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

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Manuscripts

Review

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5 1 **Title:** Orangutans, enamel defects, and developmental health: A comparison of Borneo and  
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7 2 Sumatra  
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45 18 **Text pages:** 35 (including bibliography), 6 figures, 6 tables  
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50 20 **Abbreviated title:** enamel hypoplasia in orangutans  
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23 **ABSTRACT**

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

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5 44 patterning, shared with apes from a wide range of ecological and temporal sources,  
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7 45 rLEH is more likely attributable to disease than to malnutrition.  
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11 47 **Key words:** *Pongo pygmaeus*; *P. abelii*; infancy; dentition; stress  
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18 50 **RESEARCH HIGHLIGHTS**  
19

20 51 Orangutans are disappearing for largely known reasons. Most dental defect types  
21  
22 52 support Sumatra being deemed a better habitat than Borneo. However, one enigmatic  
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24 53 defect, repetitive linear enamel hypoplasia, occurs on both islands but is more severe  
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26 54 in Sumatra.  
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## 56 INTRODUCTION

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

## 76 Developmental defects of enamel

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4 77 There is a strong link between developmental health of growing orangutans and  
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6 78 habitat quality [Knott, 1998; Delgado and van Schaik, 2000]. Enamel formation has  
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9 79 been the subject of numerous studies, e.g. [Osborn, 1981; Nanci, 2012]. Hypoplastic  
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11 80 defects of enamel are usually attributed to metabolic stress that results in abnormal  
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14 81 secretion of matrix prior to full mineralization of the enamel. It is widely accepted that  
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16 82 enamel hypoplasia is a non-specific marker of systemic stress [Goodman and Rose,  
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19 83 1990]. However, this rather bleak assessment can be mitigated somewhat by  
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21 84 distinguishing among the (admittedly complex) etiologies of different types of  
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24 85 hypoplastic defects as well as by asking whether the timing, severity, prevalence and  
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26 86 epidemiology of a defect type may be sufficiently distinctive as to suggest a specific  
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29 87 etiology. Our study is designed to explore the striking patterning of rLEH among  
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31 88 orangutans in the hope of elucidating etiology.

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34 8935  
36 90 *Timing of bone mass defects*

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38 91 'Localized hypoplasia of the primary canine' (LHPC) and 'maxillary lateral incisor  
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40 92 defect' (MLID) share a common proximate etiology-insufficient bone growth in the  
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43 93 face-but are created at different times in infancy (Fig. 1). Both LHPC and MLID are  
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45 94 crypt fenestration defects in which reduced bone growth in the face leads to creation  
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48 95 of an enamel defect prior to eruption [Skinner, 1986b; Skinner and Hung, 1989;  
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50 96 Skinner et al., 2014].

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55 98 LHPC is caused by fenestration of the labial crypt wall, normally protecting the  
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4 99 unerupted, forming milk canine crown, when cranio-facial bone growth in the infant  
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7 100 fails to keep up with crown formation [Skinner and Newell, 2003]. In humans and  
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10 101 apes, the affected part of the tooth crown, exposed to trauma through a fenestration,  
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12 102 forms in the months shortly after birth [Skinner and Newell, 2003; Stojanowski and  
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14 103 Carver, 2011]; consequently it is assumed that, among breast-feeding cohorts, LHPC  
15  
16 104 reflects condition of the mother as much as the infant. Approximately 91% of  
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19 105 orangutans are affected [Lukacs, 2001]. Notably, there is no difference between  
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21 106 Sumatran and Bornean orangutans in the occurrence of LHPC.  
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24 107  
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26 108 The second crypt fenestration defect, MLID, is attributed to abnormal contact of the  
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28 109 labial surface of the somewhat less-formed upper lateral incisor crown with the  
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31 110 incisal edge (or fenestration margin) of the more mineralized central incisor through a  
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33 111 fenestrated inter-crypt boney septum in under-developed jaws with pre-eruptive  
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36 112 dental crowding [Hannibal, 2016; Skinner et al., 2016]. MLID is created in the first few  
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38 113 years of an orangutan's life (ca. 2-5 years) when the infant is increasingly reliant on  
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40 114 foraging for itself. We define infancy as that period during which lactation occurs [van  
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43 115 Noordwijk et al., 2013] which lasts as long as 5.5 years in Bornean orangutans and 6-  
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45 116 7.5 years in Sumatran [van Noordwijk et al., 2009]. Notably, almost all incisor and  
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48 117 canine crown formation occurs within this age span except perhaps for the cervical  
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50 118 fifth of the male canine [Beynon et al., 1991]. Orangutans are markedly affected (59%)  
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52 119 by MLID but the lesion is far more common in Bornean orangutans (71%) than  
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55 120 Sumatran (29%) [Skinner et al., 2016].  
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7 122 Considering the occurrence of both LHPC and MLID, it can be concluded that: a)  
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9 123 nutrition for infants from both islands is more adequate when the infant is more fully  
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11 124 reliant on breast milk than in later infancy; and b) Sumatra provides a more suitable  
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13 125 nutritional habitat for infants than does Borneo. The latter inference is well supported  
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15 126 in the literature. There are several lines of ecological evidence indicating that Sumatra  
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17 127 provides a superior habitat for orangutans due, fundamentally, to volcanically-derived  
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19 128 soils [Wich et al., 2011]. In Sumatra there are more months in the year with high fruit  
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21 129 availability and a trend towards shorter low fruit periods [Delgado and van Schaik,  
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23 130 2000; Marshall et al., 2009; Wich et al., 2011]. Unlike Bornean orangutans, Sumatran  
24  
25 131 orangutans spend more time on high quality foods like fruit and insects and a lower  
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27 132 percentage on bark and vegetation. Moreover, Sumatran orangutans seem less reliant  
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29 133 on fallback foods than are Bornean, being able to find figs and fruit year round  
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31 134 [Russon et al., 2009]. In Borneo, there are months where fruit is a minor part of the  
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33 135 diet while in Sumatra fruit is always a major part of the diet [Morrogh-Bernard et al.,  
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35 136 2009]. Not surprisingly, therefore, orangutan population density is higher in Sumatra  
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37 137 [van Schaik et al., 2009].

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45 138 (Figure 1 about here)  
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50 140 *Timing of repetitive linear enamel defects*  
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53 141 The third type of enamel defect (linear enamel hypoplasia) is a more direct  
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55 142 manifestation of abnormal secretion in which transverse furrows of thinned enamel  
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4 143 are created. All ape samples, including fossil forms, commonly show several furrows  
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6 144 on the incisor and canine dental crowns. The remarkable ubiquity of repetitive LEH in  
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9 145 time and space, spanning millions of years (Miocene to present) and thousands of  
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11 146 kilometers from Spain to China and Africa [Skinner et al., 1995; Skinner and  
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13 147 Roksandič, 1995; Guatelli-Steinberg and Skinner, 2000; Brunet et al., 2002], suggests  
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15 148 that a pervasive and common stressor may underlie the phenomenon. However, we  
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17 149 should be explicit that the etiology of rLEH cannot as yet be attributed with  
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19 150 confidence to malnutrition and/or disease, the fundamental agents behind the  
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21 151 metabolic stress associated with enamel hypoplasia [Goodman and Rose, 1990].  
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28 153 LEH have been reported in the canine teeth of orangutans among whom they tend to  
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30 154 commence at about 2.5 years of age [Skinner and Hopwood, 2004] and recur  
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32 155 throughout crown formation [Skinner, 2014b] (up to about six to nine years of age  
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34 156 depending on sex [Beynon et al., 1991; Schwartz and Dean, 2001] (Fig. 1). Among  
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36 157 orangutans in general the stressful events recur on average about every six or twelve  
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38 158 months; Sumatran animals showing significantly more annual episodes of stress while  
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40 159 Bornean animals show more semi-annual episodes [Skinner, 2014b]; a pattern  
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42 160 interpreted to provide mild support for Sumatra being the better habitat. However,  
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44 161 Sumatran orangutans are reported to show more LEH defects per tooth [Guatelli-  
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46 162 Steinberg et al., 2012], which seems incompatible with their longer cycle (but which  
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48 163 may reflect different subjective thresholds of LEH visibility between investigators,  
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50 164 reinforcing our contention that measurement of LEH furrows should be pursued).  
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7 166 Roughly 83% of orangutans from museum collections show  $\geq 1$  episodes of LEH per  
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9 167 tooth [Hannibal and Guatelli-Steinberg, 2005]. When nearly all individual apes in both  
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11 168 Sumatra and Borneo are affected by rLEH, another way has to be found to measure  
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14 169 comparative developmental stress in an informative manner. A more telling test of  
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17 170 island differences would be to measure, not the prevalence or periodicity of the  
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19 171 episodic stress events, but their duration and intensity. We hypothesize that width  
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21 172 and depth measurements of LEH serving as proxies for duration and intensity of  
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24 173 stress, respectively, will be less in Sumatran orangutans, a prediction based on Borneo  
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26 174 being deemed the poorer habitat.

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29 17530  
31 176 **METHODS**

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33 177 Specimens of extant animals examined in this study consist only of skeletal remains  
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36 178 from museum collections; all animals were dead prior to our study. Proposed  
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38 179 examination was approved by curators of museums listed in Table I. All examinations  
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41 180 were performed at the institution and no hard tissue was removed or transported.  
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43 181 This research adheres to the American Society of Primatologists principles for the  
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45 182 ethical treatment of primates.

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50 184 Orangutans in this study have been previously described [Skinner, 2014b]. They come  
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53 185 from three museums in Germany and one in Holland whose collections were  
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55 186 examined in 1999 and 2000. The animals were taken from the wild in the late 19<sup>th</sup> and

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4 187 early 20<sup>th</sup> century; hence provenience information is imprecise or, in a few cases,  
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6 188 unknown. The majority of the Bornean animals come from the Sintang and Sekelau  
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8 189 region in eastern West Borneo. The Sumatran animals are from the far north end of  
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10 190 the island near Atjeh (Aceh) and Deli. Here we employ a species distinction between  
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12 191 the two island populations (*P. abelii* in Sumatra and *P. pygmaeus* in Borneo) reflecting  
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14 192 the marked genetic and morphological differences between the two island taxa  
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16 193 [Goossens et al., 2009].

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21 194 (Table I about here)  
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26 196 Animals for the previous study [Skinner, 2014b] were chosen because they showed  
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28 197 countable perikymata between two or more episodes of LEH on a single tooth (incisor  
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30 198 or canine). Where defects were demonstrably bilateral, the antimere with more  
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32 199 visible features on the outer enamel surface was chosen. An individual counts only  
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34 200 once. Here we report LEH defects on an enlarged sample of teeth from the same  
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36 201 animals to include slightly worn, but measurable, LEH with uncountable perikymata.  
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38 202 Still, the sample (Table I) is biased towards younger individuals with comparatively  
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40 203 little labial wear, and purposefully excludes animals whose teeth show only a single  
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42 204 episode of LEH, since the latter animals did not afford an opportunity to study the  
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44 205 interval between episodes and, hence, LEH periodicity. We acknowledge that sample  
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46 206 sizes are disparate and that for Sumatra (N=9 animals), small. However the sample  
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48 207 sizes for repetitive LEH events, whose occurrence we consider to be independent of a  
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50 208 previous event, are reasonably large (N=30 for Sumatra and 133 for Borneo).  
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4 209 Nevertheless, any inferences made in our study will need to be tested against other  
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6 210 and larger samples before firm conclusions can be drawn.  
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## 11 212 **Terminology**

13 213 Usages of the word 'stress' are considered so discordant that hopes of a standard  
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15 214 definition are forlorn [King and Murphy, 1985]. Our definition is not as strict as theirs-  
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17 215 that vital physiological function must be impaired-but we do consider that reduced  
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19 216 cellular secretion of enamel matrix, sufficient to affect contour of the outer enamel  
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21 217 surface, qualifies as indicative of physiological stress. While methods employed in this  
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23 218 study are a deliberate move away from a subjective threshold of perception of a  
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25 219 hypoplastic furrow towards measurement, we employ the term 'salience' to mean the  
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27 220 subjective visibility of depressions in the outer enamel surface (conflating width and  
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29 221 depth), often enhanced by vegetable staining, since this threshold has historically  
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31 222 guided our researches and has formed the basis of communication among scholars.  
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33 223 We should be clear however that the salience of linear enamel hypoplasia, whether  
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35 224 objectively or subjectively assessed, reflects the host animal's experience of stress  
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37 225 mediated by many factors, not stress *per se*. In other words, measurements of enamel  
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39 226 hypoplasia do not measure stress at all-they measure the response to stress; but  
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41 227 forging a link between stress and enamel defects is challenging. LEH reflects the  
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43 228 potential interaction, during development, of many factors (e.g., individual  
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45 229 immunocompetence, foraging efficiency, food acquisition skills and social rank);  
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47 230 meteorological influences (e.g., seasonality, insolation, rainfall cycles); and abiotic  
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4 231 factors (e.g., soil type) with a variety of stressors (e.g., disease, malnutrition)  
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7 232 [Eckhardt and Protch von Zieten, 1993; Guatelli-Steinberg, 1998, 2000; Guatelli-  
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9 233 Steinberg and Skinner, 2000; Guatelli-Steinberg, 2001; Chollet and Teaford, 2010;  
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11 234 Kirchoff, 2010]. Historically, there has been acceptance of a simple separation of  
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13 235 defects into narrow or wide [Sarnat and Schour, 1941; Corruccini et al., 1985;  
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15 236 Bermudez de Castro and Perez, 1995] with little or no concern for depth; and, yet,  
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17 237 depth contributes to the salience of an LEH. The latter authors speculated that narrow  
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19 238 and wide grooves might represent infection and dietary deficiency, respectively.  
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21 239 Blakey et al. [1994] used the phrase 'major growth arrests' to describe very wide  
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23 240 defects. Similarly, Ensor and Irish observed what they termed 'continuous chronic  
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25 241 enamel hypoplasia' and captured the phenomenon with the concept of 'total  
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27 242 hypoplastic area' [1995]. Many authors have invoked a threshold of 0.4 to 0.5mm  
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29 243 width of defect to distinguish shorter (acute) and longer episodes of stress  
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31 244 [Hutchinson and Larsen, 1988; Ensor and Irish, 1995; Duray, 1996; Vann, 2008].  
32  
33 245 There seems to be general acceptance that the width of a hypoplastic defect provides  
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35 246 a reasonable estimate of duration of a stress event or events. Here, we draw a clear  
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37 247 distinction between widths and depths.  
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47 248 *Width*

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49 250 The accepted method for estimating duration of stress in humans is to count the  
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51 251 number of perikymata in the occlusal wall of a defect and multiply this figure by the  
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53 252 known or inferred Retzius periodicity in days [Hillson and Bond, 1997]. We question  
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4 253 the generalization, made by Hubbard and colleagues [2009], that perikymata counting  
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6 254 is more accurate than measuring defect widths, mostly because in chimpanzee,  
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8 255 bonobo and orangutan canine teeth, at least, perikymata spacing is more uniform  
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10 256 throughout most (ca. middle 80%) of the crown than it is in human canine teeth [Dean  
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12 257 and Reid, 2001; Guatelli-Steinberg et al., 2012; O'Hara, 2016]. In our study, 94% of  
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14 258 LEH occur within crown deciles four through nine. According to a recent study by  
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16 259 O'Hara [2016] the median number of perikymata among these deciles ranges from  
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18 260 only 26 to 28 per decile. Also, a recent study reports a range of only nine or ten days  
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20 261 per perikyma in both Sumatran and Bornean orangutans [Smith, 2016]. Consequently,  
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22 262 we feel that measurements of the width of LEH defects should give a reasonable  
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24 263 estimate of duration of stress (as qualified above) in each taxon. Both perikymata  
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26 264 count and width are proxies for time. Fundamentally, it is much easier to obtain a  
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28 265 sufficiently large sample to detect differences between populations by measuring  
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30 266 widths than counting perikymata. As Hubbard and colleagues observe:  
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32 267 "Bioarchaeologists are faced with the choice of using a more accurate method  
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34 268 (perikymata counting) on a small sample (given the small number of defects with  
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36 269 continuously visible perikymata within them), or using a potentially less accurate  
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38 270 method (measuring defect widths) on a larger sample" [2009:178]. Here we have  
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40 271 elected to eschew perikymata counts in favour of width measurements, on an  
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42 272 enlarged sample.  
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46 274 We define 'width' as a direct measure, along an imaginary line joining the two  
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4 275 shoulders of a hypoplastic defect, taken from the occlusal shoulder to a place  
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7 276 orthogonal to the deepest point of a defect, taken in an occlusal-cervical axis. This  
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9 277 amounts to roughly half the width of most furrows. In our view, enamel deposition  
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11 278 after the end of a stressful episode restores normal enamel contour; consequently,  
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13 279 width of what can be termed the 'recovery of normal enamel contour phase' is a  
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15 280 function of both enamel geometry and depth of the furrow but is not necessarily  
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17 281 informative of the actual time required to recover from stress [Hillson and Bond,  
18  
19 282 1997]. For the purposes of this study, we do not distinguish between plane-form  
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21 283 versus furrow-form defects. Our study is based on furrow-form defects but it is  
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23 284 conceivable that some of the more worn LEH without countable perikymata in the  
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25 285 occlusal wall are actually plane-form defects.  
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32  
33 287 *Depth*

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35 288 Depth is deemed by us to be an indirect measure of the intensity of stress (as  
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37 289 mediated by anatomical factors-e.g., occlusal vs. cervical location [Hillson and Bond,  
38  
39 290 1997; Hubbard et al., 2009], environmental factors [Chollet and Teaford, 2010] and  
40  
41 291 individual frailty [King and Ulijaszek, 1999]). How do we know that the more severe a  
42  
43 292 stressor, the deeper will be a hypoplastic defect? Numerous studies support the  
44  
45 293 inference that there is a dose-dependent reaction of secretory phase ameloblasts to  
46  
47 294 increasing levels of a stressor: e.g., a) fluoride [Suckling and Thurley, 1984; Kierdorf et  
48  
49 295 al., 2004]; b) reduced age at death [White, 1978; Cook and Buiskstra, 1979; Goodman  
50  
51 296 and Armelagos, 1988; Duray, 1996]; c) reduced availability of fat soluble vitamins  
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4 297 (Mellanby M. 1929 cited in [Mellanby, 1941; Goodman et al., 1991] although the effect  
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6 298 here may be due to decreased bone growth impinging on the dental crown); d)  
7  
8  
9 299 parasitism [Suckling et al., 1986]; e) maternal hyperglycemia in diabetic rats [Silva-  
10  
11 300 Sousa et al., 2003]; and f) seasonal insolation [Zadsinska et al., 2013].  
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15  
16 302 The notable unevenness along the floor of an LEH furrow [Boyde, 1970] necessitates  
17  
18 303 multiple measurements; with the instrument described below, we take 516  
19  
20 304 measurements over a space of 1600 microns. We define depth as the orthogonal  
21  
22 305 distance from a plane connecting the two high points on the margins of a defect to its  
23  
24 306 deepest point. In this study, measurements are un-scaled since there are no  
25  
26 307 differences in average enamel thickness between island taxa [Smith et al., 2012].  
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### 32 309 **Impressions and casts**

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35 310 Enamel was cleaned of surface residue with dilute acetone. Molds of enamel surfaces  
36  
37 311 affected with rLEH were taken in Coltene President Plus Jet impression material,  
38  
39 312 supported by Coltene Lab Putty and polysiloxane activator (Coltene® , Cuyahoga  
40  
41 313 Falls, Ohio, USA). Casts were made in Araldite MY 753 epoxy resin with XD 716  
42  
43 314 hardener (Ciba-Geigy®, Toms River, New Jersey, USA). Close-up photographs of each  
44  
45 315 tooth cast were combined into a photomontage with Adobe Photoshop Elements 10  
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47 316 (Adobe®, San Jose, California, USA).  
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### 53 318 **Identification of LEH events**

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4 319 Irregularities of the enamel surface were visualized in the first instance under oblique  
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6 320 lighting at low magnification (ca. 6X). Macroscopically visible LEH are the focus of our  
7  
8 321 study. Our threshold for a measurable LEH does not include every minor vicissitude  
9  
10 322 involving, for example, just a single perikyma. Low power (ca. 10-15X) pictures of a  
11  
12 323 whole crown were recorded and the rLEH provisionally numbered consecutively  
13  
14 324 starting at the apex: 1, 2, and so on. The task of matching LEH between low and high  
15  
16 325 magnification pictures was accomplished by noting small irregularities (e.g.,  
17  
18 326 scratches) or imperfections in the cast (e.g., bubbles) that could be located on both  
19  
20 327 images. A visual comparison was made of LEH salience between scanner  
21  
22 328 photomontages and the lower power photographs and sketches, and a final decision  
23  
24 329 made as to the location and number of LEH events (Table II). Only one tooth per  
25  
26 330 individual was employed so as to avoid statistical redundancy (i.e., where one event  
27  
28 331 might be recorded on both an incisor and canine from a single individual); canines  
29  
30 332 being given preference as they typically show more LEH per crown.  
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38 (Table II about here)  
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#### 41 **Instruments**

42  
43 335 Epoxy casts were examined with a 'µ surf Mobile Plus' optical scanner (OS) and  
44  
45 336 analysed with µ soft Analysis Premium 6.2 software from NanoFocus® AG  
46  
47 337 (Oberhausen, Germany). This instrument enables the analysis of 3-D structures and  
48  
49 338 geometries in the micrometer and nanometer range. The precise 3-D topography is  
50  
51 339 computed from the acquisition of a large number of confocal filtered height sections  
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53 340 (typically ≥600). The OS consists of a compact confocal probe mounted on a stable L-  
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4 341 stand with motorized movement to focus in the z-axis (maximum resolution = 1 nm).  
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6 342 The sample to be measured is secured to an x/y precision measurement table. For  
7  
8 343 contactless measurement of surface topography, a sample is positioned on the  
9  
10 344 measurement table and the confocal unit moved stepwise in the z axis. In this  
11  
12 345 instrument, magnification was usually performed with a 10X lens that provides a  
13  
14 346 square field of view 1600 $\mu$ m on a side. Width and depth measurement outputs are  
15  
16 347 averages, calculated by the instrument, from 516 measurements over this space. Prior  
17  
18 348 to trigonometric analysis, scanner images were leveled, missing points filled in, and  
19  
20 349 form removed. The latter step optimizes measurement of defect depth by minimizing  
21  
22 350 the effect of object curvature.  
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### 31 **Data Manipulation (trigonometry)**

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33 353 It was deemed desirable to determine true depths, not depth in relation to the  
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35 354 instrument plane, since the object's surface is rarely level or completely flat. This is  
36  
37 355 performed trigonometrically from width and depth measures originally taken  
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39 356 orthogonal to the instrument's plane (Fig. 2).  
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43 (Figure 2 about here)  
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### 47 **Anatomical factors**

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50 360 As illustrated by Guatelli-Steinberg [2001, 2003], where striae of Retzius emerge at an  
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52 361 acute angle with the outer enamel surface (typical of more occlusal imbricational  
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54 362 enamel compared to cervical, especially in incisors), all else being equal, LEH width  
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4 363 occlusally is increased and apparent salience, decreased. In order to expedite  
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6 364 comparisons of defect salience between taxa, we first compare measures of defect  
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8 365 width and depth among crown deciles.  
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10 366  
11  
12 367 Since we would like to know if LEH salience compared along the crown reflects real  
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14 368 differences in the intensity of experienced stress or simply constraints of enamel  
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16 369 geometry , we need to measure the obligatory effect on furrow depth created by a  
17  
18 370 narrowing of an incremental stria of enamel. A simple trigonometric calculation (ratio  
19  
20 371 of sine angles) shows that changing stria angle from 10° (near the occlusal tip of a  
21  
22 372 canine tooth) to 45° (nearer the cervix) [Guatelli-Steinberg et al., 2012], reduces depth  
23  
24 373 of a defect at the cervix to about 0.72 of the depth near the occlusal tip. The lesson  
25  
26 374 here is that, in furrow-form defects where incremental striae are narrowed, the same  
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28 375 amount of stress will produce shallower defects near the cervix. Consequently, we  
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30 376 suggest, as a reasonable threshold, that only if more cervically-located LEH are deeper  
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32 377 than defects near the occlusal tip, is a real difference in the felt intensity of stress  
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34 378 being signaled.  
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### 380 **Statistical analysis**

381 Preliminary analysis, using Kolmogorov-Smirnov and Shapiro-Wilks tests, showed  
382 that width measures are normally distributed while depths are not, being significantly  
383 left skewed (Fig. 3, Table III). Consequently, statistical analysis of difference between  
384 mean widths relies on parametric (two-tailed Student's t, ANOVA) tests on original

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4 385 values; while depths are evaluated with non-parametric tests (Mann-Whitney and  
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6 386 Kruskal-Wallis) on original values or, alternatively, original values are converted to  
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9 387 natural logs so that the normalized distribution can be evaluated with parametric  
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11 388 tests. Given significant variation in LEH measures between tooth types (incisors and  
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13 389 canines) original measurements are also converted to z-scores (value minus mean for  
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15 390 a particular combination of tooth type divided by standard deviation for that  
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17 391 category) in order to maximize sample size. Alpha is set at 0.05.

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21 392 (Table III about here)

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23  
24 393 (Figure 3 about here)

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## 27 395 **RESULTS**

### 28 29 30 396 **Anatomic Variables: Crown decile, Sex, Tooth type**

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32 397 Our goal is to see if LEH defects are, as hypothesized, wider and deeper in Bornean  
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34 398 orangutans; i.e., that Sumatra provides the better developmental habitat for infant  
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36 399 orangutans. Firstly, however, we need to test whether one has to control for basic  
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38 400 anatomical variables: location of LEH within the tooth crown (crown deciles  
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40 401 numbered from occlusal to cervical), sex (male vs. female) and tooth type (incisors vs.  
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42 402 canines). Sample sizes are not sufficient to test each sub-group separately; so, initially,  
43  
44 403 we consider simply each variable by itself, lumping the other variables. The following  
45  
46 404 analysis of difference between means (for each of the three variables of crown  
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48 405 location, sex and tooth type) employs parametric tests for widths and non-parametric  
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50 406 statistics for depth (Table IV). We found that measures of LEH width do not vary  
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54 407 significantly among deciles, sex, or tooth types. Depths vary significantly only between  
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4 408 tooth types (Fig. 4). Subsequent analyses of both width and depth ignore LEH location  
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6  
7 409 and sex. Since depths are not only non-normally distributed but also vary as a function  
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9 410 of tooth type, statistical analysis requires control of these variables.

11 (Table IV about here)

13 (Figure 4 about here)

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19 414 Given relative stasis in the maternal contribution to the infant orangutan's diet [van  
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21 415 Noordwijk et al., 2013], the growing infant must rely increasingly on its own  
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23 416 resources (both foraging ability and immunity to disease). Thus, it can be predicted  
24  
25 417 that later LEH events will be more severe. However, as noted above, the depth of an  
26  
27 418 LEH is naturally decreased towards the cervix of a tooth due to the angle with which  
28  
29 419 striae reach the outer enamel surface. In our study of the canine teeth, the ratio of  
30  
31 420 decile 4 (more occlusal) to decile 9 (more cervical defect) median depths is 0.49-  
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33 421 notably less than the value of 0.72 predicted by enamel geometry-suggesting that the  
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35 422 intensity of the stressor in real terms increases with age of the animal (albeit  
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37 423 episodically) thus agreeing with the prediction.  
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#### 45 **Island variable**

46  
47 426 It was shown in Table IV that width and depth of LEH furrows do not vary  
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49 427 significantly as a function of their location on a tooth crown. If larger or different  
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51 428 samples were obtained this finding may not be confirmed, in which case it would be  
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53 429 desirable to know whether the distribution of LEH along the crown does or does not  
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4 430 differ between island populations. It is clear from Figure 5 and Table V that both

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6  
7 431 island samples in this study show the same distribution of LEH along the crown.

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9 432 (Figure 5 about here)

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11 433 (Table V about here)

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16 435 In this study, widths and depths of LEH are considered to be proxies for duration and

17  
18 436 intensity of felt stress, respectively. There is no difference in measures of width

19  
20 437 between the Bornean and Sumatran orangutans; but there is a clear difference in

21  
22 438 depths (Table VI). LEH defects among Sumatran orangutans are significantly deeper

23  
24 439 than those for Bornean orangutans-difference between medians = 10.0 $\mu$ m. However,

25  
26 440 it must be remembered that, while there are no significant differences in depths from

27  
28 441 different portions of the crown, depths do differ between tooth types (slightly but

29  
30 442 significantly deeper on canines) (Fig. 4). For this reason, Table VI includes a section in

31  
32 443 which depth measures are expressed as z-scores (that is, deviation of either a natural

33  
34 444 log of depth or untransformed measure from the mean for each sub-group of tooth

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36 445 type (incisors and canines)) and subjected to parametric and non-parametric tests of

37  
38 446 difference in means. This manipulation permits lumping of both tooth types,

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40 447 confirming that depths are significantly shallower on average in Bornean orangutans

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42 448 (Student's  $t=-2.487$ ,  $df=161$ ,  $P=0.014$ ; Mann-Whitney= $-2.509$ ,  $P=0.012$ ) (Fig. 6).

43  
44 449 (Table VI about here)

45  
46 450 (Figure 6 about here)

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4 452 While our purpose here is not to convert measured LEH widths directly into precise  
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6 453 measures of duration, but to compare two samples for relative durations, we can  
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9 454 nevertheless ask if the observed mean widths are at all compatible with what we  
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11 455 know about perikymata widths and Retzius periodicity. In our study, mean width of a  
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13 456 single perikyma is 73 $\mu$ m for incisors (N=4 in mid-crown third) and 69 $\mu$ m for canines  
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15  
16 457 (N=3 in mid and cervical thirds) (cf., mean of 68 to 84 $\mu$ m reported by O'Hara [2016]).  
17  
18 458 Dividing mean width by the corresponding perikyma widths noted above yields a  
19  
20 459 rough estimate of 4.9 and 5.5 perikymata per LEH, respectively, which (assuming that  
21  
22 460 one perikyma represents nine to ten days [Schwartz et al., 2001; Smith, 2016]),  
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24 461 suggests stress lasts six to eight weeks in both island taxa.  
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## 31 463 **DISCUSSION**

32  
33 464 Our hypothesis that Bornean orangutans would show wider and deeper hypoplastic  
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35 465 defects than do Sumatran orangutans is not supported by our results. However, there  
36  
37 466 are several caveats that must be considered before accepting such results. Firstly, our  
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39 467 study is of specifically repetitive linear enamel hypoplasia, not LEH in general; i.e., this  
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41 468 cohort may be biased towards more susceptible individuals. Secondly, in that widths  
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43 469 of LEH do not differ between the island samples but depths do, we have to ask  
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45  
46 470 whether these simply reflect Type II and Type I errors, respectively. The relative cost  
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48 471 of a Type I error is more than that for a Type II error since, for the latter, a real  
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50 472 difference will manifest itself after further studies [Toft and Shea, 1983]. To avoid  
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52 473 Type I error one can invoke a higher alpha value than, say, 1 in 20 (0.05). As may be  
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4 474 seen in Table VI, the observed P value for island orangutan differences in depth is  
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7 475 <0.02, meeting this requirement. Thirdly, we can use power analysis to determine if  
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10 476 our sample sizes are adequate. Depths of LEH are much more variable than are widths  
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12 477 (Table III); consequently, selection of an adequate sample size, in which both will be  
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14 478 compared between populations, will be dictated by variability in depths. Depths,  
15  
16 479 however, are not normally distributed. Power analysis assumes a normal distribution.  
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18  
19 480 Natural log transformation of observed depths normalizes the distribution from  
20  
21 481 which one can determine the median, and difference between the median and 68<sup>th</sup>  
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23 482 percentile of the distribution. Antilogs of these values (18.6 $\mu$ m and 7.4 $\mu$ m,  
24  
25 483 respectively) approximate the mean and standard deviation of a normal distribution.  
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28 484 Power analysis indicates that sample sizes required to detect a real difference in mean  
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31 485 depths of LEH between two populations (with a SD of 7.4 $\mu$ m and optional delta value  
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33 486 of 5 $\mu$ m) are 35 LEH for each island taxon. Our sample of Sumatran orangutan LEHs is  
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36 487 30, a bit below the required minimum. However, it is permissible [Motulsky, 1995] to  
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38 488 reduce the required size of one sample by 25% (from, say, N=35 Sumatran orangutans  
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41 489 to N= 26) if one doubles the size of the other; i.e., to N $\geq$ 70 Bornean. In our case, the  
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43 490 Bornean sample is 133 events, which means our study samples should be quite  
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45 491 sufficient to detect a minimum difference in mean depths, between samples, of only  
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48 492 5 $\mu$ m. Our observed difference in mean and median LEH depth values between island  
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50 493 populations of orangutans is 10.2 $\mu$ m and 10.0 $\mu$ m, respectively. We conclude that our  
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53 494 findings are robust, with the caveats noted above.  
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4 496 Both island taxa exhibit LEH of similar duration (six to eight weeks on average). This  
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7 497 result is surprising in that previously published estimates of duration of 'whole  
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9 498 furrows' (including width as defined in this study and the recovery of enamel contour  
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11 499 phase) were on the order of six to seven weeks [Skinner and Hopwood, 2004]; in  
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13 500 other words, the current results suggest that stress lasts longer than previously  
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16 501 thought.

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21 503 Contrary to expectation, Sumatran orangutans show significantly deeper/more severe  
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23 504 LEH compared to Bornean (median =28 $\mu$ m, 18 $\mu$ m, respectively). While, conceivably,  
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26 505 Bornean orangutans, who show more reliance on abrasive fall-back foods, might wear  
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28 506 away more enamel surface, the amount of wear required to render uncountable  
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31 507 perikymata, which are only about one micron in surface relief, is so minimal that the  
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33 508 effect on furrow depth is probably not germane to our study. As noted in the  
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36 509 introduction, there are very few published measurements of LEH depth in non-human  
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38 510 primates. Interestingly, median depth of LEH among only three Fongoli chimpanzees  
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41 511 (18 LEH), with a marked dry season, is 32 $\mu$ m (excluding plane-form defects) [Skinner  
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43 512 and Pruett, 2012], only slightly more than in Sumatran orangutans (median = 28 $\mu$ m).  
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45 513 The shallower LEH of Bornean orangutan LEH compared to both Sumatran  
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47 514 orangutans and Fongoli chimpanzees remains to be understood.

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52 516 We may ask 'How consistent are the studies of enamel hypoplasia in providing  
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55 517 support for the notion that Sumatra provides a better habitat?' They are not. As  
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4 518 reviewed earlier LHPC, attributed to facial bone thinning in early infancy, does not  
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7 519 differ between the islands while MLID, attributed to undergrowth of the maxilla, is  
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9 520 significantly worse in Bornean orangutans. Together, these findings suggest that, as  
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11 521 the growing infant is forced by static maternal milk reserves [van Noordwijk et al.,  
12  
13 522 2009] to forage for itself, cranio-facial growth falters more in Borneo (supporting the  
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15 523 notion). However, the current study shows that LEH furrows do not differ in width  
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17 524 (equated here with duration) between the island populations; whereas depth  
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19 525 (equated with intensity of stress) is significantly more marked in Sumatran  
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21 526 orangutans (contradicting the notion).  
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28 528 The genesis for our research is the alarming decline in numbers of orangutans; their  
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30 529 high rates of different types of developmental defect of enamel in comparison to other  
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32 530 large apes; and, most particularly, the observation that orangutans from both Sumatra  
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34 531 and Borneo commonly show repetitive episodes of linear enamel hypoplasia whose  
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36 532 cause is unknown. If we can accept the support provided by crypt fenestration defects,  
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38 533 that nutrition for orangutans is indeed better in Sumatra, we could conclude that  
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40 534 more severe rLEH among Sumatran orangutans must be attributed to that other major  
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42 535 cause of enamel hypoplasia-disease. If our results are valid then we can direct future  
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44 536 research and fieldwork towards detecting a disease stressor in orangutan habitats  
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46 537 with a mean duration of about two months and a periodicity of six months (or  
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48 538 multiples thereof), but one which is more severe in Sumatra.  
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6  
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Table I. Individual animals

<u>Taxon</u>	<u>Sex</u>			<u>Total</u>
	<u>Male</u>	<u>Female</u>	<u>Unknown</u>	
<i>Pongo abelii</i>	5 <sup>1,2,3</sup>	4 <sup>2,3</sup>	-	9
<i>P. pygmaeus ssp.</i>	3 <sup>4</sup>	2 <sup>4</sup>	-	5
<i>P. p. pygmaeus</i>	7 <sup>5,6</sup>	8 <sup>5,6,7</sup>	1 <sup>5</sup>	16
<i>P. p. wurmbii</i>	1 <sup>8</sup>	3 <sup>8,9</sup>	-	4
<i>P. p. morio</i>	1 <sup>10</sup>	-	-	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

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

For Peer Review

Table III. Descriptive statistics for measurements of Width ( $\mu\text{m}$ ) and Depth ( $\mu\text{m}$ ) (all variables combined)

<u>Measure</u>	<u>N</u>	<u>Median</u>	<u>Mean</u>	<u>SD</u>	<u>CV</u>	<u>Skewness</u>	<u>Test of Normality</u>			
							<u>K-S<sup>1</sup></u>		<u>S-W<sup>2</sup></u>	
							<u>Value</u>	<u>P value</u>	<u>Value</u>	<u>P value</u>
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

For Peer Review



Table IV. Tests of difference in mean measures ( $\mu\text{m}$ ) for anatomic variables: parametric for widths, non-parametric for depths

Variable		Statistic				
<u>A. Crown decile</u>		t-test <sup>1</sup> /ANOVA <sup>2</sup> ; M-W <sup>3</sup> /K-W <sup>4</sup>				
<b>Width</b>	<u>N</u>	<u>Mean±SD</u>	<u>Median</u>	<u>Value</u>	<u>df</u>	<u>P value</u>
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 <sup>2</sup>	7	0.371
<b>Depth</b>						
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 <sup>4</sup>	7	0.408
<u>B. Sex</u>						
<b>Width</b>						
Male	79	361.9±148.0	355.7			
Female	75	392.3±155.8	384.1	-1.243 <sup>1</sup>	152	0.216
<b>Depth</b>						
Male	79	22.5±14.4	18.0			
Female	75	24.8±17.2	19.3	-0.642 <sup>3</sup>	-	0.521
<u>C. Tooth type</u>						
<b>Width</b>						
Incisor	71	360.5±166.8	347.1			
Canine	92	392.0±135.0	382.6	-1.330 <sup>1</sup>	161	0.185
<b>Depth</b>						
Incisor	71	21.2±16.1	16.6			
Canine	92	24.8±15.0	21.1	-2.149 <sup>3</sup>	-	0.032

1. Students 't'
2. Analysis of variance
3. Mann-Whitney
4. Kruskal-Wallis

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

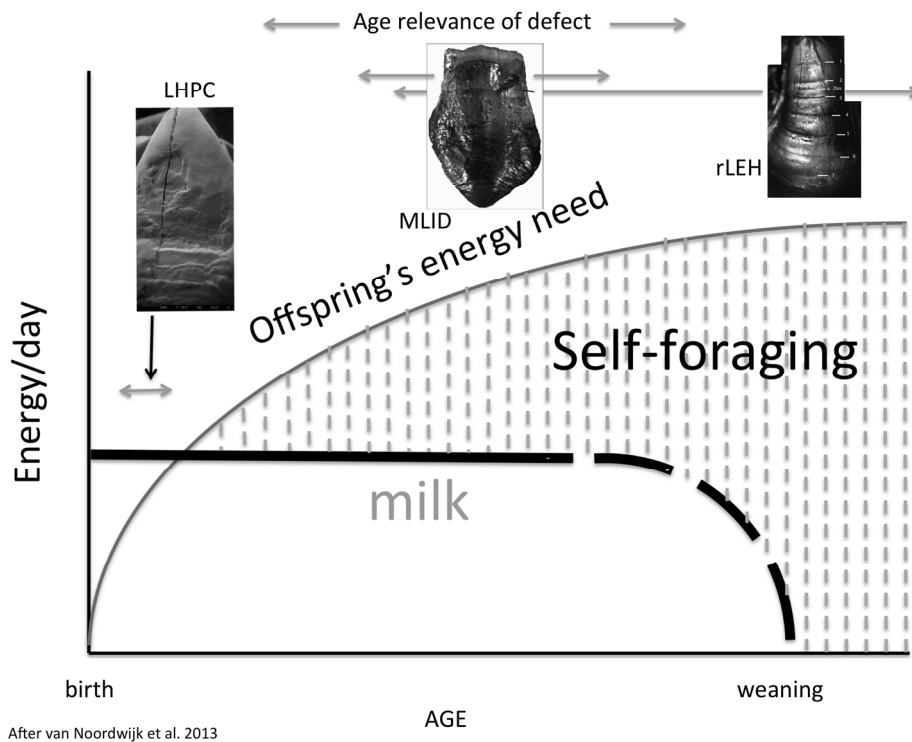
Decile	Island			
	Borneo <sup>1</sup>		Sumatra <sup>1</sup>	
	<u>N</u>	<u>Percent</u>	<u>N</u>	<u>Percent</u>
One				
Two				
Three	2	1.5		
Four	15	11.5	3	10.0
Five	20	15.3	5	16.7
Six	23	17.6	4	13.3
Seven	26	19.9	6	20.0
Eight	19	14.5	6	20.0
Nine	20	15.3	4	13.3
Ten	<u>6</u>	4.6	<u>2</u>	6.7
Total	131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

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

1. Student's t
2. Mann-Whitney U



After van Noordwijk et al. 2013

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)

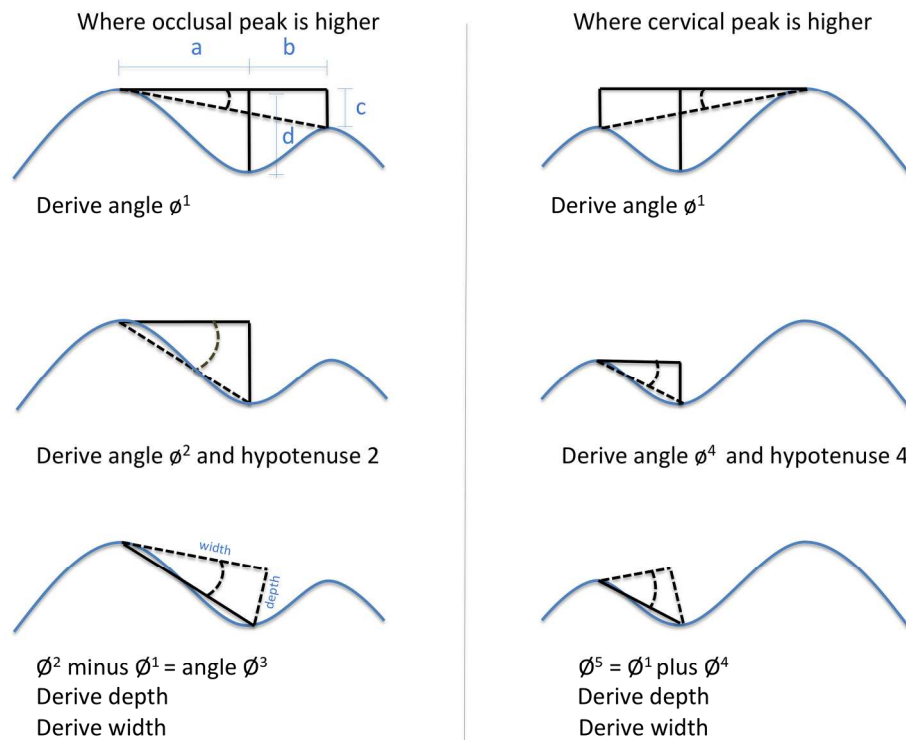


Fig. 2. Conversion of measurements of LEH furrows, relative to the plane of the scanner instrument, to measures of width and depth using trigonometry. Measures 'a' plus 'b' equals distance between two shoulder peaks, 'a'=distance of occlusal peak to deepest point of furrow, 'b'=distance from deepest 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.

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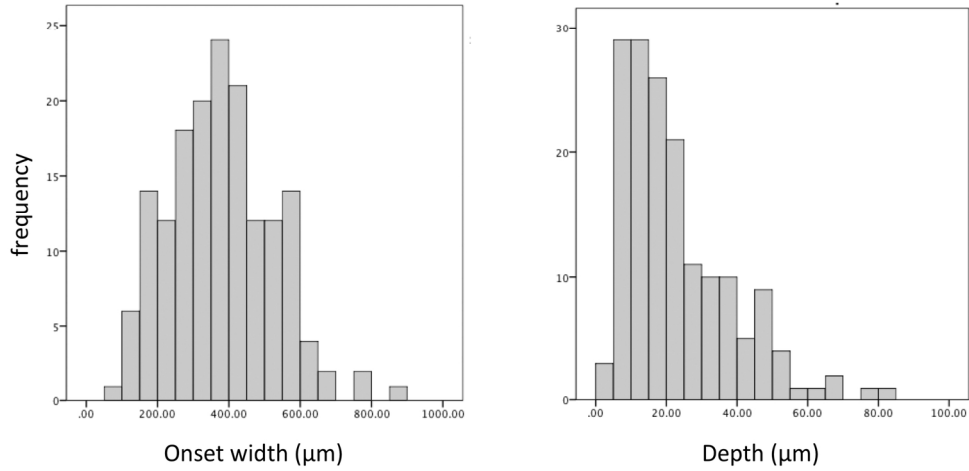


Fig. 3. Distribution of raw measurements ( $\mu\text{m}$ ) of width and depth of episodes of linear enamel hypoplasia (combined tooth types, sexes, crown deciles and taxa). Widths are normally distributed while depths are decidedly non-normally distributed.

83x45mm (600 x 600 DPI)

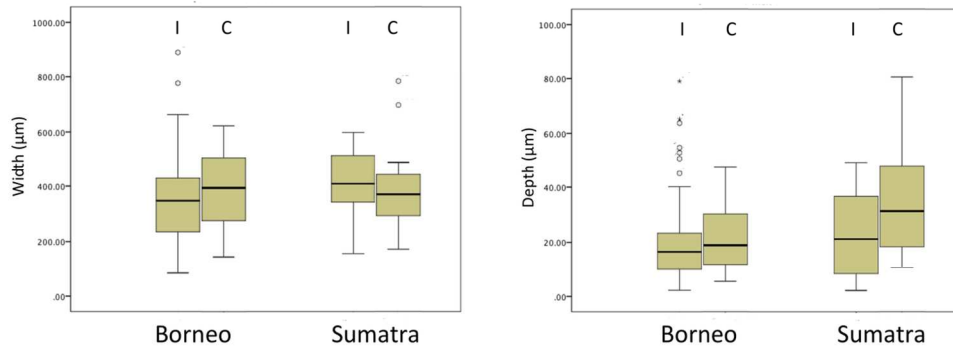


Fig. 4. Dispersion (quartiles) of raw measurements ( $\mu\text{m}$ ) of width and depth separated by tooth type and island source (sexes combined). There is no statistically significant difference of width measurements among sub-groups(decile, sex, tooth type) but depth measurements do differ significantly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

59x23mm (600 x 600 DPI)

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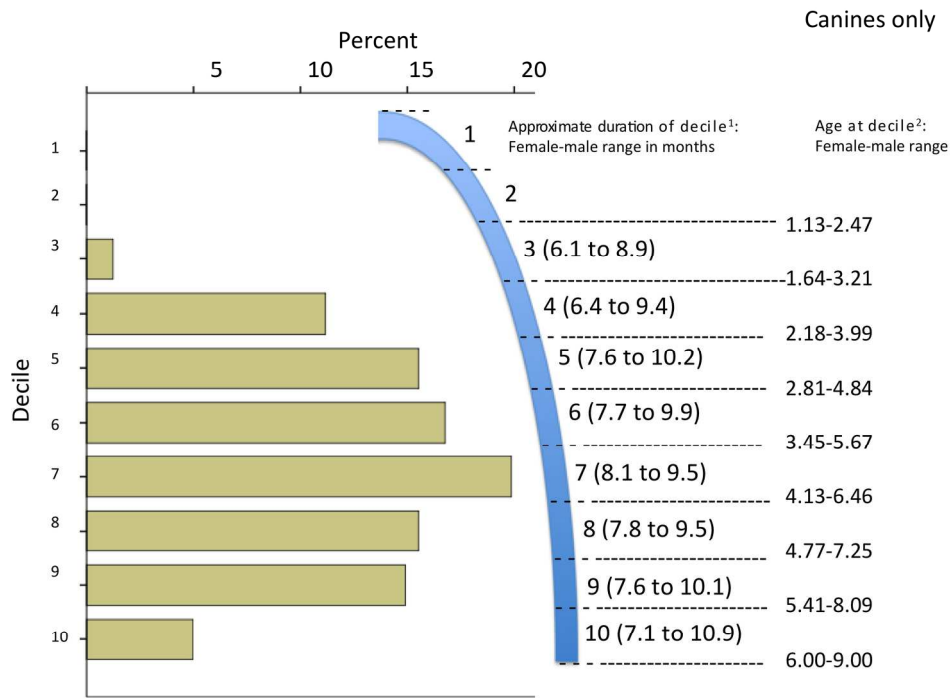


Fig. 5. Distribution of LEH among canine crown deciles. Approximate duration (months) of each decile is derived, with permission, from O'Hara [2016] and age at each decile is reconstructed by further recourse to Beynon et al. [1991]

101x76mm (600 x 600 DPI)



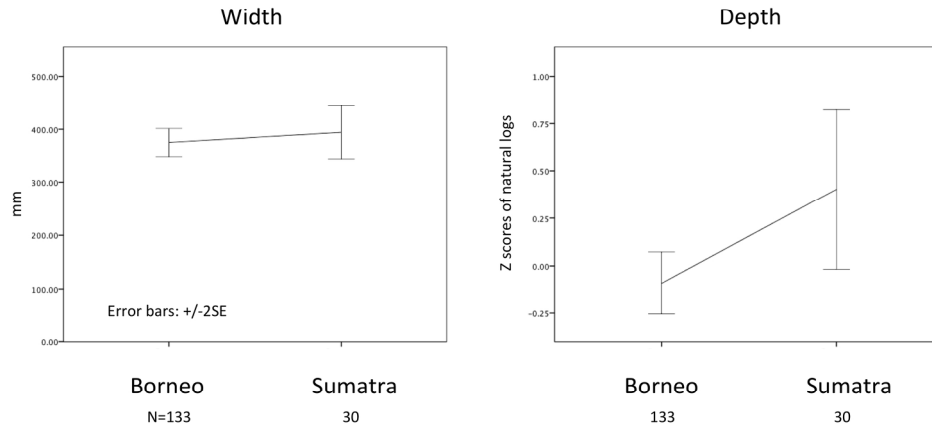


Fig. 6. LEH measurements compared between Bornean and Sumatran orangutans. Widths are raw measures combined all sub-groups of sex, decile and tooth type. Depths are normalized through natural log transformation and converted to z scores so as to combine tooth types. LEH from Sumatran orangutan animals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

101x67mm (600 x 600 DPI)