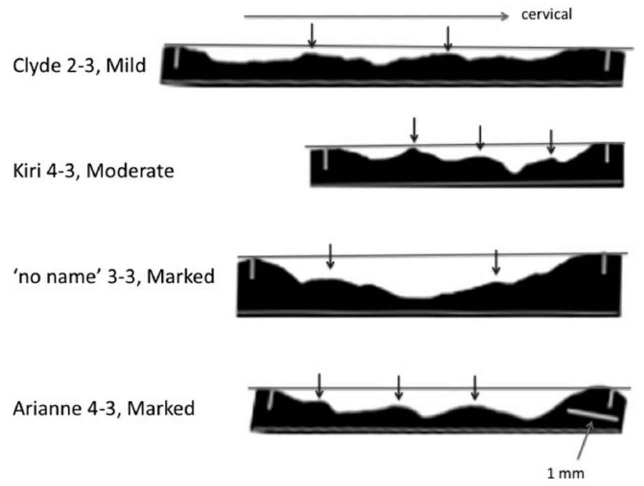

Fig. 4. Severity of waisting (vertical scale exaggerated by 300%); downward pointing arrows indicate times of apparently ameliorated stress (see text for further details).

TABLE 3. Relationship between degree of waisting and depths of linear enamel hypoplasia events on an affected tooth

Degree of waisting	*N*	rLEH mean depth (μm)
None	18	−19.3
Mild	13	−20.8
Moderate	31	−23.9
Marked	20	−35.3

crown, of lower canines especially, affecting the contour of both the inner and outer enamel surfaces of most but not all individuals. We consider this change to reflect some form of stress commencing at about age 3 years and lasting up to 2 years or more till the end of infancy. The average age of maximum waisting centers on 4.3 years. Enamel spanning the waisted area is observed to be thinner or of normal thickness. In addition, in some individuals, we observed brief episodes of milder stress, in the form of repetitive linear enamel hypoplasia (rLEH) occurring throughout crown formation that is superimposed on the waisted area. In brief, we find three distinct abnormal variations in enamel contour (Fig. 5).

DISCUSSION

We have defined waisting as "macroscopic" to emphasize that a threshold of visibility has to be crossed. There likely exists a continuum from unaffected, to slightly affected, to markedly so. For example, tooth 11,792 in Figure 1 may show waisting near the cervix, but the contour change is insufficient for a diagnosis of presence. Another problem of diagnosis occurs when there is a pronounced cingulum that accentuates any waisting and must be ignored (e.g., tooth 12,176 in Fig. 1). We do not understand as yet the distribution within the mouth of the occurrence of cingula. For both these reasons, our estimate of the prevalence of waisting is conservative.

Is waisting a real phenomenon? We think so. While one might suggest that some canines are naturally waisted, we have found that more than half do so, a level of expression that has not been reported before. Also an incisor shows waisting; which seems quite abnormal in this tooth type. Finally, a similar condition has been described in pigs from Medieval sites (Dobney and Ervynck, 1998; Witzel et al., 2006) and attributed to under-nutrition (Ervynck and Dobney, 1999). These authors observe that there is reduced prevalence of enamel depressions in permanent second molars, whose crown formation occurs during winter shortages, among semi-confined pigs compared to herded pigs; the latter experiencing less controlled provisioning (Ervynck and Dobney, 1999).

Waisting, by itself, is not a problem of enamel formation but of dentin formation. We are not the first to have noticed abnormal contours in the enamel-dentin junction; nor the first to highlight the occurrence of broad depressed areas of enamel. In a study questioning the relationship between enamel hypoplasia and weaning, Blakey et al. observed wide, depressed areas of enamel, which they termed Major Growth Arrests (Blakey et al., 1994). Skinner and Goodman (1992) suggested that wide shallow enamel defects could be produced by sustained mild stress; they did not consider dentin. Similarly, Ensor and Irish (1995) observed what they termed "continuous chronic enamel hypoplasia" and captured the phenomenon with the concept of Total Hypoplastic Area, but did not report dentin involvement. However, Schwartz and Dean (2001) comment that the EDJ "…may undulate and is often sinuous, especially in regions where hypoplasias have affected the tooth" (p. 274). Tanya Smith's doctoral thesis (2004) illustrates (her Fig. 5.1) a broad depression in the outer enamel surface of a chimpanzee first molar metaconid with a corresponding undulation in the enamel-dentin junction.

We now think that Keith Dobney was the first to describe what we term "waisting" of dental crowns (Dobney and Ervynck, 1998). Considering the feature a special variety of LEH, termed a "depression," it was defined as a shallow or deep, horizontally oriented groove of the enamel with rounded margins usually found in the cervical half of the lingual crown surface of M1 and M2 in pigs (Witzel et al., 2006). The authors specifically remarked that the EDJ was affected and, interestingly, that there was an accompanying slight reduction in enamel thickness due to more closely spaced striae. Also of note is their observation that apparently unaffected control pigs had slight depressions in the same area (average depth = 105 μm) whereas diagnosed depressions averaged 347-μm deep. They suggested a threshold of 200 μm as appropriate for diagnosis of a "depression" feature (Witzel et al., 2006). Average depth of the waist in the Tai chimpanzees is 173 μm (excluding teeth with large cingula).

We do not know the cause of this condition. Etiology has at least two components: an agent, external to the infant chimpanzee, and the infant's own tissue responses; these can be termed ultimate and proximate levels of explanation for expression of the condition. We will consider both; but emphasize that our discussion is speculative.

Ultimate causation: the weaning process among infant chimpanzees

Given the reconstructed timing and span of the depression combined with what is known about chimpanzee infancy, we strongly suspect that waisting is linked to the weaning process. We consider weaning to be an extended and variable process of growing independence from the mother, increasing foraging efficiency, and decreasing reliance on lactation (Dirks et al., 2010). Clearly, weaning may involve psycho-social stress as the mother rejects attempts by the infant to nurse; a behavior which can manifest years before complete weaning

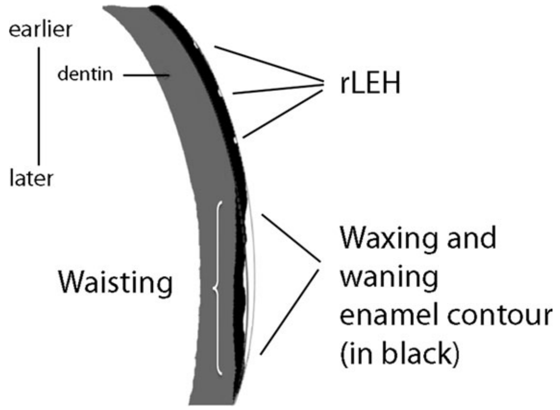

Fig. 5. Schema illustrating three varieties of developmental defect observed in Taï chimpanzee canine teeth: repetitive linear enamel hypoplasia (rLEH)—a short period surface phenomenon not including the dentin; waisting—a very long period depression of enamel contour including the dentin; undulations—intermediate periods of alternating thicker and thinner enamel. Lighter lines represent the contour of the unaffected outer enamel and EDJ surfaces.

has been accomplished. The following discussion shows that any one of these components of the weaning process could be implicated in waisting of the dental crown; nor are they mutually exclusive. Teasing apart these factors is not possible as yet.

There is a thoughtful analysis of accentuated enamel striae among baboon infants by Dirks et al. (2002) which examines the likelihood that weaning considered in all its social, nutritional, psychological and disease aspects results in abnormal enamel formation. They conclude that there is no easy characterization of the etiology of enamel defects like ‘weaning’ or ‘rainfall patterns’ but rather that each baboon experiences stress related to the interaction of individual life history and the environment (Dirks et al., 2002). We concur. We observed notable variation in the ages of onset and cessation of waisting, which we think reflects variation in the weaning process as experienced by individual animals.

Martin has observed that weaning is a singularly ill-defined term (Martin, 1984). Here we use the term “weaning" in a broad sense, to include the complete process of an infant chimpanzee’s achieving nutritional and psychological independence from the mother (Fletcher, 2001). According to Fletcher this includes: reducing contact and spatial proximity, reduced grooming of infant, dwindling suckling bouts, and full nutritional weaning. Defined this way “weaning" spans years and is not restricted to the rather abrupt resumption of estrus, new pregnancy and cessation of lactation which can occur quickly, over only six months or so in chimpanzees, marking an abrupt end to lactational weaning (Clark, 1977).

Dirks et al. (2010) recognize four dietary stages in the weaning process among baboons: prenatal intake, a period of exclusive suckling, a period of mixed feeding including both suckling and independent foraging, and finally, exclusive dependence on independent foraging. Unfortunately, we do not have field observations of this study’s chimpanzees during their infancy. At Taï, lactation lasts up to 4 years (Gagneux et al., 1999). In general chimpanzees are nutritionally weaned at age four (Mizuno et al., 2006). Chimpanzee infants at Gombe in East Africa are probably no longer nutritionally dependent on the mother’s milk by the fourth year (Clark, 1977) and wean on average at 5 years (Kennedy, 2005). Among six Gombe infants, suckling was last observed at age 5 years 3 months (range 50-86 months). In general, the literature provides a consistent picture of increasing weaning conflict between a persistent infant and a mother who is increasingly disinclined to breast feed, with nutritional consequences starting at age 3 years, intensifying and completing through the next 2 years during which time the mother resumes estrus, becomes pregnant, and ceases milk production. Postpartum amenorrhea in the mother ceases at just under 4 years of infant age. Inter-birth interval among Taï chimpanzees averages 5.76 years (Lathouwers and Elsacker, 2005); at Mahale 5 years 6 months to 5 years 8 months (Nishida et al., 2003).

Weaning and psychological stress

Basically, in our definition, weaning is a protracted process spread over a 2 to 4-year period (age 3–5+ years). The best description of the psychological effects of the weaning process among chimpanzees is from Gombe (Clark, 1977). Clark defines weaning as independence from the nipple. Nipple denial is very disturbing for the infant. Rejection by the mother can start in the second year of life but is infrequent until the resumption of estrus (average 3 years eight months) when rejections intensify. At this age, while nursing is infrequent, the infant will strive to prolong suckling behaviors as long as possible, stopping only when milk dries up with the next pregnancy. This occurs fairly abruptly and may produce emotional depression lasting a year or more (Clark, 1977). Both Dirks (Dirks et al., 2002; Dirks et al., 2010) and Maistripieri (2002) consider that “weaning" includes the stress for the infant which may accompany the mother’s resumption of estrus and conception.

Weaning and physiological stress

Thus far we have provided a short overview of the lactational and psychological aspects of weaning which might affect crown formation. Now we would like to discuss briefly the foraging aspect of the weaning process in which the infant chimpanzee becomes increasingly responsible for its own nutrition. We think it likely that a physiological problem sufficient to affect crown formation so obviously in some chimpanzee infants as seen in this series whose ages span the weaning period would have systemic effects and would be associated with growth faltering of the individual (King and Ulijaszek, 1999). Guatelli-Steinberg (2001) remarks that recently weaned juvenile primates may be more susceptible to malnutrition than are adults especially during periods of food scarcity. At Taï, there is a strong transmission of phenological knowledge from mother to infant. Boesch (1994) reports that the transmission of “nut-cracking behavior" which is only practiced in west African chimpanzees and takes a full 10 years to learn completely, reaches a peak at age three to 5 years among Taï mother-infant pairs. This is precisely the age spread during which we have observed coronal waisting to develop in some infant’s canine teeth. Notably, Byrne (1999) remarks that this technique of tool use may be the most difficult of such tasks for chimpanzees to learn.

Writing in 1990, Hiraiwa-Hasegawa stated that "Surprisingly, little is known about the development of independent feeding in any primate species…" (1990b). In her study of Mahale chimpanzee infants, she recorded the proportion (%) of suckling to foraging from birth to 5 years (numbers are approximate as they have been extracted from her graph 15.1); as follows:

Age (years)	0	0.5	1	2	3	4	5
%	96	34	28	22	36	16	0

Clearly infant chimpanzees start to forage at a very young age, years before lactational weaning. What is lacking from this otherwise very informative set of data is the actual protein and energy obtained through foraging as opposed to suckling. There is an important phase of solicitation for food objects by the infant and sharing by the mother amounting to ca. 20% of solid food intake up to age 2 years but dropping thereafter to only 5% or so; but that small percentage is mostly of hard to acquire, nutritionally rich items (Hiraiwa-Hasegawa, 1990a). Interestingly, she observes that the adult food repertoire is soon acquired and infants are familiar with most adult food types by age three. Infants from Mahale orphaned at less than 3 years old do not survive (Nishida et al., 2003). The rather sharp drop in suckling between 3 and 4 years of age may reflect the mother's initial efforts to discourage nipple contact (see above). Hiraiwa-Hasegawa also notes that infants may not be as equipped as adults to detoxify secondary plant compounds and so eat fewer mature leaves. Growing nutritional independence of the infant may it seems carry a physiological cost. Indeed, of three mortality peaks at Taï, the post-weaning deaths at age 5–6 years exceeded the social connectivity peak at 2.5 years (shared respiratory disease) and equaled the mortality peak at birth. Post-weaning deaths were attributed largely to malnutrition (Kuehl et al., 2008).

Malnutrition and tooth size

While it is generally acknowledged that growing teeth are under stricter genetic control than are growing bones and far less likely to show a reduction in adult size than is stature (reviewed in Bogin, 1999) there is less appreciation of good evidence from animal and human studies that teeth are quite sensitive to caloric and protein deficiencies. For example, rats fed a low protein diet comparable with that which produces kwashiorkor in humans showed atrophy of soft tissue organs and a 68% reduction in daily dentin apposition (Glick and Rowe, 1981). DiOrio et al. (1973) severely limited protein intake to suckling rat pups resulting in a 7% reduction in first molar occlusal area but with no effect on mineralization. A study designed to separate the effects of protein and calorie deficiency on tooth size using a pig model imposed a similarly severe dietary regime resulting in a reduction in mesio-distal length of third molars by 17.2% in protein deficient pigs and 32.6% in calorie deficient pigs (Luke et al., 1979). Enamel depressions in non-experimental pig molars, which we think comparable to 'waisting' in chimpanzee teeth, are attributed to undernutrition (Witzel et al., 2006). Clearly, severe malnutrition or under-nutrition makes for smaller teeth in animals. A more realistic level of malnutrition, in human subjects, is reported by Harris et al. (2001) who found that second generation (post-war) Chinese had significantly larger cheek teeth; although amounting to only1%, this is a relatively dramatic secular change in only one generation. They also cite a study in Sweden showing a 9% increase over only 160 years. There is good evidence from archaeological studies that reduced tooth size is linked to heightened risk of mortality. Guagliardo (1982) reported that the juvenile cohort at Averbach (a Late Mississippian site in Tennessee) had significantly smaller canines, first premolars and first molars, ranging from 3.64% to 1.6%, respectively. In conclusion, it would seem reasonable to conclude that dental crown waisting in Taï infant chimpanzees could reflect dietary quality. Ideally, with larger samples, one could measure cervical area of the crown in affected vs. unaffected teeth.

Proximate causation: dentin formation

The proximate etiology of waisting, at the tissue level, remains to be elucidated. Clearly, the coronal contour in affected teeth deviates temporarily from the normal cone shaped trajectory of a forming canine tooth in an inward direction; that is, towards the dentinal pulp. We see no obvious mechanism that would shift the epithelial cervical loop, where ameloblasts arise from the inner enamel epithelium (Radlanski and Renz, 2004), from its normal direction unless there is differential pressure on the inner enamel epithelium producing a net lower pressure on the pulpal side of the dental primordium. We can offer at best only a partial explanation, at the cellular level, for the waisting phenomenon. Because osmotic pressure is controlled by the protein content of blood capillaries in the dental pulp (Kim et al., 2008), one could speculate that a relative lack of plasma protein would reduce interstitial pulp pressure, thus providing a mechanism for waisting to occur. Equally, since dentin is composed primarily of collagen strengthened with mineral ions, especially calcium, a sheer lack of sufficient nutrients, could reduce the bulk of pre-dentin matrix (Linde and Goldberg, 1993). In either case, thin sections of affected teeth may be required to look for abnormal inter-globular spaces in the dentin (Dourda, 1985). Observations of enamel depressions in pigs (Dobney and Ervynck, 1998; Witzel et al., 2006) imply that an animal model to test these alternatives is available.

CONCLUSIONS

We have described the development of a mild "waist" in anterior teeth from seven of nine Taï chimpanzee individuals. It forms as an external, encircling depression in the contour of the outer enamel surface; more easily seen labially. It is not a thinning of enamel per se, but rather a slight decrement in dentinal crown volume, evident at the enamel-dentin junction, occurring between 3 and 6 years of age with maximum expression at about age 4.3 years. The timing and span of coronal waisting are consistent with descriptions of the weaning process at Taï and other chimpanzee study sites. Confirmation of a link between "waisting" and "weaning" will require a better understanding of the underlying developmental cause. We acknowledge that the coronal waisting could be due to just one aspect of weaning; e.g., socio-psychological stress, or inefficient foraging, and not linked to lactation at all. Nevertheless, we think that the significance of our observation will be threefold. First, we believe that the onset, severity and duration of the weaning process, as experienced by some individual chimpanzees, are permanently recorded in the shape of the anterior dental crown. Second, degree of coronal waisting linked to species characteristic weaning behaviors can be compared among apes; e.g., weaning among most mountain gorillas is characterized as "gradual and without overt conflict or infant distress" (Fletcher, 2001). Third, since hominoid anterior teeth are often preserved as fossils, this lesion may provide another means of investigating evolution of the derived human pattern of early lactational weaning, shortened inter-birth interval, and relatively helpless young who must depend on others in the social group to survive (Bogin, 1997; King and Ulijaszek, 1999; Gurven and Walker, 2006; Dean and Smith, 2009). In that we are reporting a newly recognized phenomenon, whose timing we think is clearly linked to infant development, we call for more detailed studies of the weaning process in nonhuman primates.

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