

# Developmental Defects in the Teeth of Three Wild Chimpanzees From the Tai Forest

Tanya M. Smith<sup>1\*</sup> and Christophe Boesch<sup>2</sup>

<sup>1</sup>Department of Human Evolutionary Biology, Harvard University, Cambridge, MA 02138

<sup>2</sup>Department of Primatology, Max Planck Institute for Evolutionary Anthropology, Leipzig, D-04103, Germany

**KEY WORDS** hypoplasia; accentuated line; stress; seasonality; life history

**ABSTRACT OBJECTIVES:** Developmental defects in teeth (accentuated lines and hypoplasias) have played a critical role in studies of childhood disease, nutrition, weaning, environmental variation, and early mortality. While these enigmatic structures have been lauded for their potential insights into human evolution, few studies have examined defects in individuals of known histories.

**MATERIALS AND METHODS:** Here we document defects in the molars of three wild juvenile chimpanzees from the Tai forest (*Pan troglodytes verus*) and compare them with behavioral, epidemiological, and environmental records.

**RESULTS:** Accentuated lines of differing intensities were found throughout molar crown and root growth, and were most common in a juvenile who demonstrated slow skeletal growth and prolonged maternal dependence. These defects were observed in association with some but not all injuries and disease outbreaks in this community. A

10-year record of accentuated line frequency across individuals shows a significant negative correlation with rainfall, but does not correlate with fruit availability or reveal significant annual trends. Several hypoplasias formed between ~0.6 and 5.8 years of age on molar crowns and roots of the three individuals, however, available behavioral and epidemiological records do not explain their causation.

**DISCUSSION:** While teeth may provide precise and accurate records of illness and trauma in some cases, inferring seasonal cycles, social stress, or weaning in living or fossil primate dentitions requires additional evidence beyond the presence, absence, or degree of expression of these defects. Studies that microsample bulk and trace elements may provide a more secure context for the interpretation of environmental, physiological, and dietary changes that impact dental tissue formation. *Am J Phys Anthropol* 157:556–570, 2015. © 2015 Wiley Periodicals, Inc.

## INTRODUCTION

Over the last two decades, an increasing number of studies have documented the presence of developmental defects<sup>1</sup> in tooth crowns and roots of fossil hominins (e.g., Skinner, 1996; Guatelli-Steinberg, 2003, 2004; Cunha et al., 2004; Guatelli-Steinberg et al., 2004; Lacruz et al., 2005; Smith et al., 2007a; Dean and Smith, 2009; reviewed in Smith, 2013; Hillson, 2014) and nonhuman primates (e.g., Guatelli-Steinberg, 2001; Dirks et al., 2002; Skinner and Hopwood, 2004; Guatelli-Steinberg and Benderlioglu, 2006; Schwartz et al., 2006; Chollet and Teaford, 2010; Dirks et al., 2010; Guatelli-Steinberg et al., 2012; Skinner et al., 2012; Skinner and Pruett, 2012; Skinner, 2014). External defects are known as hypoplasias, which may be pit-form, plane-form, or furrow-form disruptions (often termed linear enamel hypoplasias) (reviewed in Suckling, 1989; Goodman and Rose, 1990; Hillson, 1996; Hillson and Bond, 1997; Guatelli-Steinberg, 2001; Witzel et al., 2008; Hillson, 2014). Irregular (aperiodic) internal lines are often termed accentuated lines, accentuated striae, or less commonly “pathological lines” or “Wilson bands” (reviewed in Goodman and Rose,

1990; FitzGerald and Saunders, 2005; Witzel et al., 2008; Hillson, 2014) (Fig. 1).

Numerous causes have been suggested for the formation of hypoplasias, including illnesses such as rickets, scurvy, measles, smallpox, diabetes, allergies, syphilis, as well as fever, diarrhea, nematode infection, viral or bacterial inoculation during childhood, malnutrition, vitamin A or D deficiency, and direct pressure on the developing tooth germ (Suckling, 1989; Goodman et al., 1991; Hillson, 1996; Dirks et al., 2002; Guatelli-Steinberg and Benderlioglu, 2006; reviewed in Hillson, 1996; Skinner et al., 2012; Hillson, 2014). Others have suggested that enamel hypoplasias in wild nonhuman primates may be related to seasonal patterns of rainfall, resource availability, temperature, and/or

Additional Supporting Information may be found in the online version of this article.

Grant sponsor: Max Planck Society, Harvard University, National Science Foundation, Radcliffe Institute for Advanced Study, and the Wenner-Gren Foundation.

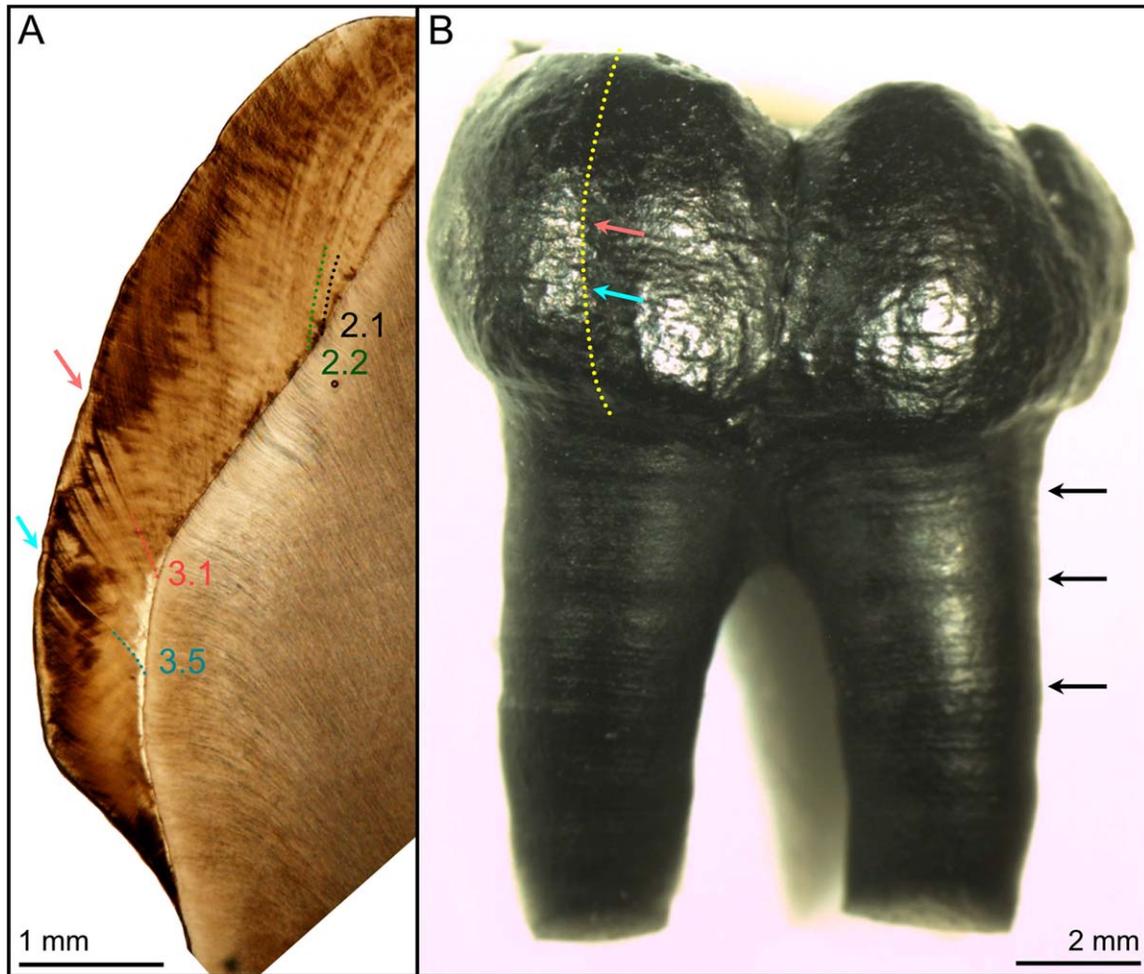
\*Correspondence to: Tanya M. Smith, Department of Human Evolutionary Biology, Harvard University, 11 Divinity Avenue, Cambridge, MA 02138, USA. E-mail: tsmith@fas.harvard.edu

Received 2 July 2014; revised 24 February 2015; accepted 25 February 2015

DOI: 10.1002/ajpa.22741

Published online 27 March 2015 in Wiley Online Library (wileyonlinelibrary.com).

<sup>1</sup>The term “developmental defect” refers to an atypical structural variant formed during dental development, in contrast to “stress,” which may imply that the defect is of known etiology. The term “stress” has been used by scholars in numerous ways (reviewed in Temple and Goodman, 2014), and its use in this article reflects that of the specific work referenced. See Hillson (2014) for a recent review of the terminology of developmental defects in teeth.



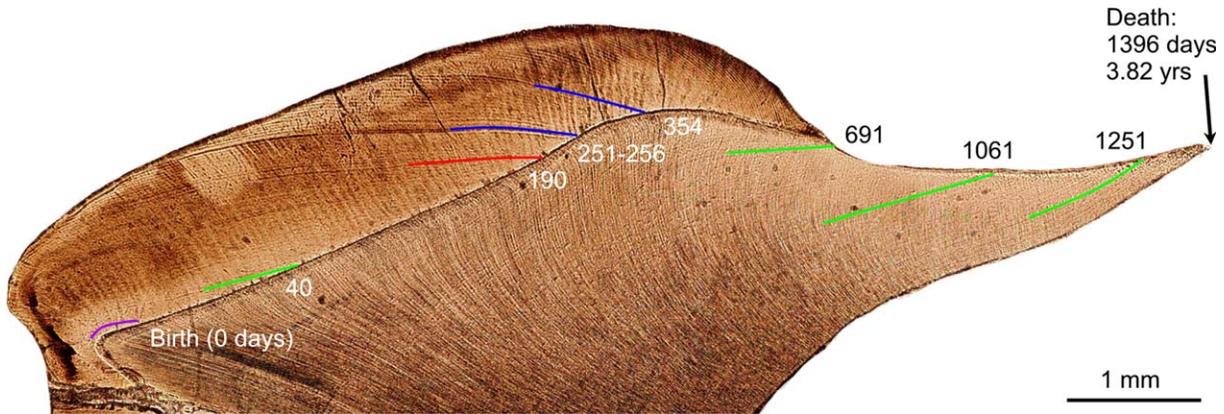
**Fig. 1.** Accentuated lines and hypoplasias in the crown and root of the lower M2 of wild chimpanzee Lefkas. **A**) Histological section showing accentuated lines (dotted lines) in the enamel at 2.1, 2.2, 3.1, and 3.5 years of age. **B**) Cast of the same tooth illustrating the approximate plane of section (yellow dotted line), the presence of two marked hypoplasias corresponding to accentuated lines at 3.1 and 3.5 years (pink and blue arrows, respectively), and several root hypoplasias formed after 4.5 years of age (black arrows). [Color figure can be viewed in the online issue, which is available at [wileyonlinelibrary.com](http://wileyonlinelibrary.com).]

tropical disease cycles (e.g., Skinner, 1986, 2012; Swindler and Beynon, 1993; Skinner et al., 1995; Guatelli-Steinberg and Skinner, 2000; Dirks et al., 2002; Skinner and Hopwood, 2004; Chollet and Teaford, 2010; Skinner and Pruetz, 2012). For example, Skinner and Hopwood (2004) hypothesized that seasonal moisture cycles in the tropics led to semianual or annual hypoplasias in great ape anterior teeth, possibly due to increases in parasitic diseases and/or malnutrition. Chollet and Teaford (2010) compared hypoplasia frequencies in wild *Cebus apella* canines from 54 sites in Brazil collected between 1909 and 1976, and found significant differences in hypoplasia frequency among habitat types, as well as between groups with different mean annual temperatures. Surprisingly they did not find a relationship between hypoplasia frequency and the number of dry months or variation in rainfall amounts. Similarly, Skinner (2014) recently argued that differences in hypoplasia occurrence between orangutan species reflect ecological differences.

Skinner et al. (2012) described gross developmental defects termed “coronal waisting” in the anterior teeth of several Tai forest (Ivory Coast) chimpanzees, which they

suggested are structurally distinct from more spatially discrete linear enamel hypoplasias. These broad growth disruptions were estimated to occur between ~3 and 6 years of age, with durations ranging from ~2 to 4 years, which these authors ascribe to the weaning process, although the nursing histories of these individuals were unknown. Weaning has frequently been invoked as a potential cause of more traditionally-defined hypoplasias, although evidence from populations with historic records or behavioral histories is often contradictory (e.g., Goodman et al., 1987; Blakey et al., 1994; Moggi-Cecchi et al., 1994; Katzenberg et al., 1996; Skinner, 1996; Wood, 1996; Humphrey, 2008). Several of these studies infer that weaning is the cause of hypoplasias due to the position of hypoplasias on tooth crowns and their presumed age at formation. A direct association between hypoplasias and observed or documented post-natal dietary transitions has yet to be demonstrated.

Relatively little is known about the causes of accentuated line formation in humans, save for the neonatal line associated with birth (e.g., Rushton, 1933; Schour, 1936; Whittaker and Richards, 1978; Eli et al., 1989),



**Fig. 2.** Accentuated lines in the enamel and dentine of the mesiolingual cusp of the upper M1 of wild chimpanzee Piment. The neonatal (birth line) is indicated in purple, four minor accentuated lines are in green (40, 691, 1,061, and 1,251 days of age), one moderate accentuated line is in red (190 days of age), and two major accentuated lines are in blue (251–256 and 354 days of age). The major accentuated line at 354 days corresponds to a hypoplastic pit in the mesiobuccal cusp (Supporting Information Fig. 1). The two minor lines in the dentine at 1,061 and 1,251 days of age are evident as root hypoplasias (not shown). Modified from Smith and Tafforeau (2008). [Color figure can be viewed in the online issue, which is available at [wileyonlinelibrary.com](http://wileyonlinelibrary.com).]

postnatal immunizations (Birch and Dean, 2014), and parturition in females who are still growing their third molars (Dean and Elamin, 2014). Studies of nonhuman primates have emphasized potential relationships between accentuated lines and rainfall patterns, changes in nutrition, social disruptions, poor physical growth, disease, physical trauma, weaning, and maternal life history events (Bowman, 1991; Macho et al., 1996; Dirks, 1998; Dirks et al., 2002; Schwartz et al., 2006; Dirks et al., 2010; Smith, 2013; Austin et al., in review). Macho et al. (1996) examined accentuated lines in 11 molars of the fossil monkey *Theropithecus oswaldi*, and suggested that annual seasonal signals were apparent in the timing of accentuated lines, as well as shorter intervals that may reflect the onset of the rainy season and the peak of the dry season. Dirks et al. (2002) compared accentuated lines in two wild female baboons from Ethiopia to concurrent rainfall patterns, and reasoned that certain lines may correspond to postweaning wet and dry seasons, although they concluded that it was not possible to decouple rainfall trends from potential life history events. Dirks et al. (2010) combined studies of accentuated lines with trace element analysis of two baboons from Uganda, and hypothesized that accentuated lines at certain ages indicated physiological stress due to a reduction or cessation of suckling.

While numerous studies have speculated on the etiology of external hypoplasias or internal accentuated lines in teeth, few have examined these structures in nonhuman primates with documented developmental histories, and in most cases these individuals were from captive or semi-captive environments (Bowman, 1991; Dirks et al., 2002; Guatelli-Steinberg and Benderlioglu, 2006; Schwartz et al., 2006; Smith, 2013; Austin et al., in review). Guatelli-Steinberg and Benderlioglu (2006) documented a significant decrease of hypoplastic defects in rhesus macaques in the Cayo Santiago community once provisioning began. Schwartz et al. (2006) illustrated how the history of a captive gorilla was precisely reflected by accentuated lines, including an eye injury and surgery, subsequent hospitalizations, and several enclosure transfers before its accidental death. Similarly, Smith (2013) documented a series of accentuated lines in a juvenile macaque that

appeared to correspond to an enclosure transfer, leg injury and subsequent hospitalization, and a series of bouts of dehydration/diarrhea that led to multiple hospitalizations and eventual euthanasia. Elemental and molecular analyses of this individual's first molar revealed additional developmental disruptions that are not evident from light microscopy (Austin et al., in review).

Although these studies strongly suggest that a causative relationship exists between poor nutrition, injury, illness, and developmental defects, they do not clarify the potential impact of environmental variation, social stress, or disease outbreaks in natural environments, which are frequently invoked as additional causative agents. Here we provide the first examination of development defects in wild primates with associated environmental, epidemiological, and behavioral information. We compare patterns of hypoplasias and accentuated lines in three wild chimpanzees from the Taï forest with rainfall patterns, fruit availability, illnesses, and specific events within the community over a 10-year period. In particular, we aim to assess suggestions that the timing of developmental defects may be used to infer seasonality in rainfall patterns, resource availability, and/or disease cycles. While results from three individuals require additional validation in order to draw firm conclusions about the causation of development defects, we also present these findings in order to stimulate additional histological studies of wild primates of known developmental histories.

## MATERIALS AND METHODS

We assessed developmental defects in five first and second molars (M1 and M2) of three known-age juvenile wild chimpanzees (*Pan troglodytes verus*) from the Taï forest (Côte d'Ivoire) that formed between February 1986 and April 1996. Histological sections were prepared from crown complete molar teeth (Piment: M1; Goshu: M1 and M2; Lefkas: M1 and M2), and crown formation time was assessed from counts and measurements of incremental growth lines as detailed in Smith et al. (2010). First and second molars were temporally registered through accentuated line patterns (illustrated in Smith et al., 2010: Fig. 1, p. 366), yielding a continuous record of development

beginning before birth and continuing until 4.5 to 6.4 years of age for Lefkas and Goshu, respectively.

Accentuated lines were spatiotemporally mapped, and their expression was categorized as minor, moderate, or major.<sup>2</sup> This qualitative characterization was based on the appearance of lines in low and medium resolution overviews created with 4× and 10× objective lenses (Fig. 2). Minor accentuated lines were identified as subtle lines that could be spatiotemporally distinguished from regular periodic incremental growth lines and/or areas of homogeneous mineralization (assessed with polarized light). Major accentuated lines were identified as marked structural variations in enamel or dentine, which often manifest as dark bands (encompassing more than 1 day of formation), representing a marked change in hard tissue mineralization. Moderate accentuated lines were defined as intermediate developmental disturbances in comparison to minor and major accentuated lines. Defect occurrences were converted to specific chronological ages and dates by adding postnatal developmental time to the date of birth.

High-resolution casts of all molars were examined for hypoplasias with stereomicroscopy, and ages were determined for each defect when possible through registry with the corresponding histological section. Casts of the upper central incisors of Goshu were also examined. Hypoplasias identified on casts and in thin sections were classified as one of two common structural variants: linear (furrow-form) disruptions or pit-form defects. Linear defects appear as circumferential depressions on the crown or root surface, and are the most common type of hypoplasia (Hillson and Bond, 1997). Pit-form hypoplasias are localized pits or depressions of the crown surface that may be singular or multiple (illustrated in Hillson and Bond, 1997: Fig. 7, p. 99).

We also include preliminary observations of hypoplasias from computed tomographic (CT) scans of two jaws, which were identified as deviations in the normal curvature of the outer enamel surface. Scans were conducted with the BIR Actis 300/225 laboratory microtomographic scanner, and virtual sections were created for the upper dentition of Piment (voxel size 109 microns) and the lower dentition of Lefkas (voxel size 27 microns) according to established protocols (Smith et al. 2007b). Although Skinner et al. (2012) have successfully employed micro-CT imaging to illustrate developmental defects at the enamel-dentine junction, laboratory grade micro-CT does not allow for the temporal quantification of defects or the detection of minor defects, as is the case with synchrotron micro-CT (Le Cabec et al., in press). Although limited, we have included observations from CT scans in order to present a more comprehensive assessment of hypoplasias in virtual sections of unerupted teeth (which cannot be classified as pit-form or linear hypoplasias without higher resolution images of entire tooth surface).

Defect formation dates were subsequently compared with field notes on maternal behavior, disease out-

breaks, and infant injuries (Table 1) (Boesch and Boesch-Achermann, 2000; unpublished notes), as well as rainfall patterns and fruit availability (Polansky and Boesch, 2013). Due to the fission-fusion social structure of this community (Boesch and Boesch-Achermann, 2000), certain individuals were seen more frequently than others, and individual health state, disease, and injuries were recorded whenever individuals were observed. Events producing visible behavioral changes or morphological alterations were systematically recorded, but field conditions prohibited more subtle characterizations of community dynamics or consistent monitoring of all members. Similarly, information on hormone levels and parasite loads are not available during our study period (March 1986–April 1996). Rainfall data span the majority of the study period from August 1987 through March 1996, while fruit phenology data (% trees with fruit) postdate the period captured in the molar crowns, as systematic fruit abundance quantification began in 1997 (Anderson et al., 2005). Variation in fruit abundance across years appears to be lower than variation across seasons within a year, thus an aggregate of data from 1998–2012 was employed to approximate earlier seasons patterns. Assessments of fruit availability have been shown to relate to individual energy balance and protein gain in chimpanzees (N'guessan et al. 2009).

We acknowledge that defect records in teeth may not precisely match potential causative events due to slight errors in the estimation of ages, as well as the fact that hypoplasias and accentuated lines do not necessarily manifest as discrete or distinct events. Hillson (2014) suggested that defect formation may be the result of progressive events, such as developing an illness or experiencing malnutrition, for which the negative consequences may be the result of a sustained period of physiological stress in excess of 1 day. We suspect that the ages assigned to defects in our study subjects reflect causative events to within a few weeks, as estimated ages at death for Piment and Goshu differ from field notes by less than a month (Smith et al., 2010). (Lefkas' age at death could not be estimated histologically, but was known from field notes.) Thus our interpretations of events such as injuries or disease outbreaks include consideration of potential defects in the month before and after the event of interest was observed to have occurred.

The nonparametric Jonckheere–Terpstra test for trends was employed to assess seasonal signals in defect frequency (testing minor, moderate, and major defects independently, as well as cumulatively). We also tested for correlations between defect frequency and rainfall, as well as defect frequency and fruit availability, using the Pearson product-moment correlation coefficient and IBM SPSS (v. 21) software. Two linear regression tests were also conducted using R (v. 2.14, R Development Core Team, 2011): ordinary least squares (OLS) with cumulative monthly defect frequency as the dependent variable, and monthly rainfall and fruit availability as the independent variables; and a mixed effects model with the degree of defect (minor, moderate, major) as the dependent variable, and rainfall and fruit availability as the independent variables. Given the limited number of individuals and the potential nonindependence of defects within these individuals, we interpret nonsignificant results with caution due to limited statistical power (potential Type II error).

<sup>2</sup>Others have chosen to similarly distinguish the degree of defect expression qualitatively (e.g., Skinner and Hopwood, 2004), as there is no consensus about the appropriate threshold for the diagnosis of developmental defects in teeth (Hillson, 2014). We chose to follow the logic of Skinner and Hopwood (2004) to provide a comprehensive assessment of developmental defects, including those that may be considered relatively minor, in order to try to assess their causation.

TABLE 1. Observational data on three wild chimpanzee juveniles, their mothers, and community level disease outbreaks

Month	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
January		Goshu observed to have monkey pox, appeared emaciated									
February						Piment born					
March			Goshu born								
April							Goshu attacked by a leopard, dies				
May											
June											
July											
August											
September					Community showed runny noses (rhinorrhea)	Goshu's brother born; Piment's mother engaged in aggressive behavior against group females		Lefkas' foot first observed to be missing two toes, injury likely 1 to 2 months prior	Lefkas' mother showed discolored skin patches for 2 weeks		
October						Lefkas born; Piment's mother continued aggression and became alpha female; community showed runny noses (rhinorrhea)	Ebola outbreak				
November											
December							Lefkas' sister died, mother appeared distressed				End of record for Lefkas

TABLE 2. Accentuated lines in the molar crowns of three wild chimpanzee juveniles

Month	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
January			G moderate				G major	P minor, L minor	P minor, L minor	L moderate	
February						Piment (P) born	P major	L moderate L minor	L moderate	L 2 moderate	
March	Goshu (G) born					P minor			L minor		
April	G moderate		G minor		G moderate			L moderate	L minor	L major	End of record for Lefkas
May	G minor		G minor	G minor			L major & minor		L moderate	L 2 moderate	
June							L minor	L moderate			
July		G moderate			G moderate	G moderate	L minor		P minor		
August		G major	G moderate	G moderate	G moderate	P moderate	Goshu dies				
September						Lefkas (L) born;	L major		L minor		
October						P major	L minor				
November						Lefkas major	L 2 minor	L major	Piment dies; L moderate	L minor	
December								L moderate	L 2 moderate		

Months with minor lines are noted but not highlighted, those with moderate lines are highlighted in light gray, and months with major lines are highlighted in dark gray. When multiple day events overlapped two successive months they are indicated for the month that included the majority of dates. G = Goshu, P = Piment, L = Lefkas. Months with births and deaths are indicated for each individual. Note Goshu showed several undated minor accentuations between 29 August, 1989 and 24 July, 1990.

TABLE 3. Accentuated line frequencies during the first 4 years of life

Individual	0–1 yrs	1–2 yrs	2–3 yrs	3–4 yrs	Total
Piment	3	2	1	1 <sup>a</sup>	7
Goshu	2	3	4	3	12
Lefkas	4	8	7	10	29
Total	9	13	12	14	

Data from Table 2 (summation of minor, moderate, major classes).

<sup>a</sup>Piment died at 3.8 years of age, and thus the 3 to 4-year category is missing the final 3 months.

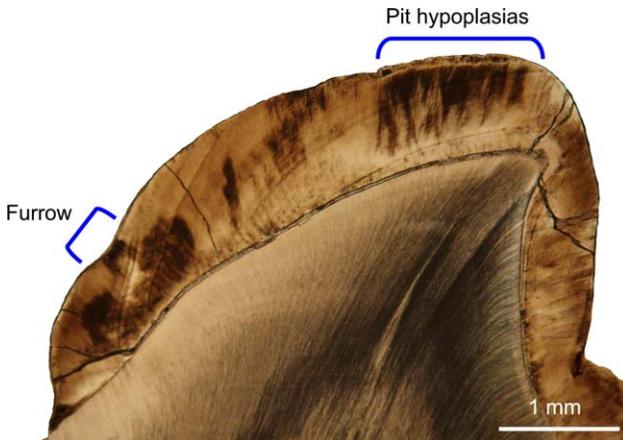


Fig. 3. Lower M1 mesiolingual cusp showing atypical enamel formation in Lefkas. Pit hypoplasias were originally identified on casts; they are shown here in cross section in the upper lateral enamel and were estimated to have formed between 0.6 and 0.9 years of age. Matching accentuated lines in the mesiobuccal cusp suggests that the hypoplastic furrow defect in the lower lateral enamel formed between 1.3 and 1.5 years of age. [Color figure can be viewed in the online issue, which is available at [wileyonlinelibrary.com](http://wileyonlinelibrary.com).]

## RESULTS

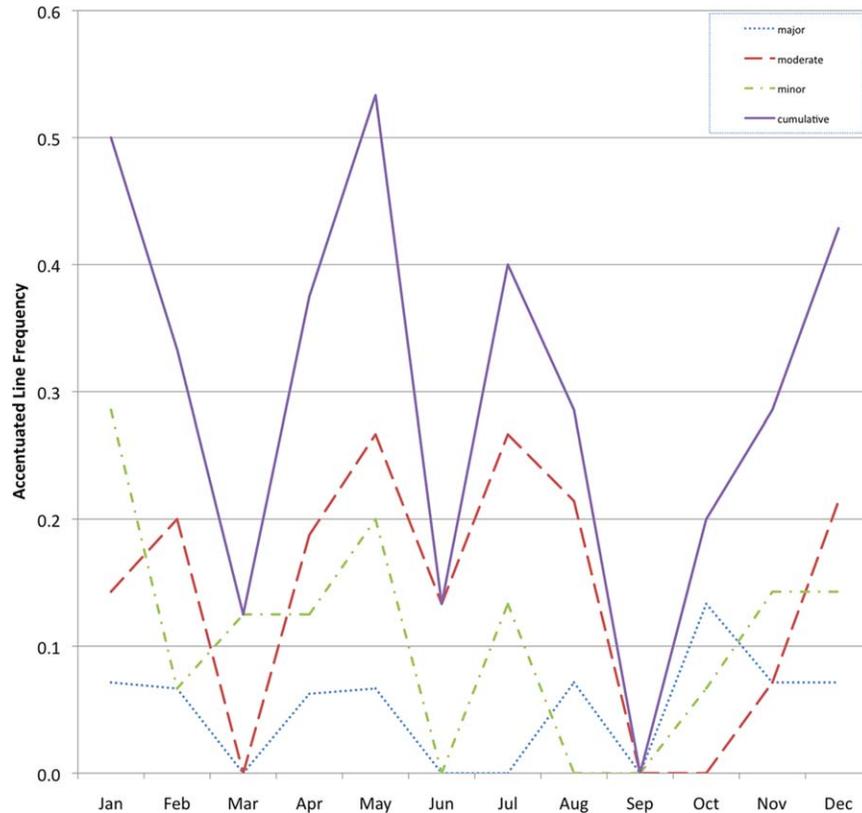
Out of a total of 178 months sampled across the three individuals, we observed a minimum of 53 internal accentuated lines within 46 of these months (Table 2). Piment, a female born in February 1991 who died of Ebola at 3.8 years of age, showed a minimum of seven internal accentuated lines (four minor, one moderate, two major; Fig. 2). The first major defect formed between 251 and 256 days of age, which was during a month when other chimpanzees in the community were observed to have rhinorrhea (runny noses) for approximately 10 days (although direct observations of Piment were not made during this period). A second major line at 354 days of age corresponded with an isolated pit-form hypoplasia on her upper M1 mesiobuccal cusp (Supporting Information Fig. 1). Field notes do not contain information that coincides with the timing of these defects. Another pit-form hypoplasia was also observed on her M1 distobuccal cusp (Supporting Information Fig. 1) and broad hypoplastic defects were observed on the surfaces of her anterior and premolar teeth (Supporting Information Fig. 2). Piment's mother (Poupee) was observed to be aggressively displaying to other females when Piment was 7 to 8 months of age, at which time she became the dominant alpha female; Piment did not

show any obvious defects between the accentuated lines at 190 and 251 days of age. Poupee was believed to have a problem with lactation, as 4 years before Piment was born she was seen with an abscess in her left breast possibly due to mastitis. Moreover, three infants born before Piment died by or before they reached two years of age. Piment had fewer internal accentuated lines than either of the other two individuals during their first 4 years of life (Table 3), but she did show proportionally more accentuated lines from 0 to 2 years of age than from 2 to 3.8 years of age.

A second female individual (Goshu) was born in March 1986 and appeared to have died of a leopard attack at 6.4 years of age, showing a minimum of 16 internal accentuated lines (four minor, 10 moderate, two major). The two major lines formed at 1.4 years (illustrated in Smith et al., 2010: Fig. 1, p. 366) and 5.8 years of age. The first event corresponded to a hypoplasia that encircled the M1 crown near the cervix. The second event lasted approximately 14 days, and correlated with a marked hypoplastic defect on the M2 root surface (Supporting Information Fig. 3). Field notes do not include information that coincides with the timing of these lines. Broad hypoplastic defects were observed on casts of the upper central incisors that resembled the "coronal waisting" defects described by Skinner et al. (2012), although it was not possible to assign ages to these regions due to poor preservation of long-period growth lines (perikymata). Field notes suggest that Goshu contracted monkey pox at 10 months of age, although there were no marked accentuated lines for several months before or after this episode. Goshu's mother, low-ranking Goma, began mating when Goshu was approximately 2 years of age. Her next infant was born when Goshu was 5.5 years old, and Goshu did not show any obvious defects at this age. Goma demonstrated some atypical nursing behavior with subsequent offspring, which included carrying, nursing, and nesting with an older son (Gargantua) and a newborn infant (Gisele) for 2 months. It is not clear if Goshu experienced a protracted weaning process of this nature, but it is highly likely that she was fully weaned before her death at 6.4 years of age.

Our final juvenile is the male Lefkas, who was born in October 1991 and died of pneumonia at 7.6 years of age. Thirty accentuated lines were identified in his lower M1 and M2 crowns, covering the first 4.5 years of his life; subsequent development could not be tracked into the M2 roots due to histological section obliquity and poor preservation of long-period growth lines (periradicular bands). During this period, Lefkas' teeth revealed five major accentuated lines, 14 moderate lines, and a minimum of 11 minor lines. Major accentuated lines occurred at 0.2, 0.6, 1.0, 2.1, and 3.5 years of age. Numerous small pit-type hypoplasias were noted on the upper 1/4 to 1/3 of several cusps of his M1, which formed between ~0.6 and 0.9 years of age (Fig. 3). A broad furrow hypoplastic defect was observed on the lingual side of the crown (but not the buccal side) at ~1.4 years of age. The M2 crown showed two hypoplasias at 3.1 and 3.5 years of age, followed by several hypoplasias on the roots after age 4.5 (Fig. 1). Gross developmental defects were also noted in micro-CT scans of his lower incisors and premolars, but not on the lower canines or molars (Supporting Information Fig. 4).

At approximately 1 year of age, Lefkas was noted to be small compared with similarly-aged infants. He showed slow physical development for the next few



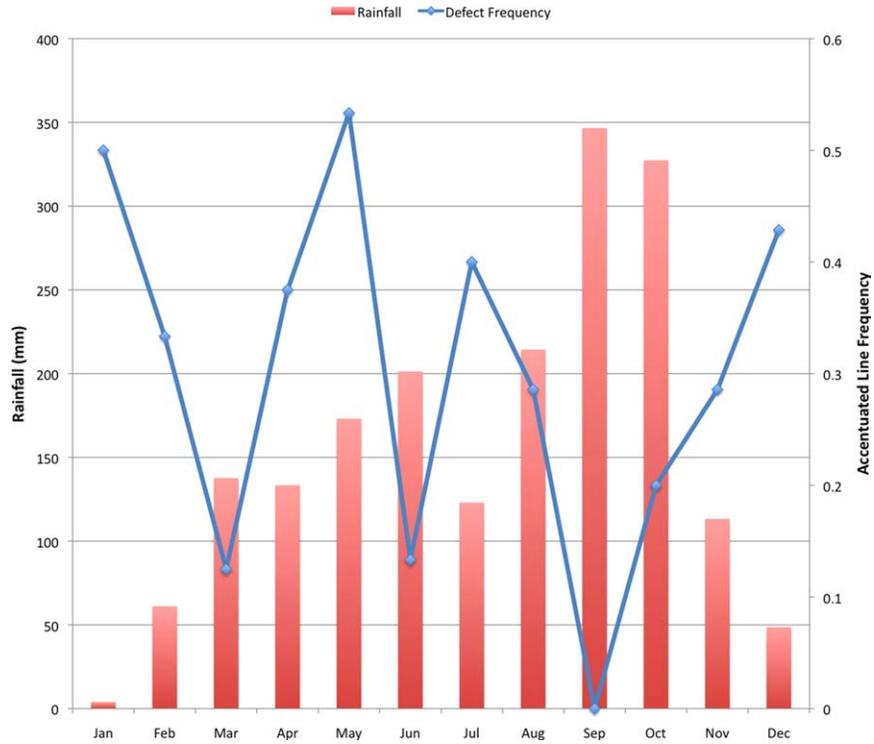
**Fig. 4.** Monthly frequency of accentuated lines from 1986 to 1996. Each calendar month is represented by 14 to 16 summed months among the three individuals. Frequencies were calculated as the number of events during a particular month divided by the total number of months that developmental data were available (Table 1). For example, in all Januarys between 1987 and 1996, seven events were observed in all individuals over the 14 total months that one or more individuals were sampled, thus the cumulative frequency of January defects is 7/14 or 0.5. [Color figure can be viewed in the online issue, which is available at [wileyonlinelibrary.com](http://wileyonlinelibrary.com).]

years of his life, until he appeared to have caught up with his peers by about age four. Lefkas was 13 months of age when his older sister (Lychee) died, which appeared to impact the behavior of his high-ranking mother Loukoum. His lower M1 showed accentuated lines that month and during each of the subsequent 3 months. At 22 months of age Lefkas' right foot was observed to be missing two toes. While the injury that led to the loss of toes was not observed, it was estimated to have occurred when the community was widely scattered and difficult to follow 1 to 2 months earlier. A moderate accentuated line (band) lasting approximately four days was recorded at 20 months of age, which may relate to the original injury as there were no other discernable defects between 20 and 24 months of age. Shortly before Lefkas turned 3 years old, his mother was observed to show several discolored skin patches that remained evident for 2 weeks, although Lefkas was not reported to have had similar skin problems nor did he show accentuated lines that month. Lefkas' younger sibling was born at the end of August 1997 (after the period of development recorded in his M1 and M2 crowns), and he continued to beg for food from his mother at this time and periodically until his death at 7.6 years of age.

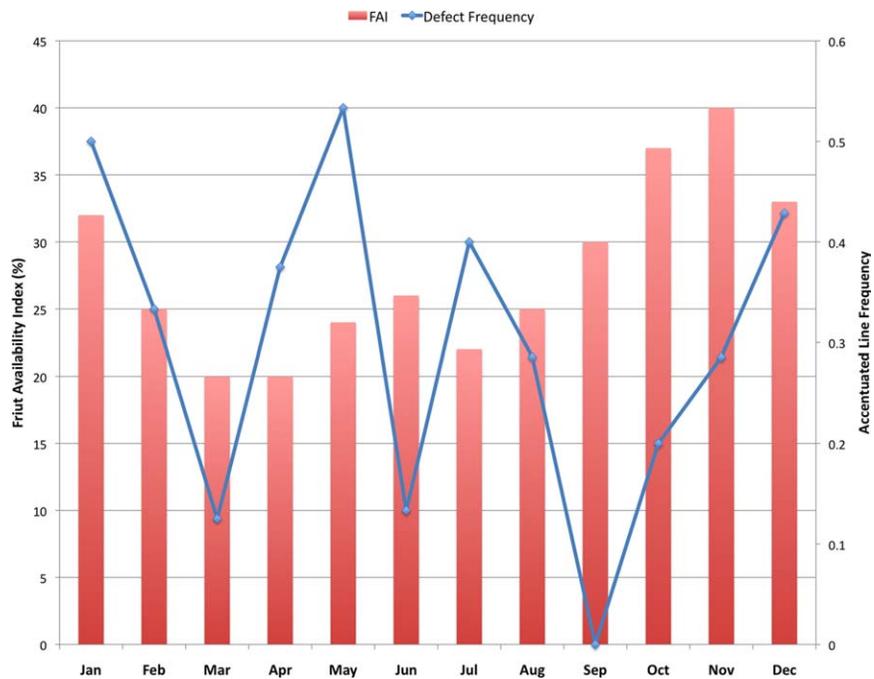
We combined data on accentuated lines from the three individuals to examine potential seasonal signals. The frequency of minor, moderate, and major lines was broadly consistent across months (Fig. 4); no significant

annual trends were apparent for any individual accentuated line class or for the cumulative frequencies of all defects ( $P > 0.05$ ). Accentuated lines were most common during January, May, July, and December. No accentuated lines were apparent in September. In order to decouple potential physiological disruptions associated with early life diet transitions, we excluded data from the first 2 years of life for all three individuals and found the same pattern of high frequencies in January, May, July, and November/December. (A similar lack of annual trends in accentuated line frequencies was also found when data for the first 2.5 years of life were excluded.) Thus it does not appear that there are marked ontogenetic differences in the overall patterns of accentuated line formation of these individuals, and data from all ages for the three individuals were used in subsequent analyses.

Comparisons of cumulative defect frequencies with rainfall patterns during the study period (Fig. 5; Supporting Information Table 1) showed a significant negative correlation ( $r = -0.690$ ,  $P < 0.05$ ). Fewer defects were observed during the three months with the greatest rainfall (August–October) than the 3 months with the least rainfall (December–February) (7 vs. 16 defects). A comparison of cumulative defect frequency with average fruit availability (Fig. 6) did not reveal a significant correlation ( $r = 0.059$ ,  $P > 0.05$ ); the 4 highest months of fruit availability (October–January) showed similar numbers of accentuated lines as the 4 months with the lowest fruit availability (March–May, July) (20 vs. 22 defects).



**Fig. 5.** Cumulative accentuated line frequency and rainfall patterns in the Tai forest. Rainfall data span August 1987 to March 1996 (from Polansky and Boesch, 2013). [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]



**Fig. 6.** Cumulative accentuated line frequency and fruit availability patterns in the Tai forest. Fruit availability data span 1998–2012 (from Polansky and Boesch, 2013). [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

For the OLS model, rainfall was found to be significantly negatively associated with cumulative defect frequency ( $b = -0.001075$ ,  $P = 0.018$ ; while controlling for fruit availability), but there is no evidence of association between fruit availability and cumulative defect frequency ( $b = 0.000234$ ,  $P = 0.969$ ; while controlling for rainfall). (Here the lowercase "b" stands for the slope value; for example a 1 mm increase in rainfall is associated with a  $-0.001075$  reduction in the average cumulative defect frequency.) Adding in an autoregressive term for the residuals did not significantly improve the fit of the model, suggesting that there is no autocorrelation in the data related to the temporal trends. For the linear mixed model, rainfall was again found to be negatively associated with defect frequency ( $b = -0.000357$ ,  $P = 0.0198$ ; while controlling for fruit availability and defect class), but there was no evidence of a significant association between fruit availability and defect frequency ( $b = -0.000074$ ,  $P = 0.9715$ ; while controlling for rainfall and defect class).

## DISCUSSION

This is the first report of accentuated lines and hypoplasias of known calendar dates and chronological ages in wild chimpanzees, which spans almost 15 combined years of developmental histories between 1986 and 1996. Our study underscores the challenge of relating defects to particular events in the histories of wild juvenile primates (Dirks et al., 2002, 2010). Few populations of primates are monitored consistently or continuously over extended time periods, and even fewer field sites have associated dental remains from securely identified community members. Studies of accentuated lines have been limited to small numbers of teeth due to the semidescriptive and time-intensive nature of preparing and analyzing histological sections, as well as the challenge of obtaining teeth from individuals with associated prospective developmental information.

Assessments of hypoplasias in wild primates have not been subject to the same limitations as studies of accentuated lines, as it is not necessary to section teeth to determine the presence of external defects in erupted teeth, facilitating access to larger samples that may be assessed rapidly. While this approach may be applied to characterize defects within and across populations, it is difficult to precisely determine the age of formation of hypoplasias, as well as the duration between multiple hypoplasias. Long-period increments expressed on the tooth surface (perikymata) show a consistent periodicity (number of days between lines) within individuals, but vary among individuals (e.g., chimpanzee periodicities have been reported to range from 5 to 9 days: Schwartz et al., 2001; Smith et al., 2010). This implies that the same number of long-period lines on the surface of different individuals' teeth may represent an almost two-fold difference in developmental time. Studies that employ a single estimated value for the long-period line periodicity in order to calculate the timing between defects are likely to be fairly imprecise due to normal variation in long-period line periodicity.

### Relationship of accentuated lines to hypoplasias

An important result of this study is the finding that moderate or major accentuated lines often underlie both pit-type and linear hypoplasias in molar crowns and roots (also see images in Witzel et al., 2008; Hillson,

2014). This is in contrast to Simpson (1999), who stated that accentuated lines and hypoplasias have "a different structural and temporal signature, suggesting that they are the products of physiological disruptions with different courses, timings and durations" (p. 259). Of the six hypoplastic defects that could be precisely aged with a reasonable degree of confidence, hypoplasias in Piment and Goshu were observed with underlying major accentuated lines (Supporting Information Figs. 1 and 3), and multiple hypoplasias in Lefkas' molars also showed underlying marked accentuated lines (Figs. 1 and 3). Accentuated lines appear to be more sensitive recorders of developmental disruptions than hypoplasias, particularly in molar teeth, and it is often easier to assign precise ages to these internal defects.

Other methodological considerations may also impact the precision of studies of accentuated line and hypoplasia frequency and timing. Accentuated lines vary in their clarity and degree of expression across teeth and among molar cusps of a single tooth (also see Hillson, 2014), which may be partially due to the orientation of the plane of section and the geometry of the tooth crown. (In the current study we quantified accentuated lines in the cusp from each molar that showed the least section obliquity and the most well-defined lines.) Similarly, both Lefkas and Piment showed variable hypoplasia expression on different sides of their M1 crowns (a phenomenon also noted by Hillson, 2014), complicating interpretations of potential causative events. Hypoplasia expression also varies across simultaneously forming teeth, which is particularly evident in comparisons of Lefkas' lower dentition (Supporting Information Fig. 4). Hillson (2014) and others have noted that linear hypoplasias are less common on postcanine teeth than on incisors and canines, which is partially supported by our CT scan results, and impacts interpretation of the results of the current study. Although a number of studies have assessed the frequency and/or location of hypoplasias to infer physiological stress during development, further investigation into the etiology and variation of these defects is warranted to understand the "developmental threshold" that leads to hypoplastic defect formation in some teeth and marked accentuated lines in others (Witzel et al. 2008).

### Rainfall, illness, and mortality

The results of this study do not support suggestions that developmental defects in wild primate dentitions are positively associated with moisture cycles or rainy seasons (contra Skinner, 1986; Macho et al., 1996; Dirks et al., 2002; Skinner and Hopwood, 2004). We found a significant negative correlation between rainfall and accentuated line frequency, and this association remained significant when defect class and/or fruit availability were held constant. Annual or semiannual trends were also not apparent in the timing of hypoplastic defects on the three individual's molars. Piment's molar showed two pit-type hypoplasias before 1 year of age with no subsequent hypoplasias, Goshu's molars showed two events at 1.4 and 5.8 years of age, and Lefkas' molars showed defects at  $\sim 0.6$  to 0.9, 1.4, 3.1, and 3.5 years of age. Although it remains possible that developmental defects on anterior teeth show a positive association with rainy seasons, additional evidence is needed before defects are employed to compare "seasonal stress levels" between or among living and fossil apes. One

potential solution is the integration of oxygen isotope microsampling, which reveals fine differences in heavy and light oxygen isotopes (Passey and Cerling, 2002; Ambrose, 2006; Wiedemann-Bidlack et al., 2008; Balasse et al., 2011), with the temporal mapping of developmental defects (Franz-Odenaal et al., 2003). This approach yields an independent assessment of variation in rainfall during the developmental period, which could be related to the timing of either external or internal developmental defects.

Skinner (1986) estimated that hypoplastic bands formed on chimpanzee and gorilla canines from Cameroon every 6 months, which he related to cyclic disease and/or malnutrition stress during major and minor rainy seasons. Skinner et al. (1995) suggested that these semianual linear enamel hypoplasias were likely due to malaria infection. However, it appears that the duration of the malaria transmission season is highly variable within Cameroon and throughout central Africa (illustrated in Gemperli et al., 2006: Fig. 2, p. 1035). Guatelli-Steinberg and Skinner (2000) expanded upon Skinner's (1986) hypothesis to suggest that the causative factor could be hookworm infection, which may show a seasonal rhythmicity. Yet studies of nematode infections in African apes suggest that parasite prevalence is variable across years and season (Huffman et al., 1997; Rothman et al., 2008; Gillespie et al., 2010; Masi et al. 2012), with only mixed support for a positive or consistent relationship between infections and rainy seasons. Chapman et al. (2009) notes that numerous factors may influence patterns of parasitism, including range use, forest quality, group size, diet, seasonality, and behavioral strategies, and that additional field studies are needed to understand the interrelationships among these factors.

It is difficult to routinely assess the health of wild primates from behavioral observations alone, as they have a tendency to mask or hide the symptoms of illnesses (Leendertz et al., 2006). Comprehensive health monitoring of wild primates requires the collection and analysis of fecal and urine samples (Krief et al., 2005; Lonsdorf et al., 2006), which were unfortunately unavailable for the individuals included in this study. Several disease outbreaks occurred in the community during the study period, and accentuated lines were not consistently observed in our subjects during these events (although behavioral observations of our three individuals during these outbreaks were limited). Rhinorrhea (runny nose) was common during the rainy seasons, affecting large numbers of the community during September 1990 and October 1991 (Boesch and Boesch-Achermann, 2000). Goshu did not show defects during either of these months, while Piment showed a major accentuated line during October 1991. A monkey pox outbreak was also documented in early 1987, and although Goshu appeared to have contracted this disease, she did not show associated developmental defects. Lefkas' mother was also noted to have had an ailment that affected her skin, but this did not correspond with corresponding accentuated lines in Lefkas. Future studies of developmental defect frequencies in immunocompromised individuals (e.g., Suckling et al., 1986) may be helpful to determine why certain individuals show developmental defects in their teeth during periods of illness while others seem to be unaffected.

Our sample of chimpanzees is currently too small to speculate on the relationship between developmental

defects and mortality. The period from 1986 to 1996 was a time of high infant mortality in the Taï forest; the number of infants declined by 50% (Boesch and Boesch-Achermann, 2000; Kuehl et al., 2008). This was due in part to epidemic infections, including two outbreaks of Ebola in October 1992 and November 1994. More broadly, the population size of the community has declined substantially since consistent monitoring began in 1982, with mortality peaks in 1988, 1989, and during the Ebola outbreaks (Boesch and Boesch-Achermann, 2000). Kuehl et al. (2008) reported that Taï infant mortality profiles from 1984 to 2006 did not show seasonal signals, nor was there any relationship between infant mortality and rainfall patterns. However, mortality events did show a relationship with fruit abundance, as it appears that social connectivity facilitates infectious disease outbreaks (Kuehl et al., 2008), and chimpanzee group sizes increase during periods of peak fruit abundance. In summary, although parasite infection has been shown to experimentally induce hypoplasias in sheep incisors (Suckling et al., 1986), results on the Taï forest individuals suggest that it is premature to employ accentuated lines or hypoplasias on molar teeth as specific or reliable indicators of rainy seasons or immunological challenges in wild primates.

#### Developmental defects, nutrition, and weaning

Comparisons of accentuated line frequency and fruit availability do not support the hypothesized negative relationship proposed by Skinner and Hopwood (2004); defect frequency was comparable between periods of high and low fruit availability. Moreover, we did not find a significant association between fruit availability and cumulative accentuated line frequency when defect class and/or rainfall were held constant. Chimpanzees have a broad and diverse diet, and it does not appear that our juveniles consistently experienced seasonal nutritional deficiencies severe enough to impact their molar development. Lefkas, who showed a slow rate of early skeletal development and the greatest number of accentuated lines and hypoplasias, may have experienced aperiodic bouts of malnutrition. His behavior of continuing to beg for food from his mother long after the birth of his sibling is atypical for juvenile chimpanzees. However, his accentuated line frequencies are fairly uniform across the year (save for August and September), suggesting that these lines are not simply due to seasonal patterns of fruit availability or disease cycles.

Direct observational data on suckling and solid food consumption are unavailable for the three juveniles in this study, although maternal observations allow some inferences about developmental defects and nursing history. For example, while it was unclear if Piment's mother Poupee had difficulty producing milk while nursing Piment, observations of a breast abscess and the loss of three previous infants before two years of age are highly indicative of a systemic problem with lactation. Piment's M1 revealed multiple large pit hypoplasias and marked accentuated lines before 1 year of age, and she showed proportionally more accentuated lines during the first two years of her life than during the following 1.8 years. Eastern chimpanzees begin to supplement mother's milk by about 6 to 12 months of age (reviewed in Smith et al., 2013), although suckling does not cease before age 4, and may continue until as late as ~7 years of age (Pusey, 1983). Individuals in the Taï community

are believed to suckle until ~4 to 5 years of age (Gagneux et al., 1999; Boesch and Boesch-Achermann, 2000).

Numerous studies suggest that aspects of the weaning process may lead to developmental defect formation (reviewed in Skinner et al., 2012; Smith, 2013), although there are no direct tests of this association. Recent evidence from free-ranging rhesus macaques has shown that weaning conflict may create physiological stress in infants; an increasing frequency of maternal rejection led to elevated levels of metabolic hormones (Mandalaywala et al., 2014). Skinner et al. (2012) argued that broad defects seen on the anterior teeth of other Tai chimpanzees (termed “coronal waisting”) are likely due to the weaning process. Both Piment and Goshu showed similar broad surface depressions in the lower half of their anterior teeth, which are likely to have formed between ~3 and 4 years of age (Reid et al., 1998). Lefkas showed more discrete classical hypoplastic defects on his incisors and premolars, which are likely to have formed after the M2 hypoplasias at 3.1 and/or 3.5 years of age. However micro-CT scans did not show any gross defects on his lower canines before his death at 7.6 years of age. Given the variation in defect expression and duration in the two individuals who are likely to have completed weaning (Goshu and Lefkas), additional observational and nutritional data are required to determine if hypoplasias (broadly defined) are reliable indicators of the weaning process in wild primates.

Accentuated lines have also been invoked as potential indicators of nutritional stress associated with early life diet transitions. The three individuals in our study did not show markedly greater frequencies of accentuated lines from ~2 to 4 years of age than during preceding years. Dirks et al. (2010) conducted an innovative study of accentuated lines and trace elements in two wild male baboons of unknown developmental histories. Several of these lines appeared to correlate with changes in the trace element strontium, which may increase with the introduction of solid foods. However accentuated lines were also apparent during periods of constant strontium levels, leading the authors to conclude that additional research was necessary to establish a relationship between accentuated lines and weaning stress. Moreover, strontium levels do not always change as expected with dietary transitions; trace elements such as barium appear to be more consistent in primates of known dietary histories (Austin et al., 2013).

Austin et al. (2013) undertook a temporal mapping study of barium, which is naturally elevated in mother's milk. They examined barium in M1s from four rhesus macaques, including one that showed an abrupt weaning transition at 166 days of age, but this individual did not show any corresponding developmental defect. In contrast, a major accentuated line was found to be coincident with a marked decline in barium levels at 1.2 years of age in a juvenile Neanderthal, indicating an abrupt diet shift. In this instance it is not clear if the accentuated line indicates the cessation of suckling, or if this coincided with an environmental, physiological, or immunological challenge that produced an accentuated line in conjunction with the end of maternal milk input. Additional studies on individuals of known developmental histories are needed to conclusively establish if and how physiological transitions such as weaning cause accentuated lines, and whether certain individuals may show more pronounced responses to this dietary transition.

### Developmental defects and behavior

Pronounced social or psychological stress represents another proposed cause of developmental defects in juvenile primate dentitions (Bowman, 1991; Dirks et al., 2002; Schwartz et al., 2006; Dirks et al., 2010). Bowman (1991) documented defect timing in several captive juvenile rhesus macaques, suggesting that certain events appear to be related to social disruptions, poor physical growth, separation from the mother, and/or the death of the mother. Dirks et al. (2002) suggested that accentuated lines in wild baboons may be caused by the stress of their mother's return to mating behavior. Social stress may have a marked effect on hormone levels such as cortisol in wild chimpanzees (Muller and Wrangham, 2004), and cortisol in rhesus macaque mothers' milk has been shown to affect infant temperament and weight gain (Hinde et al., 2015), although the relationship between cortisol levels, social stress, and reproductive state in female chimpanzees is complex (Emery Thompson et al., 2010; Markham et al., 2014).

We find limited support for possible social correlates of developmental defects in the three wild chimpanzees. Piment's mother engaged in aggressive behavior in September/October 1991 when she was quite young (as she become the alpha female), but it is not possible to decouple this from the respiratory infection that occurring in October 1991, during which time Piment showed a marked developmental defect lasting for several days. Our samples are insufficient to test for any potential relationship between maternal rank in a dominance hierarchy and the frequency of developmental defects in dependent offspring. The individuals with the higher ranking mothers (Piment and Lefkas) showed the highest and lowest numbers of defects over their first four years of life, while Goshu showed an intermediate number of defects and was born to a low ranking mother. Lefkas showed marked defects during the month his older sibling died, which also appeared to have impacted the behavior of his mother. In contrast, Goshu did not show developmental defects around the time her sibling was born. The two Ebola epidemics in October 1992 and November 1994 also represent two extremely disrupting social events; 17% and 28% of the community died during these outbreaks, respectively, including preferred social partners of our subjects' mothers (Boesch and Boesch-Achermann, 2000). Lefkas showed a marked accentuated line during the first outbreak, although Piment did not show any defects. During the second outbreak, which led to the death of Piment, Lefkas showed a moderate defect.

In conclusion, hypoplasia expression is highly variable within the tooth crown and across teeth forming at the same time (also see Hillson, 2014). We concur with Suckling (1989) and Guatelli-Steinberg et al. (2012), who stated that “the specific cause(s) of a hypoplastic defect cannot be discerned from its physical characteristics (p. 191).” They also note there are no studies demonstrating a clear association between hypoplasias and “particular ecological circumstances,” and we were unable to find evidence of this in the current study. Accentuated lines appear to be more consistent in their expression within and among synchronously forming teeth, leading to numerous applications for matching teeth and developing developmental chronologies (e.g., Beynon et al., 1991; Reid et al., 1998; Dirks et al., 2002; Schwartz et al., 2006; Smith et al., 2007a). Like hypoplasias,

accentuated lines appear to have multiple causes that are difficult to relate to specific environmental or immunological disruptions. The only consistently identified defect is the neonatal line in the deciduous teeth and permanent first molar of monkeys, apes, and humans, and without complementary elemental information, we caution against the use of postnatal developmental defects as indicators of specific events in an individual's history.

### ACKNOWLEDGMENTS

Uta Schwartz assisted with the management of the chimpanzee skeletal collection, Nancy Tang helped with figure preparation, and Steven Worthington provided invaluable statistical support. Katie Hinde, Fabian Leendertz, Zarin Machanda, Charlie Nunn, and Erik Scully engaged in helpful discussions of this research, and Kate Carter, Daniel Green, Adeline Le Cabec, Zarin Machanda, Nancy Tang, and two anonymous reviewers provided helpful comments on the manuscript. The authors acknowledge the Ministry of the Environment and Forests and the Ministry of Research, Côte d'Ivoire for allowing fieldwork in Tai National Park and for permitting the export of skeletal remains, as well as the Centre Suisse de Recherche Scientifique for their support.

### LITERATURE CITED

- Ambrose SH. 2006. A tool for all seasons. *Science* 314:930–931.
- Anderson DP, Nordheim EV, Moermond TC, Gone Bi ZB, Boesch C. 2005. Factors influencing tree phenology in Tai national park, Côte d'Ivoire. *Biotropica* 37:631–640.
- Austin C, Smith TM, Bradman A, Hinde K, Joannes-Boyau R, Bishop D, Hare DJ, Doble P, Eskenazi B, Arora M. 2013. Barium distributions in teeth reveal early life dietary transitions in primates. *Nature* 498:216–219.
- Austin C, Smith TM, Farahani RMZ, Hinde K, Carter EA, Lee J, Lay PA, Kennedy BJ, Sarrafpour B, Wright RJ, Wright RO, Arora M. In review. Multimodal imaging uncovers system-specific stress signatures in primate teeth. *Proc Natl Acad Sci USA*.
- Balasse M, Obein G, Ughetto-Monfrin J, Mainland I. 2012. Investigating seasonality and season of birth in past herds: a reference set of sheep enamel stable isotope oxygen ratios. *Archaeometry* 54:349–368.
- Beynon AD, Dean MC, Reid DJ. 1991. Histological study on the chronology of the developing dentition in gorilla and orangutan. *Am J Phys Anthropol* 86:189–203.
- Blakey ML, Leslie TE, Reidy JP. 1994. Frequency and chronological distribution of dental enamel hypoplasia in enslaved African Americans: a test of the weaning hypothesis. *Am J Phys Anthropol* 95:371–383.
- Boesch C, Boesch-Achermann H. 2000. *The chimpanzees of the Tai forest*. Oxford: Oxford University Press.
- Bowman JE. 1991. *Life history, growth and dental development in young primates: a study using captive rhesus macaques*. Ph.D. Dissertation. Cambridge: Cambridge University.
- Birch W, Dean MC. 2014. A method of calculating human deciduous crown formation times and of estimating the chronological ages of stressful events occurring during deciduous enamel formation. *J Forensic Legal Med* 22:127–144.
- Chapman CA, Hodder SAM, Rothman, JM. 2009. Host-parasite dynamics: connecting primate field data to theory. In: Huffman MA, Chapman CA, editors. *Primate parasite ecology*. Cambridge: Cambridge University Press. p 463–483.
- Chollet MB, Teaford MF. 2010. Ecological stress and linear enamel hypoplasia in cebus. *Am J Phys Anthropol* 142:1–6.
- Cunha E, Ramirez Rozzi F, Bermudez de Castro JM, Martinon-Torres M, Wasterlain SN, Sarmiento S. 2004. Enamel hypoplasias and physiological stress in the sima de los huesos middle pleistocene hominins. *Am J Phys Anthropol* 125:220–231.
- Dean MC, Elamin F. 2014. Parturition lines in modern human wisdom tooth roots: do they exist, can they be characterized and are they useful for retrospective determination of age at first reproduction and/or inter-birth intervals? *Ann Hum Biol* 41:358–367.
- Dean MC, Smith BH. 2009. Growth and development of the Nariokotome youth, KNM-WT 15000. In: Grine FE, Fleagle JG, Leakey RE, editors. *The first humans: origin and early evolution of the genus Homo*. Dordrecht: Springer. p 101–120.
- Dirks W. 1998. Histological reconstruction of dental development and age of death in a juvenile gibbon (*Hylobates lar*). *J Hum Evol* 35:411–425.
- Dirks W, Humphrey LT, Dean MC, Jeffries TE. 2010. The relationship of accentuated lines in enamel to weaning stress in juvenile baboons (*Papio hamadryas anubis*). *Folia Primatol* 81:207–23.
- Dirks W, Reid DJ, Jolly CJ, Phillips-Conroy JE, Brett FL. 2002. Out of the mouths of baboons: stress, life history, and dental development in the Awash national park hybrid zone, Ethiopia. *Am J Phys Anthropol* 118:239–252.
- Eli I, Sarnat H, Talmi E. 1989. Effect of the birth process on the neonatal line in primary tooth enamel. *Pediatr Dent* 11:220–223.
- Emery Thompson M, Muller MN, Kahlenberg SM, Wrangham RW. 2010. Dynamics of social and energetic stress in wild female chimpanzees. *Horm Behav* 58:440–449.
- FitzGerald CM, Saunders SR. 2005. Test of histological methods of determining chronology of accentuated striae in deciduous teeth. *Am J Phys Anthropol* 127:277–290.
- Franz-Odenaal TA, Lee-Thorp JA, Chinsamy A. 2003. Insights from stable light isotopes on enamel defects and weaning in pliocene herbivores. *J Biosci* 28:765–773.
- Gagneux P, Boesch C, Woodruff DS. 1999. Female reproductive strategies, paternity and community structure in wild west African chimpanzees. *Anim Behav* 57:19–32.
- Gemperli A, Sogoba N, Fondjo E, Mabaso M, Bagayoko M, Briët OJT, Anderegg D, Liebe J, Smith T, Vounatsou P. 2006. Mapping malaria transmission in west and central Africa. *Trop Med Int Health* 11:1032–1046.
- Gillespie TR, Lonsdorf EV, Canfield EP, Meyer DJ, Nadler Y, Raphael J, Pusey AE, Pond J, Pauley J, Mlengeya T, Travis DA. 2010. Demographic and ecological effects on patterns of parasitism in eastern chimpanzees (*Pan troglodytes schweinfurthii*) in Gombe National Park, Tanzania. *Am J Phys Anthropol* 143:534–544.
- Goodman AH, Allen LH, Hernandez GP, Amador A, Arriola LV, Chávez A, Pelto GH. 1987. Prevalence and age at development of enamel hypoplasias in Mexican children. *Am J Phys Anthropol* 72:7–19.
- Goodman AH, Martinez C, Chavez A. 1991. Nutritional supplementation and the development of linear enamel hypoplasias in children from tezonteopan, Mexico. *Am J Clin Nutr* 53:773–781.
- Goodman AH, Rose JC. 1990. Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *Yrbk Phys Anthropol* 33:59–110.
- Guatelli-Steinberg D. 2001. What can developmental defects of enamel reveal about physiological stress in nonhuman primates? *Evol Anthropol* 10:138–151.
- Guatelli-Steinberg D. 2003. Macroscopic and microscopic analyses of linear enamel hypoplasia in Plio-pleistocene South African hominins with respect to aspects of enamel development and morphology. *Am J Phys Anthropol* 120:309–322.
- Guatelli-Steinberg D. 2004. Analysis and significance of linear enamel hypoplasia in Plio-pleistocene hominins. *Am J Phys Anthropol* 123:199–215.
- Guatelli-Steinberg D, Benderlioglu Z. 2006. Brief communication: linear enamel hypoplasia and the shift from irregular to regular provisioning in Cayo santiago rhesus monkeys (*Macaca mulatta*). *Am J Phys Anthropol* 131:416–419. 1
- Guatelli-Steinberg D, Ferrell RJ, Spence J. 2012. Linear enamel hypoplasia as an indicator of physiological stress in great apes: reviewing the evidence in light of enamel growth variation. *Am J Phys Anthropol* 148:191–204.

- Guatelli-Steinberg D, Larsen CS, Hutchinson DL. 2004. Prevalence and the duration of linear enamel hypoplasia: a comparative study of Neandertals and Inuit foragers. *J Hum Evol* 47:65–84.
- Guatelli-Steinberg D, Skinner MF. 2000. Prevalence and etiology of linear enamel hypoplasia in monkeys and apes from Asia and Africa. *Folia Primatol* 71:115–132.
- Hillson S. 1996. Dental anthropology. Cambridge: Cambridge University Press.
- Hillson S. 2014. Tooth development in human evolution and bioarchaeology. Cambridge: Cambridge University Press.
- Hillson S, Bond S. 1997. Relationship of enamel hypoplasia to the pattern of tooth crown growth: a discussion. *Am J Phys Anthropol* 104:89–103.
- Hinde K, Skibieli AL, Foster AB, Del Rosso L, Mendoza SP, Capitanio JP. 2015. Cortisol in mother's milk across lactation reflects maternal life history and predicts infant temperament. *Behav Ecol* 26:269–28.
- Huffman MA, Gotoh S, Turner LA, Hamai M, Yoshida K. 1997. Seasonal trends in intestinal nematode infection and medicinal plant use among chimpanzees in the Mahale mountains, Tanzania. *Primates* 38:111–125.
- Humphrey LT. 2008. Enamel traces of early lifetime events. In: Schutkowski H, editor. *Between biology and culture*. Cambridge: Cambridge University Press. p 186–206.
- Katzenberg MA, Herring DA, Saunders SR. 1996. Weaning and infant mortality: evaluating the skeletal evidence. *Yearb Phys Anthropol* 39:177–179.
- Krief S, Huffman MA, Sévenet T, Guillot J, Bories C, Hladik CM, Wrangham RW. 2005. Noninvasive monitoring of the health of pan troglodytes schweinfurthii in the Kibale national park, Uganda. *Int J Primatol* 26:467–490.
- Kuehl HS, Elzner C, Moebius Y, Boesch C, Walsh PD. 2008. The price of life: self-organized infant mortality cycles in chimpanzees. *PLoS One* 3:e2440.
- Lacruz R, Ramirez Rozzi F, Bromage TG. 2005. Dental enamel hypoplasia, age at death, and weaning in the Taung child. *S Afr J Sci* 101:567–569.
- Le Cabec A, Tang N, Tafforeau P. In press. Accessing developmental information of fossil hominin teeth using new synchrotron microtomography-based visualization techniques of dental surfaces and interfaces. *PLOS One*.
- Leendertz F, Pauli G, Maetz-Rensing K, Boardman W, Nunn C, Ellerbrok H, Jensen S, Junglen S, Boesch C. 2006. Pathogens as drivers of population declines: the importance of systematic monitoring in great apes and other threatened mammals. *Biol Conserv* 131:325–337.
- Lonsdorf EV, Travis D, Pusey AE, Goodall J. 2006. Using retrospective health data from the gombe chimpanzee study to inform future monitoring efforts. *Am J Primatol* 68:897–908.
- Macho GA, Reid DJ, Leakey MG, Jablonski N, Beynon AD. 1996. Climatic effects on dental development of *Theropithecus oswaldi* from Koobi Fora and Olorgesailie. *J Hum Evol* 30:57–70.
- Mandalaywala TM, Higham JP, Heistermann M, Parker KJ, Maestripietri D. 2014. Physiological and behavioural responses to weaning conflict in free-ranging primate infants. *Anim Behav* 97:241–247.
- Markham AC, Santymire RM, Lonsdorf EV, Heintz MR, Lipende I, Murray CM. 2014. Rank effects on social stress in lactating chimpanzees. *Anim Behav* 87:195–202.
- Masi S, Chauffour S, Bain O, Todd A, Guillot J, Krief S. 2012. Seasonal effects on great ape health: a case study of wild chimpanzees and western gorillas. *PLOS One* 7:e49805.
- Moggi-Cecchi J, Pacciani E, Pinto-Cisternas J. 1994. Enamel hypoplasia and age at weaning in 19th-century Florence, Italy. *Am J Phys Anthropol* 93:299–306.
- Muller MN, Wrangham RW. 2004. Dominance, cortisol and stress in wild chimpanzees (*Pan troglodytes schweinfurthii*). *Behav Ecol Sociobiol* 55:332–340.
- N'guessan AK, Ortmann S, Boesch C. 2009. Daily energy balance and protein gain among pan troglodytes verus in the Tai national park, Côte d'Ivoire. *I J Primatol* 30:481–496.
- Passey BH, Cerling TE. 2002. Tooth enamel mineralization in ungulates: implications for recovering a primary isotopic time-series. *Geochim Cosmochim Acta* 66:3225–3234.
- Pusey AE. 1983. Mother-offspring relationships in chimpanzees after weaning. *Anim Behav* 31:363–377.
- Polansky L, Boesch C. 2013. Long-term changes in fruit phenology in a west African lowland tropical rain forest are not explained by rainfall. *Biotropica* 45:434–440.
- R Development Core Team. 2011. R: a language and environment for statistical computing, version 2.14. Vienna, Austria: R Foundation for Statistical Computing.
- Reid DJ, Schwartz GT, Dean C, Chandrasekera MS. 1998. A histological reconstruction of dental development in the common chimpanzee, *Pan troglodytes*. *J Hum Evol* 35:427–448.
- Rothman JM, Pell AN, Bowman DD. 2008. Host-parasite ecology of the helminths in mountain gorillas. *J Parasitol* 94:834–840.
- Rushton MA. 1933. On the fine contour lines of the enamel of milk teeth. *Dent Rec* 53:170–171.
- Simpson SW. 1999. Reconstructing patterns of growth disruption from enamel microstructure. In: Hoppa RD, Fitzgerald CM, editors. *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press. p 241–263.
- Schour I. 1936. The neonatal line in the enamel and dentin of human deciduous teeth and first permanent molar. *J Am Dent Assoc* 23:1946–1955.
- Schwartz GT, Reid DJ, Dean C. 2001. Developmental aspects of sexual dimorphism in hominoid canines. *Int J Primatol* 22:837–860.
- Schwartz GT, Reid DJ, Dean MC, Zihlman AL. 2006. A faithful record of stressful life events preserved in the dental developmental record of a juvenile gorilla. *Int J Primatol* 22:837–860.
- Skinner MF. 1986. Enamel hypoplasia in sympatric chimpanzee and gorilla. *Hum Evol* 1:289–312.
- Skinner MF. 1996. Developmental stress in immature hominines from late Pleistocene Eurasia: evidence from enamel hypoplasias. *J Archaeol Sci* 23:833–852.
- Skinner MF. 2014. Variation in perikymata counts between repetitive episodes of linear enamel hypoplasia among orangutans from Sumatra and Borneo. *Am J Phys Anthropol* 154:125–139.
- Skinner MF, Dupras TL, Moyà-Solà S. 1995. Periodicity of linear enamel hypoplasia among *Miocene dryopithecus* from Spain. *J Paleopathol* 7:195–222.
- Skinner MF, Hopwood D. 2004. Hypothesis for the causes and periodicity of repetitive linear enamel hypoplasia (rLEH) in large, wild African (*Pan troglodytes* and *Gorilla gorilla*) and Asian (*Pongo pygmaeus*) apes. *Am J Phys Anthropol* 123:216–235.
- Skinner MF, Pruett JD. 2012. Reconstruction of periodicity of repetitive linear enamel hypoplasia (rLEH) from perikymata counts on imbricational enamel among dry-adapted chimpanzees (*Pan troglodytes verus*) from Fongoli, Senegal. *Am J Phys Anthropol* 149:468–482.
- Skinner MF, Skinner MM, Boesch C. 2012. Developmental defects of the dental crown in chimpanzees from the Tai national park, Côte d'Ivoire: coronal waisting. *Am J Phys Anthropol* 149:272–282.
- Smith TM. 2013. Teeth and human life-history evolution. *Annu Rev Anthropol* 42:191–208.
- Smith TM, Machanda Z, Bernard AB, Donovan RM, Papakyrikos AM, Muller MN, Wrangham R. 2013. First molar eruption, weaning, and life history in living wild chimpanzees. *Proc Natl Acad Sci USA* 110:2787–2791.
- Smith TM, Smith BH, Reid DJ, Siedel H, Vigilant L, Hublin J-J, Boesch C. 2010. Dental development of the Tai forest chimpanzees revisited. *J Hum Evol* 58:363–73.
- Smith TM, Tafforeau P. 2008. New visions of dental tissue research: tooth development, chemistry, and structure. *Evol Anthropol* 17:213–226.
- Smith TM, Tafforeau P, Reid DJ, Grün R, Eggers S, Boutakiout M, Hublin J-J. 2007b. Earliest evidence of modern human life history in north African early *Homo sapiens*. *Proc Natl Acad Sci USA* 104:6128–6133.
- Smith TM, Toussaint M, Reid DJ, Olejniczak AJ, Hublin J-J. 2007a. Rapid dental development in a middle paleolithic

- Belgian Neanderthal. *Proc Natl Acad Sci USA* 104:20220–20225.
- Suckling GW. 1989. Developmental defects of enamel – historical and present-day perspectives of their pathogenesis. *Adv Dent Res* 3:87–94.
- Suckling G, Elliott DC, Thurley DC. 1986. The macroscopic appearance and associated histology changes in the enamel organ of hypoplastic lesions of sheep incisor teeth resulting from induced parasitism. *Arch Oral Biol* 31:427–439.
- Swindler DR, Beynon AD. 1993. The development and microstructure of the dentition of *Theropithecus*. In Jablonski NG, editor. *Theropithecus: the rise and fall of a primate genus*. Cambridge: Cambridge University Press. p 351–381.
- Temple DH, Goodman AH. 2014. Bioarcheology has a “health” problem: conceptualizing “stress” and “health” in bioarcheological research. *Am J Phys Anthropol* 155:186–191.
- Wiedemann-Bidlack FB, Colman AS, Fogel ML. 2008. Phosphate oxygen isotope analysis on microsamples of bioapatite: removal of organic contamination and minimization of sample size. *Rapid Commun Mass Spectrom* 22:1807–1816.
- Whittaker DK, Richards D. 1978. Scanning electron microscopy of the neonatal line in human enamel. *Arch Oral Biol* 23:45–50.
- Witzel C, Kierdorf U, Schultz M, Kierdorf H. 2008. Insights from the inside: histological analysis of abnormal enamel microstructure associated with hypoplastic enamel defects in human teeth. *Am J Phys Anthropol* 136:400–414.
- Wood L. 1996. Frequency and chronological distribution of linear enamel hypoplasia in a north American colonial skeletal sample. *Am J Phys Anthropol* 100:247–259.