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Orangutans, enamel defects and developmental health: A comparison of Borneo and Sumatra

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Title: Orangutans, enamel defects, and developmental health: A comparison of Borneo and Sumatra

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ABSTRACT

Orangutans (*Pongo* sp.) show among the highest occurrence of three types of developmental enamel defect. Two are attributed to nutritional factors that reduce bone growth in the infant's face early in development. Their timing and prevalence indicate that Sumatra provides a better habitat than does Borneo. The third type, repetitive linear enamel hypoplasia (rLEH) is very common but its etiology is not understood. Our objective is to draw attention to this enigmatic, episodic stressor in the lives of orangutans. We are concerned that neglect of this possible marker of ill health may be contributing, through inaction, to their alarming decline in numbers. Width and depth of an LEH are considered proxies for duration and intensity of stress. The hypothesis that Bornean orangutans would exhibit relatively wider and deeper LEH was tested on 163 independent episodes of LEH from 9 Sumatran and 26 Bornean orangutans measured with a NanoFocus AG 'μ surf Mobile Plus' scanner. Non-normally distributed data (depths) were converted to natural logs. No difference was found in width of LEH among the two island taxa; nor are their differences in width or depth between the sexes. After controlling for significant differences in LEH depths between incisors and canines, defects are, contrary to prediction, significantly deeper in Sumatran than Bornean animals (median =28µm, 18µm, respectively). It is concluded that repetitive LEH records an unknown but significant stressor present in both Sumatra and Borneo, with an average periodicity of six months (or multiples thereof) that lasts about six to eight weeks. It is worse in Sumatra. Given this patterning, shared with apes from a wide range of ecological and temporal sources, rLEH is more likely attributable to disease than to malnutrition.

Key words: ***Pongo pygmaeus; P. abelii;*** **infancy; dentition; stress**

RESEARCH HIGHLIGHTS

Orangutans are disappearing for largely known reasons. Most dental defect types support Sumatra being deemed a better habitat than Borneo. However, one enigmatic defect, repetitive linear enamel hypoplasia, occurs on both islands but is more severe in Sumatra.

INTRODUCTION

Orangutans from Borneo and Sumatra are Critically Endangered having lost 80% of their numbers in just three generations due mostly to habitat loss, habitat fragmentation and illegal hunting [IUCN, 2017]. Our purpose here is to draw attention to an enigmatic, episodic marker of stress in the lives of orangutans, termed repetitive linear enamel hypoplasia, so as to enlist assistance from field primatologists to help find its cause. We are concerned that neglect of this possible marker of ill health may, inadvertently, be contributing to their decline through inaction. Abnormal dental formation provides a record of developmental stress in young hominoids. In an evaluation of three types of dental defect (repetitive linear enamel hypoplasia (rLEH), localized hypoplasia of the primary canine (LHPC), maxillary lateral incisor defect (MLID)) among five large apes (orangutans, mountain and lowland gorillas, chimpanzees and bonobos), orangutans have the highest or second highest prevalence [Skinner, 1986a; Guatelli-Steinberg and Skinner, 2000; Skinner and Newell, 2003; Tsukamoto, 2003; Skinner and Hopwood, 2004; Guatelli-Steinberg et al., 2012; Skinner, 2014a; Hannibal, 2016; Skinner et al., 2016]. These findings suggest that, albeit for unknown reasons, orangutans are remarkably stressed in infancy. Given the perilous nature of orangutans in the wild, there is an urgent need to document and understand developmental stress in infancy as recorded in their teeth.

Developmental defects of enamel

There is a strong link between developmental health of growing orangutans and habitat quality [Knott, 1998; Delgado and van Schaik, 2000]. Enamel formation has been the subject of numerous studies, e.g. [Osborn, 1981; Nanci, 2012]. Hypoplastic defects of enamel are usually attributed to metabolic stress that results in abnormal secretion of matrix prior to full mineralization of the enamel. It is widely accepted that enamel hypoplasia is a non-specific marker of systemic stress [Goodman and Rose, 1990]. However, this rather bleak assessment can be mitigated somewhat by distinguishing among the (admittedly complex) etiologies of different types of hypoplastic defects as well as by asking whether the timing, severity, prevalence and epidemiology of a defect type may be sufficiently distinctive as to suggest a specific etiology. Our study is designed to explore the striking patterning of rLEH among orangutans in the hope of elucidating etiology.

Timing of bone mass defects

'Localized hypoplasia of the primary canine' (LHPC) and 'maxillary lateral incisor defect' (MLID) share a common proximate etiology-insufficient bone growth in the face-but are created at different times in infancy (Fig. 1). Both LHPC and MLID are crypt fenestration defects in which reduced bone growth in the face leads to creation of an enamel defect prior to eruption [Skinner, 1986b; Skinner and Hung, 1989; Skinner et al., 2014].

LHPC is caused by fenestration of the labial crypt wall, normally protecting the unerupted, forming milk canine crown, when cranio-facial bone growth in the infant fails to keep up with crown formation [Skinner and Newell, 2003]. In humans and apes, the affected part of the tooth crown, exposed to trauma through a fenestration, forms in the months shortly after birth [Skinner and Newell, 2003; Stojanowski and Carver, 2011]; consequently it is assumed that, among breast-feeding cohorts, LHPC reflects condition of the mother as much as the infant. Approximately 91% of orangutans are affected [Lukacs, 2001]. Notably, there is no difference between Sumatran and Bornean orangutans in the occurrence of LHPC.

The second crypt fenestration defect, MLID, is attributed to abnormal contact of the labial surface of the somewhat less-formed upper lateral incisor crown with the incisal edge (or fenestration margin) of the more mineralized central incisor through a fenestrated inter-crypt boney septum in under-developed jaws with pre-eruptive dental crowding [Hannibal, 2016; Skinner et al., 2016]. MLID is created in the first few years of an orangutan's life (ca. 2-5 years) when the infant is increasingly reliant on foraging for itself. We define infancy as that period during which lactation occurs [van Noordwijk et al., 2013] which lasts as long as 5.5 years in Bornean orangutans and 6-7.5 years in Sumatran [van Noordwijk et al., 2009]. Notably, almost all incisor and canine crown formation occurs within this age span except perhaps for the cervical fifth of the male canine [Beynon et al., 1991]. Orangutans are markedly affected (59%) by MLID but the lesion is far more common in Bornean orangutans (71%) than Sumatran (29%) [Skinner et al., 2016].

Considering the occurrence of both LHPC and MLID, it can be concluded that: a) nutrition for infants from both islands is more adequate when the infant is more fully reliant on breast milk than in later infancy; and b) Sumatra provides a more suitable nutritional habitat for infants than does Borneo. The latter inference is well supported in the literature. There are several lines of ecological evidence indicating that Sumatra provides a superior habitat for orangutans due, fundamentally, to volcanically-derived soils [Wich et al., 2011]. In Sumatra there are more months in the year with high fruit availability and a trend towards shorter low fruit periods [Delgado and van Schaik, 2000; Marshall et al., 2009; Wich et al., 2011]. Unlike Bornean orangutans, Sumatran orangutans spend more time on high quality foods like fruit and insects and a lower percentage on bark and vegetation. Moreover, Sumatran orangutans seem less reliant on fallback foods than are Bornean, being able to find figs and fruit year round [Russon et al., 2009]. In Borneo, there are months where fruit is a minor part of the diet while in Sumatra fruit is always a major part of the diet [Morrogh-Bernard et al., 2009]. Not surprisingly, therefore, orangutan population density is higher in Sumatra [van Schaik et al., 2009].

(Figure 1 about here)

Timing of repetitive linear enamel defects

The third type of enamel defect (linear enamel hypoplasia) is a more direct manifestation of abnormal secretion in which transverse furrows of thinned enamel are created. All ape samples, including fossil forms, commonly show several furrows on the incisor and canine dental crowns. The remarkable ubiquity of repetitive LEH in time and space, spanning millions of years (Miocene to present) and thousands of kilometers from Spain to China and Africa [Skinner et al., 1995; Skinner and Roksandič, 1995; Guatelli-Steinberg and Skinner, 2000; Brunet et al., 2002], suggests that a pervasive and common stressor may underlie the phenomenon. However, we should be explicit that the etiology of rLEH cannot as yet be attributed with confidence to malnutrition and/or disease, the fundamental agents behind the metabolic stress associated with enamel hypoplasia [Goodman and Rose, 1990].

LEH have been reported in the canine teeth of orangutans among whom they tend to commence at about 2.5 years of age [Skinner and Hopwood, 2004] and recur throughout crown formation [Skinner, 2014b] (up to about six to nine years of age depending on sex [Beynon et al., 1991; Schwartz and Dean, 2001] (Fig. 1). Among orangutans in general the stressful events recur on average about every six or twelve months; Sumatran animals showing significantly more annual episodes of stress while Bornean animals show more semi-annual episodes [Skinner, 2014b]; a pattern interpreted to provide mild support for Sumatra being the better habitat. However, Sumatran orangutans are reported to show more LEH defects per tooth [Guatelli-Steinberg et al., 2012], which seems incompatible with their longer cycle (but which may reflect different subjective thresholds of LEH visibility between investigators, reinforcing our contention that measurement of LEH furrows should be pursued).

Roughly 83% of orangutans from museum collections show ≥1 episodes of LEH per tooth [Hannibal and Guatelli-Steinberg, 2005]. When nearly all individual apes in both Sumatra and Borneo are affected by rLEH, another way has to be found to measure comparative developmental stress in an informative manner. A more telling test of island differences would be to measure, not the prevalence or periodicity of the episodic stress events, but their duration and intensity. We hypothesize that width and depth measurements of LEH serving as proxies for duration and intensity of stress, respectively, will be less in Sumatran orangutans, a prediction based on Borneo being deemed the poorer habitat.

METHODS

Specimens of extant animals examined in this study consist only of skeletal remains from museum collections; all animals were dead prior to our study. Proposed examination was approved by curators of museums listed in Table I. All examinations were performed at the institution and no hard tissue was removed or transported. This research adheres to the American Society of Primatologists principles for the ethical treatment of primates.

Orangutans in this study have been previously described [Skinner, 2014b]. They come from three museums in Germany and one in Holland whose collections were examined in 1999 and 2000. The animals were taken from the wild in the late 19th and early 20th century; hence provenience information is imprecise or, in a few cases, unknown. The majority of the Bornean animals come from the Sintang and Sekelau region in eastern West Borneo. The Sumatran animals are from the far north end of the island near Atjeh (Aceh) and Deli. Here we employ a species distinction between the two island populations (*P. abelii* in Sumatra and *P. pygmaeus* in Borneo) reflecting the marked genetic and morphological differences between the two island taxa [Goossens et al., 2009].

(Table I about here)

Animals for the previous study [Skinner, 2014b] were chosen because they showed countable perikymata between two or more episodes of LEH on a single tooth (incisor or canine). Where defects were demonstrably bilateral, the antimere with more visible features on the outer enamel surface was chosen. An individual counts only once. Here we report LEH defects on an enlarged sample of teeth from the same animals to include slightly worn, but measurable, LEH with uncountable perikymata. Still, the sample (Table I) is biased towards younger individuals with comparatively little labial wear, and purposefully excludes animals whose teeth show only a single episode of LEH, since the latter animals did not afford an opportunity to study the interval between episodes and, hence, LEH periodicity. We acknowledge that sample sizes are disparate and that for Sumatra (N=9 animals), small. However the sample sizes for repetitive LEH events, whose occurrence we consider to be independent of a previous event, are reasonably large (N=30 for Sumatra and 133 for Borneo).

Nevertheless, any inferences made in our study will need to be tested against other and larger samples before firm conclusions can be drawn.

Terminology

Usages of the word ‘stress’ are considered so discordant that hopes of a standard definition are forlorn [King and Murphy, 1985]. Our definition is not as strict as theirs-that vital physiological function must be impaired-but we do consider that reduced cellular secretion of enamel matrix, sufficient to affect contour of the outer enamel surface, qualifies as indicative of physiological stress. While methods employed in this study are a deliberate move away from a subjective threshold of perception of a hypoplastic furrow towards measurement, we employ the term ‘salience’ to mean the subjective visibility of depressions in the outer enamel surface (conflating width and depth), often enhanced by vegetable staining, since this threshold has historically guided our researches and has formed the basis of communication among scholars. We should be clear however that the salience of linear enamel hypoplasia, whether objectively or subjectively assessed, reflects the host animal’s experience of stress mediated by many factors, not stress *per se*. In other words, measurements of enamel hypoplasia do not measure stress at all-they measure the response to stress; but forging a link between stress and enamel defects is challenging. LEH reflects the potential interaction, during development, of many factors (e.g., individual immunocompetence, foraging efficiency, food acquisition skills and social rank); meteorological influences (e.g., seasonality, insolation, rainfall cycles); and abiotic factors (e.g., soil type) with a variety of stressors (e.g., disease, malnutrition) [Eckhardt and Protch von Zieten, 1993; Guatelli-Steinberg, 1998, 2000; Guatelli-Steinberg and Skinner, 2000; Guatelli-Steinberg, 2001; Chollet and Teaford, 2010; Kirchoff, 2010]. Historically, there has been acceptance of a simple separation of defects into narrow or wide [Sarnat and Schour, 1941; Corruccini et al., 1985; Bermudez de Castro and Perez, 1995] with little or no concern for depth; and, yet, depth contributes to the salience of an LEH. The latter authors speculated that narrow and wide grooves might represent infection and dietary deficiency, respectively. Blakey et al. [1994] used the phrase 'major growth arrests' to describe very wide defects. Similarly, Ensor and Irish observed what they termed 'continuous chronic enamel hypoplasia' and captured the phenomenon with the concept of 'total hypoplastic area' [1995]. Many authors have invoked a threshold of 0.4 to 0.5mm width of defect to distinguish shorter (acute) and longer episodes of stress [Hutchinson and Larsen, 1988; Ensor and Irish, 1995; Duray, 1996; Vann, 2008]. There seems to be general acceptance that the width of a hypoplastic defect provides a reasonable estimate of duration of a stress event or events. Here, we draw a clear distinction between widths and depths.

Width

The accepted method for estimating duration of stress in humans is to count the number of perikymata in the occlusal wall of a defect and multiply this figure by the known or inferred Retzius periodicity in days [Hillson and Bond, 1997]. We question the generalization, made by Hubbard and colleagues [2009], that perikymata counting is more accurate than measuring defect widths, mostly because in chimpanzee, bonobo and orangutan canine teeth, at least, perikymata spacing is more uniform throughout most (ca. middle 80%) of the crown than it is in human canine teeth [Dean and Reid, 2001; Guatelli-Steinberg et al., 2012; O'Hara, 2016]. In our study, 94% of LEH occur within crown deciles four through nine. According to a recent study by O'Hara [2016] the median number of perikymata among these deciles ranges from only 26 to 28 per decile. Also, a recent study reports a range of only nine or ten days per perikyma in both Sumatran and Bornean orangutans [Smith, 2016]. Consequently, we feel that measurements of the width of LEH defects should give a reasonable estimate of duration of stress (as qualified above) in each taxon. Both perikymata count and width are proxies for time. Fundamentally, it is much easier to obtain a sufficiently large sample to detect differences between populations by measuring widths than counting perikymata. As Hubbard and colleagues observe: "Bioarchaeologists are faced with the choice of using a more accurate method (perikymata counting) on a small sample (given the small number of defects with continuously visible perikymata within them), or using a potentially less accurate method (measuring defect widths) on a larger sample" [2009:178]. Here we have elected to eschew perikymata counts in favour of width measurements, on an enlarged sample.

We define 'width' as a direct measure, along an imaginary line joining the two shoulders of a hypoplastic defect, taken from the occlusal shoulder to a place orthogonal to the deepest point of a defect, taken in an occlusal-cervical axis. This amounts to roughly half the width of most furrows. In our view, enamel deposition after the end of a stressful episode restores normal enamel contour; consequently, width of what can be termed the 'recovery of normal enamel contour phase' is a function of both enamel geometry and depth of the furrow but is not necessarily informative of the actual time required to recover from stress [Hillson and Bond, 1997]. For the purposes of this study, we do not distinguish between plane-form versus furrow-form defects. Our study is based on furrow-form defects but it is conceivable that some of the more worn LEH without countable perikymata in the occlusal wall are actually plane-form defects.

Depth

Depth is deemed by us to be an indirect measure of the intensity of stress (as mediated by anatomical factors-e.g., occlusal vs. cervical location [Hillson and Bond, 1997; Hubbard et al., 2009], environmental factors [Chollet and Teaford, 2010] and individual frailty [King and Ulijaszek, 1999]). How do we know that the more severe a stressor, the deeper will be a hypoplastic defect? Numerous studies support the inference that there is a dose-dependent reaction of secretory phase ameloblasts to increasing levels of a stressor: e.g., a) fluoride [Suckling and Thurley, 1984; Kierdorf et al., 2004]; b) reduced age at death [White, 1978; Cook and Buiskstra, 1979; Goodman and Armelagos, 1988; Duray, 1996]; c) reduced availability of fat soluble vitamins

(Mellanby M. 1929 cited in [Mellanby, 1941; Goodman et al., 1991] although the effect here may be due to decreased bone growth impinging on the dental crown); d) parasitism [Suckling et al., 1986]; e) maternal hyperglycemia in diabetic rats [Silva-Sousa et al., 2003]; and f) seasonal insolation [Zadsinska et al., 2013].

The notable unevenness along the floor of an LEH furrow [Boyde, 1970] necessitates multiple measurements; with the instrument described below, we take 516 measurements over a space of 1600 microns. We define depth as the orthogonal distance from a plane connecting the two high points on the margins of a defect to its deepest point. In this study, measurements are un-scaled since there are no differences in average enamel thickness between island taxa [Smith et al., 2012].

Impressions and casts

Enamel was cleaned of surface residue with dilute acetone. Molds of enamel surfaces affected with rLEH were taken in Coltene President Plus Jet impression material, supported by Coltene Lab Putty and polysiloxane activator (Coltene® , Cuyahoga Falls, Ohio, USA). Casts were made in Araldite MY 753 epoxy resin with XD 716 hardener (Ciba-Geigy®, Toms River, New Jersey, USA). Close-up photographs of each tooth cast were combined into a photomontage with Adobe Photoshop Elements 10 (Adobe®, San Jose, California, USA).

Identification of LEH events

Irregularities of the enamel surface were visualized in the first instance under oblique lighting at low magnification (ca. 6X). Macroscopically visible LEH are the focus of our study. Our threshold for a measurable LEH does not include every minor vicissitude involving, for example, just a single perikyma. Low power (ca. 10-15X) pictures of a whole crown were recorded and the rLEH provisionally numbered consecutively starting at the apex: 1, 2, and so on. The task of matching LEH between low and high magnification pictures was accomplished by noting small irregularities (e.g., scratches) or imperfections in the cast (e.g., bubbles) that could be located on both images. A visual comparison was made of LEH salience between scanner photomontages and the lower power photographs and sketches, and a final decision made as to the location and number of LEH events (Table II). Only one tooth per individual was employed so as to avoid statistical redundancy (i.e., where one event might be recorded on both an incisor and canine from a single individual); canines being given preference as they typically show more LEH per crown.

(Table II about here)

Instruments

Epoxy casts were examined with a 'μ surf Mobile Plus' optical scanner (OS) and analysed with μ soft Analysis Premium 6.2 software from NanoFocus® AG (Oberhausen, Germany). This instrument enables the analysis of 3-D structures and geometries in the micrometer and nanometer range. The precise 3-D topography is computed from the acquisition of a large number of confocal filtered height sections (typically ≥600). The OS consists of a compact confocal probe mounted on a stable Lstand with motorized movement to focus in the z-axis (maximum resolution = 1 nm). The sample to be measured is secured to an x/y precision measurement table. For contactless measurement of surface topography, a sample is positioned on the measurement table and the confocal unit moved stepwise in the z axis. In this instrument, magnification was usually performed with a 10X lens that provides a square field of view 1600µm on a side. Width and depth measurement outputs are averages, calculated by the instrument, from 516 measurements over this space. Prior to trigonometric analysis, scanner images were leveled, missing points filled in, and form removed. The latter step optimizes measurement of defect depth by minimizing the effect of object curvature.

Data Manipulation (trigonometry)

It was deemed desirable to determine true depths, not depth in relation to the instrument plane, since the object's surface is rarely level or completely flat. This is performed trigonometrically from width and depth measures originally taken orthogonal to the instrument's plane (Fig. 2).

(Figure 2 about here)

Anatomical factors

As illustrated by Guatelli-Steinberg [2001, 2003], where striae of Retzius emerge at an acute angle with the outer enamel surface (typical of more occlusal imbricational enamel compared to cervical, especially in incisors), all else being equal, LEH width occlusally is increased and apparent salience, decreased. In order to expedite comparisons of defect salience between taxa, we first compare measures of defect width and depth among crown deciles.

Since we would like to know if LEH salience compared along the crown reflects real differences in the intensity of experienced stress or simply constraints of enamel geometry , we need to measure the obligatory effect on furrow depth created by a narrowing of an incremental stria of enamel. A simple trigonometric calculation (ratio of sine angles) shows that changing stria angle from 10^{o} (near the occlusal tip of a canine tooth) to 45^{o} (nearer the cervix) [Guatelli-Steinberg et al., 2012], reduces depth of a defect at the cervix to about 0.72 of the depth near the occlusal tip. The lesson here is that, in furrow-form defects where incremental striae are narrowed, the same amount of stress will produce shallower defects near the cervix. Consequently, we suggest, as a reasonable threshold, that only if more cervically-located LEH are <u>deeper</u> than defects near the occlusal tip, is a real difference in the felt intensity of stress being signaled.

Statistical analysis

Preliminary analysis, using Kolmogorov-Smirnov and Shapiro-Wilks tests, showed that width measures are normally distributed while depths are not, being significantly left skewed (Fig. 3, Table III). Consequently, statistical analysis of difference between mean widths relies on parametric (two-tailed Student’s t, ANOVA) tests on original values; while depths are evaluated with non-parametric tests (Mann-Whitney and Kruskall-Wallis) on original values or, alternatively, original values are converted to natural logs so that the normalized distribution can be evaluated with parametric tests. Given significant variation in LEH measures between tooth types (incisors and canines) original measurements are also converted to z-scores (value minus mean for a particular combination of tooth type divided by standard deviation for that category) in order to maximize sample size. Alpha is set at 0.05.

(Table III about here)

(Figure 3 about here)

RESULTS

Anatomic Variables: Crown decile, Sex, Tooth type

Our goal is to see if LEH defects are, as hypothesized, wider and deeper in Bornean orangutans; i.e., that Sumatra provides the better developmental habitat for infant orangutans. Firstly, however, we need to test whether one has to control for basic anatomical variables: location of LEH within the tooth crown (crown deciles numbered from occlusal to cervical), sex (male vs. female) and tooth type (incisors vs. canines). Sample sizes are not sufficient to test each sub-group separately; so, initially, we consider simply each variable by itself, lumping the other variables. The following analysis of difference between means (for each of the three variables of crown location, sex and tooth type) employs parametric tests for widths and non-parametric statistics for depth (Table IV). We found that measures of LEH width do not vary significantly among deciles, sex, or tooth types. Depths vary significantly only between tooth types (Fig. 4). Subsequent analyses of both width and depth ignore LEH location and sex. Since depths are not only non-normally distributed but also vary as a function of tooth type, statistical analysis requires control of these variables.

(Table IV about here)

(Figure 4 about here)

Given relative stasis in the maternal contribution to the infant orangutan's diet [van Noordwijk et al., 2013], the growing infant must rely increasingly on its own resources (both foraging ability and immunity to disease). Thus, it can be predicted that later LEH events will be more severe. However, as noted above, the depth of an LEH is naturally decreased towards the cervix of a tooth due to the angle with which striae reach the outer enamel surface. In our study of the canine teeth, the ratio of decile 4 (more occlusal) to decile 9 (more cervical defect) median depths is 0.49-notably less than the value of 0.72 predicted by enamel geometry-suggesting that the intensity of the stressor in real terms increases with age of the animal (albeit episodically) thus agreeing with the prediction.

Island variable

It was shown in Table IV that width and depth of LEH furrows do not vary significantly as a function of their location on a tooth crown. If larger or different samples were obtained this finding may not be confirmed, in which case it would be desirable to know whether the distribution of LEH along the crown does or does not differ between island populations. It is clear from Figure 5 and Table V that both island samples in this study show the same distribution of LEH along the crown.

(Figure 5 about here)

(Table V about here)

In this study, widths and depths of LEH are considered to be proxies for duration and intensity of felt stress, respectively. There is no difference in measures of width between the Bornean and Sumatran orangutans; but there is a clear difference in depths (Table VI). LEH defects among Sumatran orangutans are significantly deeper than those for Bornean orangutans-difference between medians = 10.0μm. However, it must be remembered that, while there are no significant differences in depths from different portions of the crown, depths do differ between tooth types (slightly but significantly deeper on canines) (Fig. 4). For this reason, Table VI includes a section in which depth measures are expressed as z-scores (that is, deviation of either a natural log of depth or untransformed measure from the mean for each sub-group of tooth type (incisors and canines)) and subjected to parametric and non-parametric tests of difference in means. This manipulation permits lumping of both tooth types, confirming that depths are significantly shallower on average in Bornean orangutans (Student's t=-2.487, df=161, P=0.014; Mann-Whitney=-2.509, P=0.012) (Fig. 6).

(Table VI about here)

(Figure 6 about here)

While our purpose here is not to convert measured LEH widths directly into precise measures of duration, but to compare two samples for relative durations, we can nevertheless ask if the observed mean widths are at all compatible with what we know about perikymata widths and Retzius periodicity. In our study, mean width of a single perikyma is 73µm for incisors (N=4 in mid-crown third) and 69µm for canines (N=3 in mid and cervical thirds) (cf., mean of 68 to 84µm reported by O'Hara [2016]) . Dividing mean width by the corresponding perikyma widths noted above yields a rough estimate of 4.9 and 5.5 perikymata per LEH, respectively, which (assuming that one perikyma represents nine to ten days [Schwartz et al., 2001; Smith, 2016]), suggests stress lasts six to eight weeks in both island taxa.

DISCUSSION

Our hypothesis that Bornean orangutans would show wider and deeper hypoplastic defects than do Sumatran orangutans is not supported by our results. However, there are several caveats that must be considered before accepting such results. Firstly, our study is of specifically repetitive linear enamel hypoplasia, not LEH in general; i.e., this cohort may be biased towards more susceptible individuals. Secondly, in that widths of LEH do not differ between the island samples but depths do, we have to ask whether these simply reflect Type II and Type I errors, respectively. The relative cost of a Type I error is more than that for a Type II error since, for the latter, a real difference will manifest itself after further studies [Toft and Shea, 1983]. To avoid Type I error one can invoke a higher alpha value than, say, 1 in 20 (0.05). As may be seen in Table VI, the observed P value for island orangutan differences in depth is <0.02, meeting this requirement. Thirdly, we can use power analysis to determine if our sample sizes are adequate. Depths of LEH are much more variable than are widths (Table III); consequently, selection of an adequate sample size, in which both will be compared between populations, will be dictated by variability in depths. Depths, however, are not normally distributed. Power analysis assumes a normal distribution. Natural log transformation of observed depths normalizes the distribution from which one can determine the median, and difference between the median and 68th percentile of the distribution. Antilogs of these values (18.6µm and 7.4µm, respectively) approximate the mean and standard deviation of a normal distribution. Power analysis indicates that sample sizes required to detect a real difference in mean depths of LEH between two populations (with a SD of 7.4µm and optional delta value of 5µm) are 35 LEH for each island taxon. Our sample of Sumatran orangutan LEHs is 30, a bit below the required minimum. However, it is permissible [Motulsky, 1995] to reduce the required size of one sample by 25% (from, say, N=35 Sumatran orangutans to N= 26) if one doubles the size of the other; i.e., to N=≥70 Bornean. In our case, the Bornean sample is 133 events, which means our study samples should be quite sufficient to detect a minimum difference in mean depths, between samples, of only 5µm. Our observed difference in mean and median LEH depth values between island populations of orangutans is 10.2µm and 10.0µm, respectively. We conclude that our findings are robust, with the caveats noted above.

Both island taxa exhibit LEH of similar duration (six to eight weeks on average). This result is surprising in that previously published estimates of duration of ‘whole furrows’ (including width as defined in this study and the recovery of enamel contour phase) were on the order of six to seven weeks [Skinner and Hopwood, 2004]; in other words, the current results suggest that stress lasts longer than previously thought.

Contrary to expectation, Sumatran orangutans show significantly deeper/more severe LEH compared to Bornean (median =28µm, 18µm, respectively). While, conceivably, Bornean orangutans, who show more reliance on abrasive fall-back foods, might wear away more enamel surface, the amount of wear required to render uncountable perikymata, which are only about one micron in surface relief, is so minimal that the effect on furrow depth is probably not germane to our study. As noted in the introduction, there are very few published measurements of LEH depth in non-human primates. Interestingly, median depth of LEH among only three Fongoli chimpanzees (18 LEH), with a marked dry season, is 32µm (excluding plane-form defects) [Skinner and Pruetz, 2012], only slightly more than in Sumatran orangutans (median = 28µm). The shallower LEH of Bornean orangutan LEH compared to both Sumatran orangutans and Fongoli chimpanzees remains to be understood.

We may ask ‘How consistent are the studies of enamel hypoplasia in providing support for the notion that Sumatra provides a better habitat?’ They are not. As reviewed earlier LHPC, attributed to facial bone thinning in early infancy, does not differ between the islands while MLID, attributed to undergrowth of the maxilla, is significantly worse in Bornean orangutans. Together, these findings suggest that, as the growing infant is forced by static maternal milk reserves [van Noordwijk et al., 2009] to forage for itself, cranio-facial growth falters more in Borneo (supporting the notion). However, the current study shows that LEH furrows do not differ in width (equated here with duration) between the island populations; whereas depth (equated with intensity of stress) is significantly more marked in Sumatran orangutans (contradicting the notion).

The genesis for our research is the alarming decline in numbers of orangutans; their high rates of different types of developmental defect of enamel in comparison to other large apes; and, most particularly, the observation that orangutans from both Sumatra and Borneo commonly show repetitive episodes of linear enamel hypoplasia whose cause is unknown. If we can accept the support provided by crypt fenestration defects, that nutrition for orangutans is indeed better in Sumatra, we could conclude that more severe rLEH among Sumatran orangutans must be attributed to that other major cause of enamel hypoplasia-disease. If our results are valid then we can direct future research and fieldwork towards detecting a disease stressor in orangutan habitats with a mean duration of about two months and a periodicity of six months (or multiples thereof), but one which is more severe in Sumatra.

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Table I. Individual animals

	Sex			
Taxon	Male	Female	Unknown	Total
Pongo abelii	5[1,2,3]	4[2,3]	-	9
P. pygmaeus ssp.	3[4]	2[4]	-	5
P. p. pygmaeus	7[5,6]	8[5,6,7]	1[5]	16
P. p. wurmbii	1[8]	3[8,9]	-	4
P. p. morio	1[10]	-	-	1
Total	17	17	1	35

1. Zoological State Museum, Munich-2 males
2. Senckenberg Institute, Frankfurt-2 males, 3 females
3. Naturalis, Leiden-1 male, 1 female
4. Senckenberg Institute, Frankfurt-3 males, 2 females
5. Anthropological State Museum, Munich-6 males, 6 females, 1 unknown
6. Zoological State Museum, Munich-1 male, 1 female
7. Naturalis, Leiden-1 female
8. Naturalis, Leiden-1 male, 2 females
9. Zoological State Museum, Munich-1 female
10. Zoological State Museum, Munich-1 male

Table II. Teeth examined

		Independent LEH		
Island	Sex	Incisor	Canine	Total
Borneo	Male	32	30	62
	Female	31	31	62
	Unknown	-	9	9
Sumatra	Male	6	11	17
	Female	2	11	13
		71	99	163

Table III. Descriptive statistics for measurements of Width (µm) and Depth (µm) (all variables combined)

							Test of Normality			
							K-S[1]		S-W[2]	
Measure	N	Median	Mean	SD	CV	Skewness	Value	P value	Value	P value
Onset Width	163	369.0	378.3	150.0	39.7	0.420	0.048	0.200	0.982	0.036
Depth	163	18.61	23.3	15.6	67.0	1.294	0.139	<0.0001	0.885	<0.0001

1. Kolmogorov-Smirnov
2. Shapiro-Wilk's

Table IV. Tests of difference in mean measures (μm) for anatomic variables: parametric for widths, non-parametric for depths

Variable					Statistic		
A. Crown decile					t-test^1/ANOVA^2; M-W^3/K-W^4		
Width		N	Mean±SD	Median	Value	df	P value
	three	2	207.5±95.51	207.5			
	four	18	361.7±141.6	342.1			
	five	25	370.9±183.9	355.1			
	six	27	344.8±136.7	322.0			
	seven	32	413.9±134.9	424.8			
	eight	25	362.3±140.9	356.9			
	nine	24	387.1±141.6	372.6			
	ten	8	437.2±197.9	436.2	1.091^2	7	0.371
Depth							
	three	2	9.41±4.43	9.41			
	four	18	18.35±12.94	15.28			
	five	25	19.99±11.35	18.18			
	six	27	21.49±12.67	20.05			
	seven	32	26.39±19.79	17.77			
	eight	25	27.64±18.93	17.96			
	nine	24	25.09±13.75	21.67			
	ten	8	23.13±16.17	20.93	7.204^4	7	0.408
B. Sex							
Width							
	Male	79	361.9±148.0	355.7			
	Female	75	392.3±155.8	384.1	-1.243^1	152	0.216
Depth							
	Male	79	22.5±14.4	18.0			
	Female	75	24.8±17.2	19.3	-0.642^3	-	0.521
C. Tooth type							
Width							
	Incisor	71	360.5±166.8	347.1			
	Canine	92	392.0±135.0	382.6	-1.330^1	161	0.185
Depth							
	Incisor	71	21.2±16.1	16.6			
	Canine	92	24.8±15.0	21.1	-2.149^3	-	0.032

1. Students ‘t’
2. Analysis of variance
3. Mann-Whitney
4. Kruskall-Wallis

Table V. Distribution of independent LEH per decile compared between the two island samples of orangutans (sexes and tooth types combined)

	Island			
	Borneo[1]		Sumatra[1]	
Decile	N	Percent	N	Percent
One				
Two				
Three	2	1.5		
Four	15	11.5	3	10.0
Five	20	15.3	5	16.7
Six	23	17.6	4	13.3
Seven	26	19.9	6	20.0
Eight	19	14.5	6	20.0
Nine	20	15.3	4	13.3
Ten	6	4.6	2	6.7
Total	131		30	

1. Pearson Chi Square=1.545, df=7, P=0.981

Table VI. Tests of difference in means of LEH measures for Island variable: width measures (normally distributed) are untransformed values. Depths (non-normally distributed) are expressed as z scores of natural log of depth (parametric test) and untransformed depth (non-parametric test), permitting in both cases combination of incisors and canines.

Measure		Island	N	Mean± SD	Median	Value	df	P
Width	Original	Borneo	133	374.7±152.7	364.0			
		Sumatra	30	394.1±138.6	376.7	-0.640[1]	161	0.523
Depth	LogN	Borneo	133	-0.0908±0.940	-0.0280			
	tooth type	Sumatra	30	0.4025±1.151	0.5985	-2.487[1]	161	0.014
	Untransformed	Borneo	133		-0.3222			
	tooth type	Sumatra	30		0.3033	-2.509[2]	-	0.012

1. Student’s t
2. Mann-Whitney U

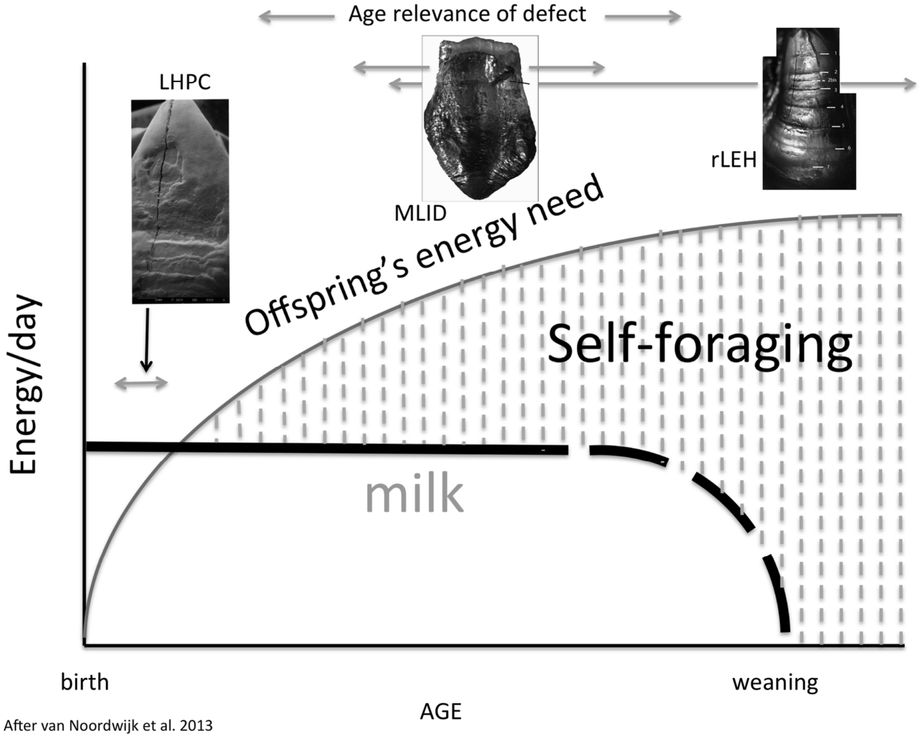

Fig. 1. Approximate age of creation of different types of enamel defects in orangutan infancy. LHPC (localized hypoplasia of the primary canine) and MLID (maxillary lateral incisor defect) are crypt fenestration defects reflecting undergrowth of the jaws. The etiology of rLEH (repetitive episodes of linear enamel hypoplasia) is not known (modified from van Noordwijk et al. 2003).

101x76mm (600 x 600 DPI)

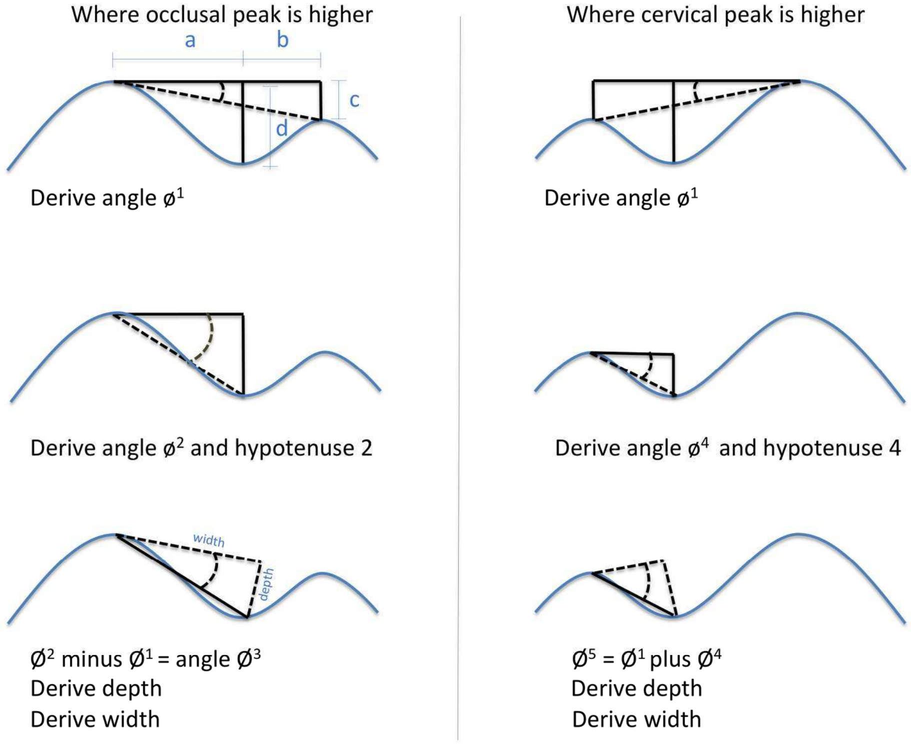

Fig. 2. Conversion of measurements of LEH furrows, relative to the plane of the scanner instrument, to measures of width and depth using trigonometry. Measures 'a' plus 'b' equals distance between two shoulder peaks, 'a'=distance of occlusal peak to deepest point of furrow, 'b'=distance from deepest point of furrow to cervical peak, 'c'=vertical difference in height between two shoulder peaks, 'd'=depth of furrow from higher peak. Vertical scale is greatly exaggerated for illustrative purposes.

101x77mm (600 x 600 DPI)

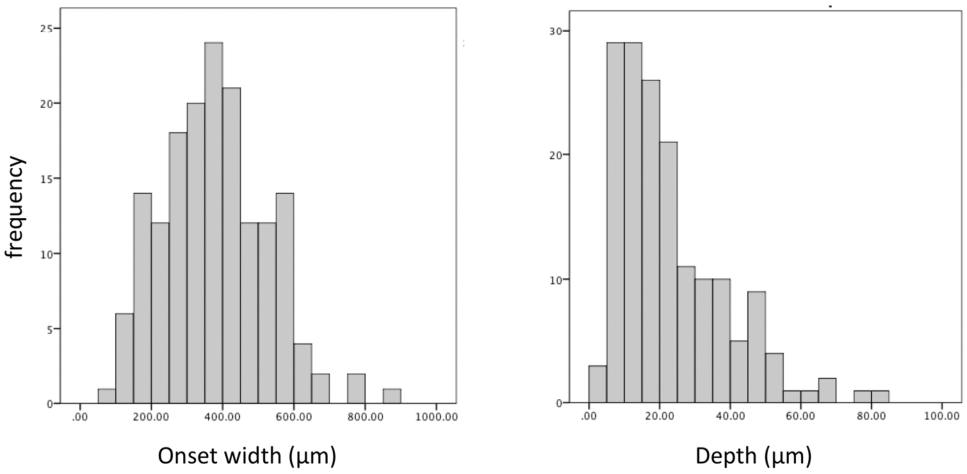

Fig. 3. Distribution of raw measurements (µm) of width and depth of episodes of linear enamel hypoplasia (combined tooth types, sexes, crown deciles and taxa). Widths are normally distributed while depths are decidedly non-normally distributed.

83x45mm (600 x 600 DPI)

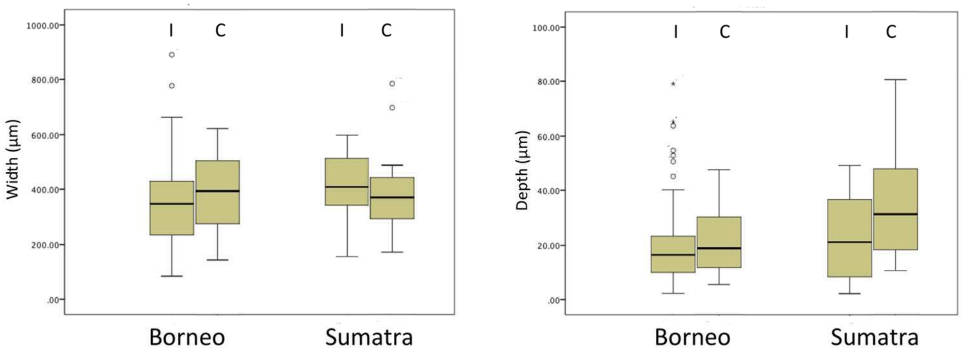

Fig. 4. Dispersion (quartiles) of raw measurements (µm) of width and depth separated by tooth type and island source (sexes combined). There is no statistically significant difference of width measurements among sub-groups(decile, sex, tooth type) but depth measurements do differ significantly between incisors and canine tooth types necessitating control of this variable in subsequent analyses (see Table IV).

59x23mm (600 x 600 DPI)

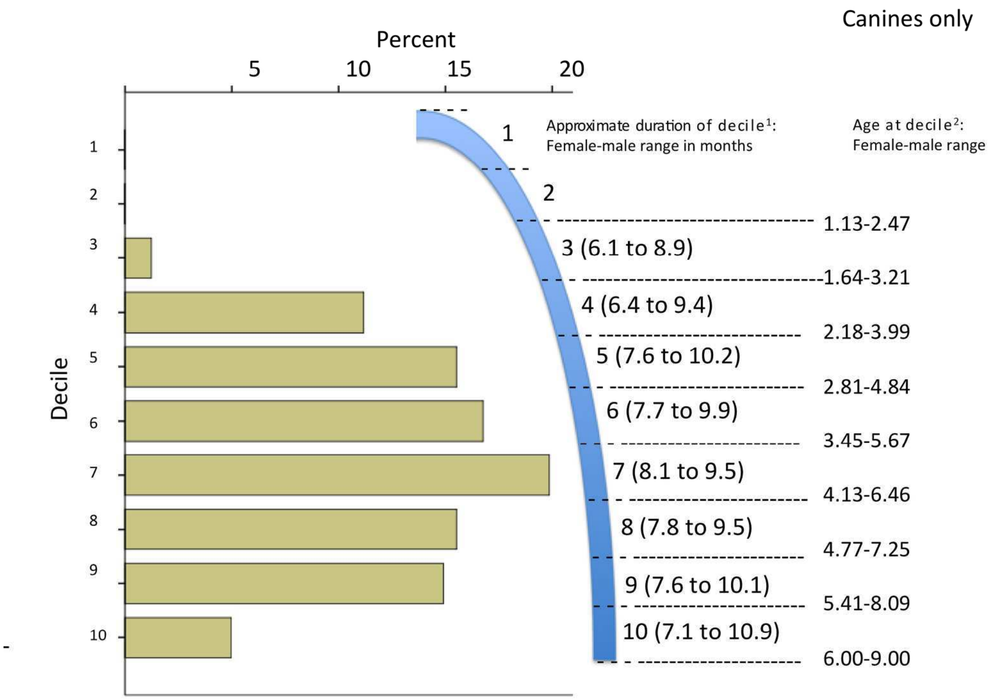

Fig. 5. Distribution of LEH among canine crown deciles. Approximate duration (months) of each decile is derived, with permission, from O’Hara [2016] and age at each decile is reconstructed by further recourse to Beynon et al. [1991]

101x76mm (600 x 600 DPI)

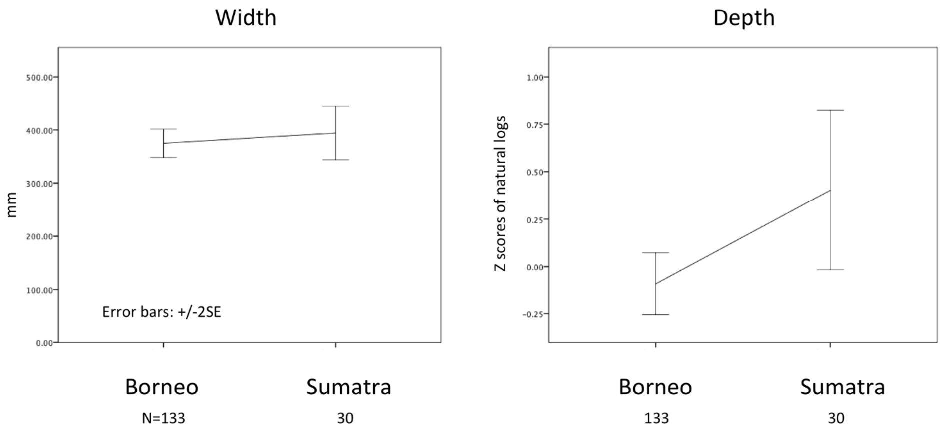

Fig. 6. LEH measurements compared between Bornean and Sumatran orangutans. Widths are raw measures combined all sub-groups of sex, decile and tooth type. Depths are normalized through natural log transformation and converted to z scores so as to combine tooth types. LEH from Sumatran orangutan animals are significantly deeper.

101x67mm (600 x 600 DPI)