



LJMU Research Online

Towle, I and Irish, JD

Recording and interpreting enamel hypoplasia in samples from archaeological and palaeoanthropological contexts

<http://researchonline.ljmu.ac.uk/id/eprint/12281/>

Article

Citation (please note it is advisable to refer to the publisher's version if you intend to cite from this work)

Towle, I and Irish, JD (2020) Recording and interpreting enamel hypoplasia in samples from archaeological and palaeoanthropological contexts. Journal of Archaeological Science, 114. ISSN 0305-4403

LJMU has developed **LJMU Research Online** for users to access the research output of the University more effectively. Copyright © and Moral Rights for the papers on this site are retained by the individual authors and/or other copyright owners. Users may download and/or print one copy of any article(s) in LJMU Research Online to facilitate their private study or for non-commercial research. You may not engage in further distribution of the material or use it for any profit-making activities or any commercial gain.

The version presented here may differ from the published version or from the version of the record. Please see the repository URL above for details on accessing the published version and note that access may require a subscription.

For more information please contact researchonline@ljmu.ac.uk

<http://researchonline.ljmu.ac.uk/>

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28

Recording and interpreting enamel hypoplasia in samples from archaeological and palaeoanthropological contexts

Ian Towle a,b,* , Joel D. Irish b,c

a Sir John Walsh Research Institute, Faculty of Dentistry, University of Otago, Dunedin, 9016, New Zealand

b Research Centre in Evolutionary Anthropology and Palaeoecology, School of Biological and Environmental Sciences, Liverpool John Moores University, L3 3AF, United Kingdom

c Evolutionary Studies Institute and Centre for Excellence in PaleoSciences, University of the Witwatersrand, Private Bag 3, WITS, 2050, South Africa

Number of text pages: 13

Number of reference pages: 6

Number of figures: 5

Number of Tables: 2

Declarations of interest: none

29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

Abstract

Enamel hypoplasia is often split into several macroscopic categories, including pit, localised, linear and plane-form defects. All types have been considered a sign of 'non-specific stress' during dental development in archaeological, as well as palaeoanthropological and other samples. There is growing evidence suggesting many defects may not be caused by illness or malnutrition during childhood, instead relating to trauma to the developing tooth, genetic conditions or specific environmental factors, i.e., may not be associated with 'stress' to the individual. In this study all types of macroscopic enamel hypoplasia were recorded, including pitting, linear, plane and localised type defects, in three extant primate species and three fossil hominin species. The aim is to compare the characteristics and prevalence of different types of enamel hypoplasia among species and discuss potential differences in aetiology. The results show that samples have diverse prevalences of different kinds of defects, and pitting, linear and localised defects likely have different aetiologies. Additionally, dental characteristics (e.g., tooth morphology, developmental timing/speed and enamel structure) heavily influence the likelihood of specific types of enamel hypoplasia forming. In sum, studies that include only one type of enamel hypoplasia, or focus on one tooth type, to generate a 'stress' rating for a sample may miss relevant information when comparing groups. Instead, it may be beneficial to record different types of defects separately, for all teeth, and then consider how genetic, environmental and tooth property factors may influence population differences.

Key words: Dental defects; stress; dental development; Amelogenesis imperfecta; localised enamel hypoplasia; pitting enamel hypoplasia; linear enamel hypoplasia; fossil hominins; primates

61

62 **1. Introduction**

63 Enamel hypoplasia is defined as the reduction of enamel thickness caused by cessation or
64 diminution of ameloblast function during the secretory stage of enamel formation (Guatelli-
65 Steinberg, 2015; Ten Cate, 1994; Xing et al., 2015; Goodman et al., 1987; Guatelli-Steinberg
66 et al., 2004; Hillson, 2014; Lukacs et al., 2001; Eversole, 1984). Defects are often characterised
67 into four broad categories, pit-form (PEH), plane-form, linear-form (LEH), and localised
68 hypoplasia (Guatelli-Steinberg, 2015; Pindborg, 1970; Seow, 1990; Hillson & Bond, 1997;
69 Skinner et al., 2016). However, splitting defects into these categories can sometimes be
70 difficult (e.g., Odgen et al., 2007; Towle et al., 2018; Ioannou et al., 2016).

71 Pitting enamel hypoplasia (PEH) can be broadly defined as numerous circular to oval
72 defects that cover an extended area of a crown. Pits can be anything from small circular pin
73 like defects up to vast irregular depressions (Hillson & Bond, 1997; Skinner, 1996).
74 Additionally, some pits form rows around the circumference of a crown, or are associated
75 with plane-form defects, whilst others are much more randomly scattered (Goodman & Rose,
76 1990; Hillson & Bond, 1997; Lauc et al., 2015; Towle and Irish, 2019). Each pit is created due
77 to cessation/diminution of ameloblast activity, but it is not clear why only some ameloblasts
78 are affected along the plane of a brown stria of Retzius during formation. Small pits are
79 created when only a few ameloblasts stop forming enamel matrix, with large pits involving
80 hundreds (Guatelli-Steinberg, 2015). The enamel between pits often appears normal, and
81 exposed Tomes' process pits can frequently be observed within pits (Hillson, 2014; Hillson &
82 Bond, 1997). There is debate in the literature to whether PEH is caused by different factors
83 than LEH, or if it is just a consequence of the crown position and tooth involved (Hillson, 2014;
84 Hillson & Bond, 1997; Goodman & Rose, 1990; Lovell & Whyte, 1999).

85 Linear enamel hypoplasia (LEH) are bands of reduced enamel on a tooth's crown, and
86 are the most common type of enamel hypoplasia reported in the literature (e.g., Dobney &
87 Eryvnyck, 2000; Goodman & Armelagos, 1985; Guatelli-Steinberg, 2004; Guatelli-Steinberg &
88 Lukacs, 1999; Skinner et al., 2015). Anterior teeth tend to have a higher prevalence of LEH,
89 likely due to enamel property and morphology differences, although defects may also be
90 harder to detect macroscopically in posterior teeth (Goodman & Rose, 1990; Hillson & Bond,
91 1997; Guatelli-Steinberg, 2003; Bocaege et al., 2010; Hassett, 2012). LEH has been directly

92 associated with malnutrition and disease in clinical and animal studies, with a variety of other
93 disturbances during development also considered in archaeological and other studies, with
94 deeper/wider LEH defects usually linked to more severe events (Goodman & Rose 1990;
95 Guatelli-Steinberg & Benderlioglu, 2006; McGrath et al., 2018; Hillson, 2014). The age the
96 individual was when a LEH defect formed can be accurately found, through several different
97 techniques that calculate the developmental timing of grooves (e.g., Goodman & Armelagos,
98 1985; Reid & Dean, 2000; Cares Henriquez and Oxenham, 2019).

99 Localised hypoplasia is characterised by isolated irregular depressions that do not
100 extend around a crown, with usually only one or two continuous defects on the tooth (Skinner
101 et al., 2016; Suckling, 1980; Skinner and Skinner, 2017; Suckling et al., 1983; Suckling et al.,
102 1986; Skinner et al. 2014; Skinner & Newell, 2003). The aetiology of many types of localised
103 defects is related to direct trauma to the tooth during development, usually associated with
104 crypt fenestration (Suckling, 1980; Skinner and Skinner, 2017; Suckling et al., 1983; Suckling
105 et al., 1986). In particular, insufficient growth space in the maxilla and mandible has been
106 associated with localised defects termed crypt fenestration enamel defects (CFEDs) (Skinner
107 et al. 2014). Localised hypoplasia is common in certain groups but scarce in others, with
108 deciduous canines in certain primate species/populations commonly affected (Skinner et al.,
109 2016; Skinner & Newell, 2003; Halcrow and Tayles, 2008; Jančová et al., 2019). Although crypt
110 fenestration may be a common cause of localised defects, other processes can cause localised
111 defects, with specific genetic mutations linked to what would typically be recorded as
112 localised enamel hypoplasia (e.g., Hart et al., 2003).

113 Plane-form enamel hypoplasia occurs when enamel matrix formation ceases, either
114 completely, or in part. This creates an area of a crown with little or no enamel deposition
115 (Hillson & Bond, 1997; Krenz-Niedbała & Kozłowski, 2013; Ogden et al., 2007; Towle et al.,
116 2017). Hillson (2014) described these defects as extreme linear defects, with one perikymata
117 significantly widened. Similarly, plane-form hypoplasia is often reported in the literature as
118 part of other types of defects, in particular PEH or localised defects (Guatelli-Steinberg, 2003;
119 Littleton & Townsend, 2005; Skinner et al., 2016; Towle et al., 2018). These defects are often
120 found alongside other severe enamel defects, including those associated with conditions such
121 as congenital syphilis (e.g., Ioannou et al., 2016).

122 Significant research into enamel hypoplasia took place during the early to mid-20th
123 century. Research that utilised rat and mouse models were particularly common (e.g.,
124 Kreshover, 1960; Schour & Massler, 1945), as well as later studies on sheep and pig (e.g.,
125 Suckling and Cutress, 1977; Suckling et al., 1983; Witzel et al., 2006). These studies highlighted
126 that nutritional deficiencies can lead to hypoplastic defects. Enamel hypoplasia has also been
127 studied in a variety of human populations, with defect frequencies varying substantially in
128 both deciduous and permanent dentitions (Goodman & Rose, 1990; Hillson, 2014; Moggi-
129 Cecchi et al., 1994; Pisanty & Garfunkel, 1977; Purvis et al., 1973; Seow, 1990; Skinner &
130 Newell, 2003). Methods used to record enamel hypoplasia varies between studies.
131 Researchers often only record LEH frequencies (e.g., Guatelli-Steinberg, 2003, 2004;
132 Miszkiewicz, 2015). Whereas other studies record all hypoplastic defects (e.g., Goodman et
133 al., 1980, 1984; Goodman & Armelagos, 1985; Ogilvie et al., 1989). Similarly, some
134 researchers only record enamel hypoplasia on certain teeth, with anterior permanent teeth
135 usually favoured (e.g., Infante & Gillespie, 1974; Lovell & Whyte, 1999). When PEH is included
136 in a study it is often not clear if this includes defects found as part of LEH grooves (e.g.,
137 Goodman et al., 1980, 1984; Goodman & Armelagos, 1985; Hillson, 1992; Sognaes, 1956;
138 Mellanby, 1929).

139 Enamel defects come in a variety of shapes and sizes and each type can be caused by
140 different factors, making differential diagnosis in ancient samples difficult. In contrast, clinical
141 studies have linked enamel hypoplasia to a variety of specific conditions and disturbances
142 (Aine et al., 2000; Croft et al., 1965; Eliot et al., 1934; Gaul et al., 2015; Grahnen & Selander,
143 1954; Nikiforuk & Fraser, 1979, 1981; Pisanty et al., 1977; Purvis et al., 1973; Radu & Soficaru,
144 2016; Seow et al., 1984; Stimmler et al., 1973; Wright et al., 1993). Most studies of
145 archaeological and other ancient, or non-human, samples, since they do not have patient
146 records, can only conclude that an individual had a 'non-specific stress' if they display enamel
147 hypoplasia, or that a population was more/less stressed than other samples depending on
148 the prevalence of a particular type of hypoplastic defect. Typically, 'stress' in this context
149 refers to illness or malnutrition. In this study all types of macroscopically visible enamel
150 hypoplasia are recorded in three extant primate species and three fossil hominin species. We
151 hypothesize that dental characteristics (morphology, developmental timing and enamel
152 structure), and specific genetic/environmental factors heavily influence enamel hypoplasia

153 prevalence, with different defects typically having diverse aetiologies. If this is the case, there
 154 should be substantial variation in the types of defects that species' display, and different types
 155 of enamel hypoplasia should typically not be associated with one another.

156 **2. Materials and Methods**

157 The samples studied include specimens assigned to *Homo naledi*, *Paranthropus robustus*,
 158 *Australopithecus africanus*, gorillas, chimpanzees and baboons (Table 1). Specimen numbers
 159 and species classifications are detailed in the Appendix. Some data presented has been
 160 published in Towle and Irish (2019), with additional data added in the present study, notably
 161 localised hypoplasia prevalence's. The hominin samples are curated at The Ditsong National
 162 Museum of Natural History and the University of the Witwatersrand. The extant primate
 163 samples are curated at the Powell-Cotton Museum, and comprise common chimpanzees (*Pan*
 164 *troglydytes*), western lowland gorillas (*Gorilla gorilla gorilla*), and olive baboons (*Papio*
 165 *anubis*). They were killed in their natural habitats (Dean & Jones, 1992; Guatelli-Steinberg &
 166 Skinner, 2000; Lukacs, 2001).

167

168 **Table 1. Number of teeth for each sample, split by observable and not observable.**

169

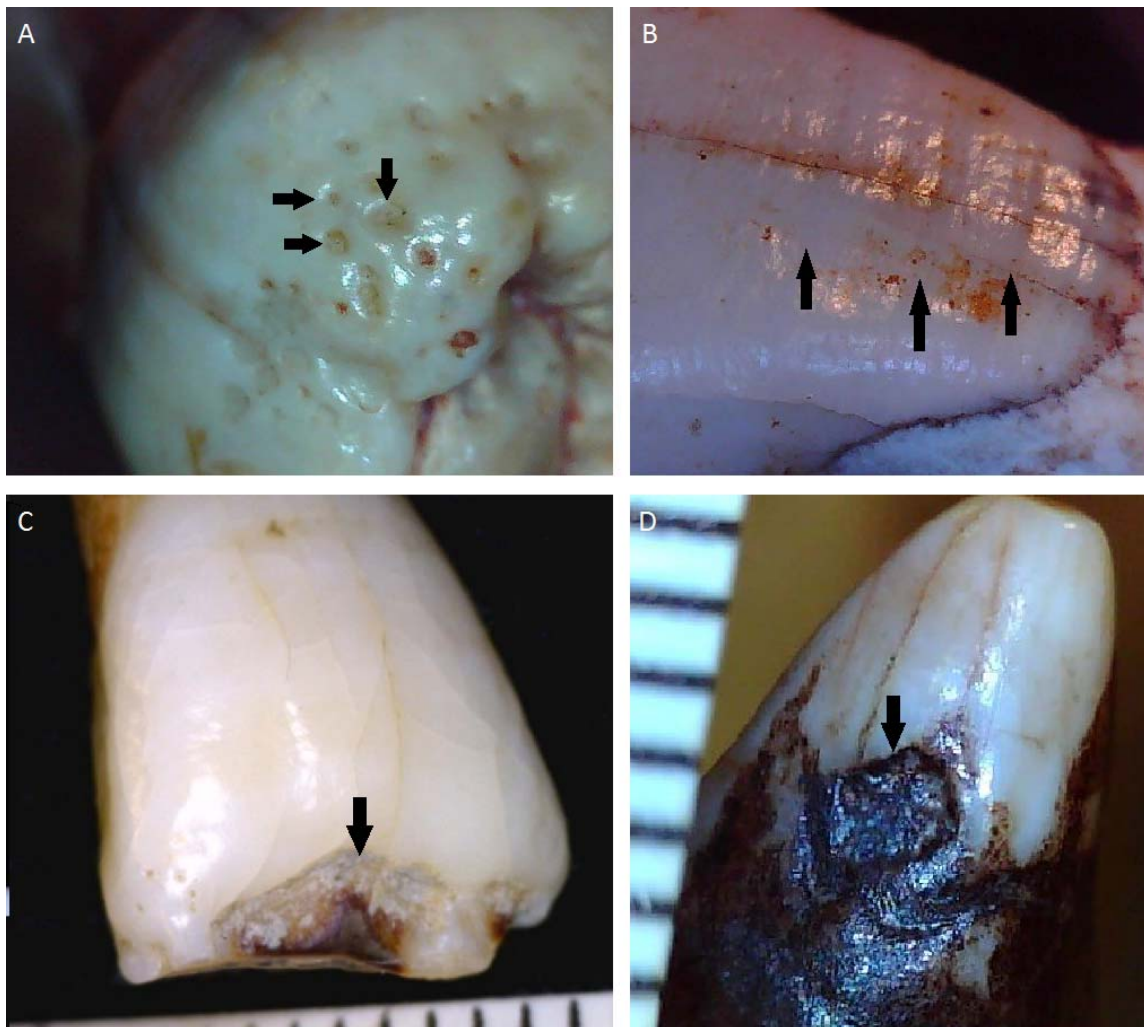
Species	Teeth observable	Not observable	Total teeth
Early <i>Homo</i>	47	19	66
<i>Australopithecus sediba</i>	10	1	11
<i>Paranthropus robustus</i>	304	127	431
<i>Homo naledi</i>	142	14	156
<i>Australopithecus africanus</i>	360	122	482
<i>Gorilla gorilla gorilla</i>	1693	392	2085
<i>Pan troglodytes</i>	1837	677	2514
<i>Papio anubis</i>	774	92	866

170

171 Teeth were held under a lamp and rotated allowing light to hit the surface at different
 172 angles. The smallest discernible macroscopic defect was recorded, with a hand lens only used
 173 to rule out postmortem damage. Methods for recording LEH follow Goodman & Rose (1990),
 174 Guatelli-Steinberg (2003), Lukacs (1989), and Miskiewicz (2015). Localised hypoplasia was
 175 recorded following Skinner et al. (2016). PEH was recorded if there was multiple circular/oval

176 enamel defects on a tooth crown. If pitting was present within a LEH band then it was
177 recorded as LEH not PEH, but the pitting was noted. Plane-form enamel hypoplasia was
178 recorded following Towle et al. (2017). If defects on a tooth didn't fit into one of these four
179 categories it was described and recorded separately, and not included in analysis.

180 To record defects each tooth was assigned a number. 0 was used to signify that there
181 were no visible defects. Teeth where it would not be possible to tell if a defect was present
182 due to post-mortem damage were marked as 8. Numbers 1, 2, 3 and 4 represent LEH,
183 localised, PEH and plane-form defects respectively. Examples of each type of defect are
184 displayed in Figure 1. Defects were photographed using a Dino-Lite® camera (Dino-Lite
185 AM2111 handheld microscope).



186
187

188 **Figure 1.** Enamel hypoplasia types. A) pitting enamel hypoplasia (*Australopithecus africanus*, SK 9);
189 B) linear enamel hypoplasia (*Homo naledi*, UW 101-38). C) plane-form enamel hypoplasia (*Homo*
190 *sapiens*, Towle et al., 2017); D) localised hypoplasia (*Gorilla gorilla gorilla*, M 667).

191

192 Due to how defects are displayed on tooth crowns, Hassett (2012) concluded that
193 enamel hypoplasia prevalence based solely on macroscopic observation could be misleading,
194 and create biases in comparing populations. To add to this debate, it has also been suggested
195 that microscopic techniques likely miss defects too, with micro-CT imaging showing enamel
196 abnormalities that do not show up in SEM or light microscopy (Marchewka et al., 2014; Xing
197 et al., 2015). However, there are advantages to macroscopic observation; it is quick,
198 inexpensive, non-destructive and allows large collections to be studied. It can therefore give
199 a good overview of health, disease and genetic conditions on a population level.

200 With increasing wear, all else being equal, fewer macroscopic enamel defects should
201 be visible on a crown. Instead of rejecting teeth worn past a certain point, all teeth with
202 remnant enamel, and not broken due to post-mortem damage, are included. This approach
203 will clearly lead to teeth being included that have had enamel defects worn away. However,
204 the alternative of excluding such teeth will also lead to bias, since an entire sample would
205 consist of individuals that died young. This methodology is also justified by the presence of
206 PEH and localized defects on severely worn teeth. There is variation in wear patterning
207 between the samples studied, however overall the average wear severity is similar between
208 species meaning wear is unlikely to have had a significant effect on overall enamel hypoplasia
209 differences (Towle and Irish, 2019; Towle, 2019). Data are presented by tooth count rather
210 than individual, with the number of hypoplastic teeth displayed as a percentage of the total
211 number of observable teeth. To compare certain groups a chi-square test of homogeneity
212 was used, with significance set at the 0.05 alpha level.

213

214 **3. Results**

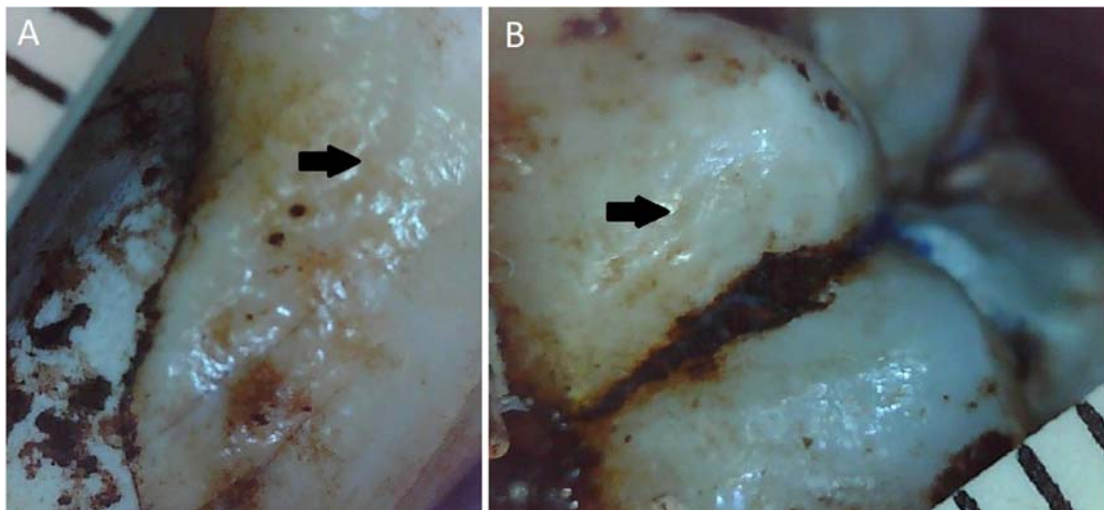
215 Table 2 displays the prevalence for the different types of enamel hypoplasia in
216 permanent and deciduous teeth of each species. The hominin samples have higher rates of
217 LEH than the extant great apes, with baboons having the lowest frequency. Localised
218 hypoplasia is not found on any of the hominin deciduous tooth samples. This is in contrast
219 with the extant primate sample in which it is common. PEH is rare in all deciduous samples
220 except *P. robustus*, in which over 40% of teeth have defects (Towle and Irish, 2019; Figure
221 2A). In specimens with pitting LEH, typically multiple rows of these defects are present on the

222 crown surface (Figure 2B). No plane-form defects were recorded in any of the hominin
 223 samples. A specific example of plane-form hypoplasia was found in the chimpanzee sample
 224 and has been published as a case study (Towle et al., 2018). Figures 3 and 4 highlight two
 225 examples of enamel defects that were difficult to categorise as one of the four enamel
 226 hypoplasia types.

227 **Table 2.** Per tooth prevalence (%) of linear enamel hypoplasia (LEH) pitting enamel
 228 hypoplasia (PEH) and localised hypoplasia for permanent and deciduous teeth.

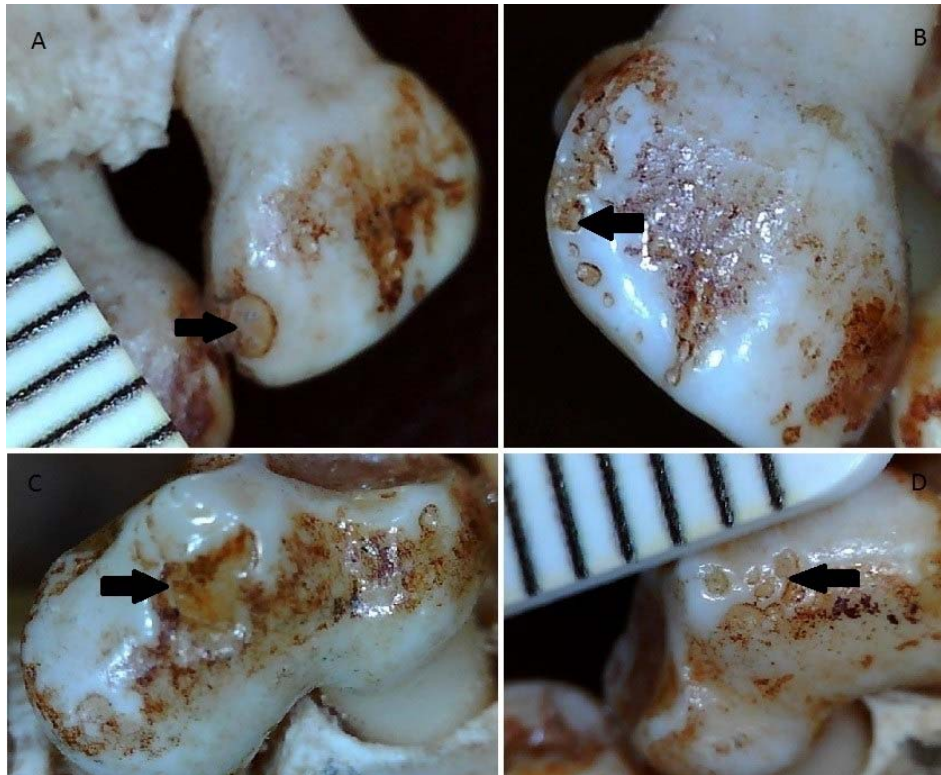
Species	Permanent teeth			Deciduous teeth	
	PEH (# teeth)	LEH (# teeth)	Localised	PEH (# teeth)	Localised
<i>Pan troglodytes</i>	0.65 (12/1837)	8.06 (148/1837)	0.98	4.23 (25/591)	5.08
<i>Gorilla gorilla gorilla</i>	2.89 (49/1693)	4.25 (72/1693)	0.95	1.39 (6/433)	12.93
<i>Papio anubis</i>	0.00 (0/774)	2.07 (16/774)	1.68	0.00 (0/107)	3.74
<i>Homo naledi</i>	0.70 (1/142)	14.79 (21/142)	0.70	0.00 (0/16)	0.00
<i>Australopithecus africanus</i>	5.03 (18/358)	15.08 (54/358)	0.28	5.00 (2/19)	0.00
<i>Paranthropus robustus</i>	14.75 (41/278)	11.51 (32/278)	1.08	41.30 (19/46)	0.00

229



230

231 **Figure 3.** Abnormal enamel in *H. naledi*. Black arrow highlights vertical 'wavy'
 232 grooves. A) Buccal surface of UW 501 (canine); B) Buccal surface of UW 377 and 1014
 233 (second molar).



234

235 **Figure 4.** Male chimpanzee displaying non-symmetric localised/pitting hypoplasia on multiple
 236 deciduous teeth (M 475). A) Upper left lateral incisor; B) Upper right lateral incisor; C) Lower left first
 237 molar; D) Lower right first molar. All buccal view. Black arrows indicate defects.

238

239 When individuals with and without localised hypoplasia are analysed separately, there
 240 is more PEH in the group with no localised enamel lesions for both gorillas and chimpanzees.
 241 For chimpanzees, in individuals with at least one localised defect, 1.2% of teeth have PEH,
 242 whereas for individuals with no localised defects 5.42% of their teeth have PEH. For gorillas,
 243 the figures are 0% and 5.77% respectively. For both species this is a statistically significant
 244 difference (gorillas: $X^2= 12.533$, 1 df, $p= 0.0004$; chimpanzees: $X^2= 4.416$, 1 df, $p= 0.0356$).

245 **4. Discussion**

246 People with amelogenesis imperfecta that display groove/linear enamel defects typically also
 247 show other enamel abnormalities, and all, or most, teeth are typically affected to some
 248 degree (Sundell and Koch, 1984, Crawford et al., 2007; Wright, 1985, Aldred et al., 2003,
 249 Chamarthi et al., 2012, Schuurs, 2012; Wright et al., 1993, Mehta et al., 2013). Additionally,
 250 animal and clinical studies have extensively shown that malnutrition and disease can cause
 251 LEH (Goodman & Rose 1990; Guatelli-Steinberg & Benderlioglu, 2006). Therefore, it is
 252 justifiable to use LEH as a basis for health during tooth development, although if accompanied

253 by other enamel abnormalities (e.g., PEH, reduced enamel thickness, hypomineralisation), a
254 genetic aetiology should also be considered. The results of the present study suggest it is
255 common for PEH to have a different aetiology than LEH. The PEH in *P. robustus* is likely genetic
256 in origin (Towle and Irish, 2019), and the clearest example of PEH in the chimpanzee sample
257 was also caused by amelogenesis imperfecta (Towle et al., 2018). Similarly, specific genetic
258 conditions and illnesses are associated with specific types of PEH in humans (Crawford et al.,
259 2007; Lauc et al., 2015). Lastly, bands of pits (pitting LEH), often show numerous bands on
260 different parts of a single tooth suggesting it may not simply be a consequence of crown
261 position that leads to these defects. These observations add support to the suggestion that
262 pitting defects in a sample may commonly have a different aetiology to LEH.

263 There is compelling evidence that many types of localised defects are caused by crypt
264 fenestration (Suckling, 1980; Skinner and Skinner, 2017; Suckling et al., 1983; Suckling et al.,
265 1986; Skinner et al. 2014; Skinner et al., 2016; Skinner, 1986; Skinner & Newell, 2003). It is
266 suggested the overarching reason may be linked to deficient growth in infancy of the
267 mandible and maxilla (Lukacs, 1999; Skinner et al., 2016). This theory is supported by studies
268 that highlight a link between general ill health and an increase in localised enamel hypoplasia
269 (Koch, 1999; Scheutzel & Ritter, 1989; Silberman et al., 1991; Skinner, 1986; Skinner & Hung,
270 1989). Studies on primates, rats and pigs, have also shown such a link, however these are not
271 based on wild populations and the animals involved were subject to severe starvation and
272 malnutrition (Dressino & Pucciarelli, 1997; Garat et al., 2006; McCance et al., 1961; Tonge &
273 McCance, 1973). Skinner et al. (2016) suggest there is a relationship between malnutrition
274 and dental overcrowding in humans, although the only significant relationship is in mouth-
275 breathing adolescents (Thomaz et al., 2010).

276 The results of the present study find individuals with localised defects on deciduous
277 canines do not show higher rates of other forms of hypoplasia. Therefore, certain
278 species/populations may be predisposed to certain types of localised enamel hypoplasia, in
279 certain teeth, due to cranial/dental morphology and therefore many of these defects may be
280 more linked to phylogeny than to the individual's health. Specific genetic and environmental
281 factors may also be important to consider (Skinner, 1996; Hart et al., 2003). In Figure 4, a
282 juvenile male chimpanzee with a full deciduous dentition has defects on the maxillary canines,
283 lateral incisors, and right first molar, as well as all mandibular teeth except the deciduous

284 second molars. If found in isolation, some teeth would likely be recorded as PEH or plane-
285 form defects, and the rest localised hypoplasia. The fact an antimere is not affected, and the
286 pattern of the defects are different on each tooth, suggests these defects may be best
287 described as localised enamel hypoplasia. This case highlights an issue in studying enamel
288 hypoplasia in fragmented collections, i.e., these defects all likely share a common aetiology,
289 but if individual teeth were found isolated several types of defect and/or aetiologies may have
290 been suggested. Even LEH on isolated teeth may be associated with other enamel
291 abnormalities. Therefore, in ancient samples it is crucial to record all teeth available, and all
292 types of enamel hypoplasia (and other enamel abnormalities if possible), to be able to help
293 rule out genetic and non-systemic factors.

294 Other unusual defects that don't fit into any of the four categories (e.g., 'wavey' or
295 'vertical' defects) are uncommon, although systematic recording of prevalence's in different
296 samples is rare. These macroscopic defects likely have a variety of aetiologies, but factors may
297 include tooth properties (e.g., underlying morphology or epithelium folding during
298 development), dentine defects, specific dietary/environmental factors (e.g., fluorosis), or
299 genetic conditions (Braunn et al., 2014; Xing et al., 2015; Musale et al., 2019). Therefore, these
300 abnormalities are not necessarily a form of enamel hypoplasia, making recording difficult in
301 ancient samples. Unusual enamel abnormalities such as these were rare in the sample's
302 studies, except in *H. naledi* in which 'wavey' and 'vertical' enamel abnormalities were
303 recorded (Figure 4). Tobias (1967) notes similar defects in a *P. boisei* specimen, but otherwise
304 such abnormalities are rarely recorded in fossil hominin samples. Other types of enamel
305 defects that are relatively common in people today and in some recent archaeological
306 samples (e.g., molar incisor hypomineralisation and plane form defects) are rare or absent in
307 earlier populations, such as in the present study, suggesting modern lifestyle (e.g., medicines,
308 environment, diet and disease) has had a significant impact on the types and prevalence of
309 enamel defects (Gualdi-Russo et al., 2017; Kühnisch et al., 2016; Ioannou et al., 2016; Ogden,
310 2007; Pramanik and Saha, 2017). Therefore, depending on the age of the sample, it may be
311 important to consider other types of enamel defects. When comparing these different types
312 of defects, histological, microscopic and micro-CT scan analysis, may offer a more complete
313 understanding of how an abnormality formed, and therefore potentially further insight into
314 timing and aetiology of specific abnormalities (Witzel et al., 2008; Hassett, 2014).

315 Genetic and environmental differences on a population level are also important to
316 consider. For example, populations that have recently undergone intensive selection in
317 relation to an enamel property (e.g., thickness or structure), may be predisposed to specific
318 types of enamel defects, due to loss of stability in specific genes or through pleiotropy effects
319 (Pavličev and Cheverud, 2015, Fiddes et al., 2018, Hlusko et al., 2018). For example, the ENAM
320 gene shows signs of strong positive selection in certain species, likely relating to enamel
321 thickness (Kelley and Swanson, 2008; Horvath et al., 2014), and mutations in this gene are
322 also associated with many types of amelogenesis imperfecta (Crawford et al., 2007, Kelley
323 and Swanson, 2008, Wang et al., 2015). Therefore, species that have recently evolved a
324 substantial increase/decrease in enamel thickness or tooth size, may be more prone to certain
325 types of enamel abnormalities (Towle et al., 2019). Other genetic factors will heavily influence
326 enamel abnormality prevalence on a population level, including founder effects, and the
327 complex, and not well understood, interaction between genotype and environmental and
328 epigenetic factors (Wang et al., 2016; Pramanik and Saha, 2017; Vieira et al., 2005; Russell,
329 1962; Musale et al., 2019).

330 Tooth properties (e.g., morphology, size and enamel structure), will also affect the
331 likelihood of enamel hypoplasia being visible on a macroscopic level, and influence the shape
332 and shape of defects (Guatelli-Steinberg et al., 2012; Braunn et al., 2014; McGrath et al.,
333 2018). For example, the angle at which striae of Retzius reach the outer enamel surface will
334 affect the depth and size of LEH defects, meaning different teeth, and surfaces, are more/less
335 likely to show macroscopic defects (Guatelli-Steinberg et al., 2012, 2017; Hillson & Bond,
336 1997; Kierdorf, Witzel, Kierdorf, Skinner, & Skinner, 2015; Hassett, 2014). Other enamel
337 properties also affect the expression of defects, including, perikymta spacing and the age of
338 ameloblasts (Hillson and Bond, 1997; Witzel et al., 2006; Witzel et al., 2008; Guatelli-Steinberg
339 et al., 2012; Hassett, 2012, 2014). It is well known that tooth development (e.g., speed and
340 total time) with also influence enamel hypoplasia prevalence's, with the results of the present
341 study supporting literature that finds higher rates of LEH in great apes than other primates
342 (e.g., Guatelli-Steinberg, 2001; Moggi-Cecchi & Crovella, 1991). This likely relates at least
343 partly to extended tooth formation, with great apes living longer through disease, nutritional
344 deficiencies and seasonal disturbances (Zihlman et al., 2007). In sum, there are a variety of
345 ways in which phylogeny influences enamel hypoplasia prevalence's, even before behaviour,

346 health and diet is considered. This is especially important considering hominin groups, and
347 primates more generally, differ substantially in terms of these dental properties.

348

349 **5. Conclusions**

350 The results of this study highlight how proportions of different kinds of enamel hypoplasia
351 varies substantially between samples. Tooth properties along with environmental and genetic
352 factors likely heavily influence frequencies. Therefore, studies that include only one form of
353 enamel hypoplasia to compare the 'stress' between populations may miss crucial
354 information. Instead, it may be more beneficial to display and described different types of
355 defects separately and attempt to understand the aetiology on an individual and population
356 bases. Incorporating tooth property and phylogeny information into analysis may also allow
357 more robust conclusions.

358

359 **Acknowledgements**

360 The authors thank L. Berger and B. Zipfel from the University of the Witwatersrand, I. Livne
361 from the Powell-Cotton Museum, and S. Potze from the Ditsong Museum of South Africa for
362 access to their collections. Data was collected thanks to a studentship from Liverpool John
363 Moores University.

364

365 **References**

366 Aine, L., Backström, M. C., Mäki, R., Kuusela, A. L., Koivisto, A. M., Ikonen, R. S., & Mäki, M.
367 (2000). Enamel defects in primary and permanent teeth of children born
368 prematurely. *Journal of Oral Pathology & Medicine*, 29(8), 403-409.

369 Braunn, P. R., Ribeiro, A. M., & Ferigolo, J. (2014). Microstructural defects and enamel
370 hypoplasia in teeth of *Toxodon Owen*, 1837 from the Pleistocene of Southern
371 Brazil. *Lethaia*, 47(3), 418-431.

372 Cares Henriquez, A., & Oxenham, M. F. (2019). New distance-based exponential regression
373 method and equations for estimating the chronology of linear enamel hypoplasia (LEH)
374 defects on the anterior dentition. *American Journal of Physical Anthropology*, 168(3), 510-
375 520.

376 Crawford, P. J., Aldred, M., & Bloch-Zupan, A. (2007). Amelogenesis imperfecta. *Orphanet*
377 *Journal of Rare Diseases*, 2(1), 17-29.

- 378 Croft, L. K., Witkop, C. J., & Glas, J. E. (1965). Pseudohypoparathyroidism. *Oral Surgery, Oral*
379 *Medicine, Oral Pathology, 20*(6), 758-770.
- 380 Dean, M. C., Jones, M. E., & Pilley, J. R. (1992). The natural history of tooth wear, continuous
381 eruption and periodontal disease in wild shot great apes. *Journal of Human Evolution, 22*(1),
382 23-39.
- 383 Dobney, K., & Ervynck, A. (2000). Interpreting developmental stress in archaeological pigs:
384 the chronology of linear enamel hypoplasia. *Journal of Archaeological Science, 27*(7), 597-
385 607.
- 386 Dressino, V., & Pucciarelli, H. M. (1997). Cranial growth in *Saimiri sciureus* (Cebidae) and its
387 alteration by nutritional factors: a longitudinal study. *American Journal of Physical*
388 *Anthropology, 102*(4), 545-554.
- 389 Eliot, M. M., Souther, S. P., Anderson, B. G., & Arnim, S. S. (1934). A study of the teeth of a
390 group of school children previously examined for rickets. *American Journal of Diseases of*
391 *Children, 48*(4), 713-729.
- 392 Eversole, L. R. (1984). *Clinical outline of oral pathology: diagnosis and treatment*.
393 Philadelphia: Lea & Febiger publishers.
- 394 Garat, J. A., Martín, A. E., Pani, M., Holgado, N. R., Meheris, H. E., & González, S. (2006).
395 Orthodontic implications of protein undernutrition in mandibular growth. A cephalometric
396 study in growing rats. *Acta Odontologica Latinoamericana, 20*(2), 73-78.
- 397 Gaul, J. S., Grossschmidt, K., Gusenbauer, C., & Kanz, F. (2015). A probable case of
398 congenital syphilis from pre-Columbian Austria. *Anthropologischer Anzeiger, 72*(4), 451-472.
- 399 Goodman, A. H., & Armelagos, G. J. (1985). The chronological distribution of enamel
400 hypoplasia in human permanent incisor and canine teeth. *Archives of Oral Biology, 30*(6),
401 503-507.
- 402 Goodman, A. H., & Rose, J. C. (1990). Assessment of systemic physiological perturbations
403 from dental enamel hypoplasias and associated histological structures. *American Journal of*
404 *Physical Anthropology, 33*(S11), 59-110.
- 405 Goodman, A. H., & Rose, J. C. (1991). Dental enamel hypoplasias as indicators of nutritional
406 status. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*. New York:
407 Wiley-Liss, 279-293.
- 408 Goodman, A. H., Allen, L. H., Hernandez, G. P., Amador, A., Arriola, L. V., Chavez, A., & Pelto,
409 G. H. (1987). Prevalence and age at development of enamel hypoplasias in Mexican
410 children. *American Journal of Physical Anthropology, 72*(1), 7-19.
- 411 Goodman, A. H., Armelagos, G. J., & Rose, J. C. (1980). Enamel hypoplasias as indicators of
412 stress in three prehistoric populations from Illinois. *Human Biology, 51*5-528.
- 413 Goodman, A. H., Armelagos, G. J., & Rose, J. C. (1984). The chronological distribution of
414 enamel hypoplasias from prehistoric Dickson Mounds populations. *American Journal of*
415 *Physical Anthropology, 65*(3), 259-266.

- 416 Grahnen, H., & Selander, P. (1954). The effect of rickets and spasmophilia on the permanent
417 dentition. I. The effect on the teeth. *Odontologisk Revy*, 5(1), 7.
- 418 Guatelli-Steinberg, D. (2001). What can developmental defects of enamel reveal about
419 physiological stress in nonhuman primates? *Evolutionary Anthropology: Issues, News, and*
420 *Reviews*, 10(4), 138-151.
- 421 Guatelli-Steinberg, D. (2003). Macroscopic and microscopic analyses of linear enamel
422 hypoplasia in Plio-Pleistocene South African hominins with respect to aspects of enamel
423 development and morphology. *American Journal of Physical Anthropology*, 120(4), 309-322.
- 424 Guatelli-Steinberg, D. (2015). Micro-to Macroscopic. In: Irish, J. D., & Scott, G. R. (Eds.), *A*
425 *companion to dental anthropology*, 450. Hoboken: Wiley-Blackwell.
- 426 Guatelli-Steinberg, D., & Lukacs, J. R. (1999). Interpreting sex differences in enamel
427 hypoplasia in human and non-human primates: Developmental, environmental, and cultural
428 considerations. *American Journal of Physical Anthropology*, 110(S29), 73-126.
- 429 Guatelli-Steinberg, D., & Skinner, M. (2000). Prevalence and etiology of linear enamel
430 hypoplasia in monkeys and apes from Asia and Africa. *Folia Primatologica*, 71(3), 115-132.
- 431 Guatelli-Steinberg, D., Larsen, C. S., & Hutchinson, D. L. (2004). Prevalence and the duration
432 of linear enamel hypoplasia: a comparative study of Neandertals and Inuit foragers. *Journal*
433 *of Human Evolution*, 47(1), 65-84.
- 434 Hassett, B. R. (2012). Evaluating sources of variation in the identification of linear
435 hypoplastic defects of enamel: a new quantified method. *Journal of Archaeological*
436 *Science*, 39(2), 560-565.
- 437 Hillson, S. (2014). *Tooth development in human evolution and bioarchaeology*. Cambridge:
438 Cambridge University Press.
- 439 Hillson, S. W. (1992). Dental enamel growth, perikymata and hypoplasia in ancient tooth
440 crowns. *Journal of the Royal Society of Medicine*, 85(8), 460-466.
- 441 Hillson, S., & Bond, S. (1997). Relationship of enamel hypoplasia to the pattern of tooth
442 crown growth: a discussion. *American Journal of Physical Anthropology*, 104(1), 89-103.
- 443 Infante, P. F., & Gillespie, G. M. (1974). An epidemiologic study of linear enamel hypoplasia
444 of deciduous anterior teeth in Guatemalan children. *Archives of Oral Biology*, 19(11), 1055-
445 1061.
- 446 Koch, M. J., Bühner, R., Pioch, T., & Schärer, K. (1999). Enamel hypoplasia of primary teeth in
447 chronic renal failure. *Pediatric Nephrology*, 13(1), 68-72.
- 448 Krenz-Niedbała, M., & Kozłowski, T. (2013). Comparing the chronological distribution of
449 enamel hypoplasia in Rogowo, Poland (2nd century AD) using two methods of defect timing
450 estimation. *International Journal of Osteoarchaeology*, 23(4), 410-420.
- 451 Kreshover, S. J. (1960). Metabolic disturbances in tooth formation. *Annals of the New York*
452 *Academy of Sciences*, 85(1), 161-167.

- 453 Lauc, T., Fornai, C., Premužić, Z., Vodanović, M., Weber, G. W., Mašić, B., & Šikanjić, P. R.
 454 (2015). Dental stigmata and enamel thickness in a probable case of congenital syphilis from
 455 XVI century Croatia. *Archives of Oral Biology*, 60(10), 1554-1564.
- 456 Lauc, T., Fornai, C., Premužić, Z., Vodanović, M., Weber, G. W., Mašić, B., & Šikanjić, P. R.
 457 (2015). Dental stigmata and enamel thickness in a probable case of congenital syphilis from
 458 XVI century Croatia. *Archives of Oral Biology*, 60(10), 1554-1564.
- 459 Littleton, J., & Townsend, G. (2005). Linear enamel hypoplasia and historical change in a
 460 central Australian community. *Australian Dental Journal*, 50(2):101-107.
- 461 Lovell, N. C., & Whyte, I. (1999). Patterns of dental enamel defects at ancient Mendes,
 462 Egypt. *American Journal of Physical Anthropology*, 110(1), 69-80.
- 463 Lukacs, J. R. (1989). Dental paleopathology: methods for reconstructing dietary
 464 patterns. *Reconstruction of Life from the Skeleton*, 1, 261-286.
- 465 Lukacs, J. R. (1999). Enamel hypoplasia in deciduous teeth of great apes: Do differences in
 466 defect prevalence imply differential levels of physiological stress? *American Journal of*
 467 *Physical Anthropology*, 110(3), 351-363.
- 468 Lukacs, J. R. (2001). Enamel hypoplasia in the deciduous teeth of great apes: variation in
 469 prevalence and timing of defects. *American Journal of Physical Anthropology*, 116(3), 199-
 470 208.
- 471 Marchewka, J., Skrzat, J., & Wróbel, A. (2014). Analysis of the enamel hypoplasia using
 472 micro-CT scanner versus classical method. *Anthropologischer Anzeiger*, 71(4), 391-402.
- 473 McCance, R. A., Ford, E. H. R., & Brown, W. A. B. (1961). Severe undernutrition in growing
 474 and adult animals. *British Journal of Nutrition*, 15(02), 213-224.
- 475 Mellanby, M. (1929). *Diet and the teeth: an experimental study. Part I. Dental structure in*
 476 *dogs*. London: Medical Research Council (H.M.S.O.).
- 477 Miszkiewicz, J. J. (2015). Linear Enamel Hypoplasia and Age-at-Death at Medieval (11th–
 478 16th Centuries) St. Gregory's Priory and Cemetery, Canterbury, UK. *International Journal of*
 479 *Osteoarchaeology*, 25(1), 79-87.
- 480 Moggi-Cecchi, J., & Crovella, S. (1991). Occurrence of enamel hypoplasia in the dentitions of
 481 simian primates. *Folia Primatologica*, 57(2), 106-110.
- 482 Moggi-Cecchi, J., Pacciani, E., & Pinto-Cisternas, J. (1994). Enamel hypoplasia and age at
 483 weaning in 19th-century Florence, Italy. *American Journal of Physical Anthropology*, 93(3),
 484 299-306.
- 485 Nikiforuk, G., & Fraser, D. (1979). Etiology of enamel hypoplasia and interglobular dentin:
 486 the roles of hypocalcemia and hypophosphatemia. *Metabolic Bone Disease and Related*
 487 *Research*, 2(1), 17-23.
- 488 Nikiforuk, G., & Fraser, D. (1981). The etiology of enamel hypoplasia: a unifying concept. *The*
 489 *Journal of Pediatrics*, 98(6), 888-893.

- 490 Ogden, A. R., Pinhasi, R., & White, W. J. (2007). Gross enamel hypoplasia in molars from
491 subadults in a 16th–18th century London graveyard. *American Journal of Physical*
492 *Anthropology*, 133(3), 957-966.
- 493 Ogilvie, M. D., Curran, B. K., & Trinkaus, E. (1989). Incidence and patterning of dental
494 enamel hypoplasia among the Neandertals. *American Journal of Physical*
495 *Anthropology*, 79(1), 25-41.
- 496 Pindborg, J. J. (1970). *Pathology of the dental hard tissues*. Philadelphia: W. B. Saunders
497 Company.
- 498 Pisanty, S., & Garfunkel, A. (1977). Familial hypoparathyroidism with candidiasis and mental
499 retardation. *Oral Surgery, Oral Medicine, Oral Pathology*, 44(3), 374-383.
- 500 Purvis, R. J., MacKay, G. S., Cockburn, F., Barrie, W. M., Wilkinson, E. M., Belton, N. R., &
501 Forfar, J. O. (1973). Enamel hypoplasia of the teeth associated with neonatal tetany: a
502 manifestation of maternal vitamin-D deficiency. *The Lancet*, 302(7833), 811-814.
- 503 Radu, C., & Soficaru, A. D. (2016). Dental developmental defects in a subadult from 16th–
504 19th centuries Bucharest, Romania. *International Journal of Paleopathology*, 15, 33-38.
- 505 Reid, D. J., & Dean, M. C. (2000). Brief communication: the timing of linear hypoplasias on
506 human anterior teeth. *American Journal of Physical Anthropology*, 113(1), 135-139.
- 507 Scheutzel, P., & Ritter, W. (1989). Alterations of teeth and jaws in children with chronic
508 renal failure. *Deutsche Zahnärztliche Zeitschrift*, 44(2), 115-118.
- 509 Schour, I., & Massler, M. (1945). The Effects of Dietary Deficiencies Upon the Oral
510 Structures. III. *The Journal of the American Dental Association*, 32(15), 1022-1030.
- 511 Seow, W. K. (1990). Enamel hypoplasia in the primary dentition: a review. *Journal of*
512 *Dentistry for Children*, 58(6), 441-452.
- 513 Seow, W. K., Brown, J. P., Tudehope, D. A., & O'Callaghan, M. (1984). Dental defects in the
514 deciduous dentition of premature infants with low birth weight and neonatal
515 rickets. *Pediatric Dentistry*, 6(2), 88-92.
- 516 Silberman, S. L., Trubman, A., Duncan, W. K., & Meydrech, E. F. (1991). Prevalence of
517 primary canine hypoplasia of the mandibular teeth. *Pediatric Dentistry*, 13, 356-60.
- 518 Skinner, M. (1996). Developmental stress in immature hominines from Late Pleistocene
519 Eurasia: evidence from enamel hypoplasia. *Journal of Archaeological Science*, 23(6), 833-
520 852.
- 521 Skinner, M. F. (1986). An enigmatic hypoplastic defect of the deciduous canine. *American*
522 *Journal of Physical Anthropology*, 69(1), 59-69.
- 523 Skinner, M. F., & Hung, J. T. W. (1989). Social and biological correlates of localized enamel
524 hypoplasia of the human deciduous canine tooth. *American Journal of Physical*
525 *Anthropology*, 79(2), 159-175.

- 526 Skinner, M. F., Rodrigues, A. T., & Byra, C. (2014). Developing a pig model for crypt
527 fenestration-induced localized hypoplastic enamel defects in humans. *American Journal of*
528 *Physical Anthropology*, 154(2), 239-250.
- 529 Skinner, M. F., & Skinner, M. M. (2017). Orangutans, enamel defects, and developmental
530 health: A comparison of Borneo and Sumatra. *American Journal of Primatology*, 79(8),
531 e22668.
- 532 Skinner, M. F., & Newell, E. A. (2003). Localized hypoplasia of the primary canine in
533 bonobos, orangutans, and gibbons. *American Journal of Physical Anthropology*, 120(1), 61-
534 72.
- 535 Skinner, M. F., Dupras, T. L., & Moya-Sola, S. (2015). Periodicity of linear enamel hypoplasia
536 among Miocene *Dryopithecus* from Spain. *Journal of Paleopathology*, 7(3), 195-222.
- 537 Skinner, M. F., Skinner, M. M., Pilbrow, V. C., & Hannibal, D. L. (2016). An enigmatic
538 hypoplastic defect of the maxillary lateral incisor in recent and fossil orangutans from
539 Sumatra (*Pongo abelii*) and Borneo (*Pongo pygmaeus*). *International Journal of*
540 *Primatology*, 37(4-5), 548-567.
- 541 Smith, T. M., Tafforeau, P., Le Cabec, A., Bonnin, A., Houssaye, A., Pouech, J., ... & Menter, C.
542 G. (2015). Dental ontogeny in Pliocene and early Pleistocene hominins. *PLoS one*, 10(2),
543 e0118118.
- 544 Sognaes, R. F. (1956). Histologic evidence of developmental lesions in teeth originating
545 from Paleolithic, prehistoric, and ancient man. *The American Journal of Pathology*, 32(3),
546 547-577.
- 547 Stimmler, L., Snodgrass, G. J. A. I., & Jaffe, E. (1973). Dental defects associated with neonatal
548 symptomatic hypocalcaemia. *Archives of Disease in Childhood*, 48(3), 217-220.
- 549 Suckling, G. (1980). Defects of enamel in sheep resulting from trauma during tooth
550 development. *Journal of Dental Research*, 59(9), 1541-1548.
- 551 Suckling, G., Elliott, D. C., & Thurley, D. C. (1983). The production of developmental defects
552 of enamel in the incisor teeth of penned sheep resulting from induced parasitism. *Archives*
553 *of Oral Biology*, 28(5), 393-399.
- 554 Suckling, G., Elliott, D. C., & Thurley, D. C. (1986). The macroscopic appearance and
555 associated histological changes in the enamel organ of hypoplastic lesions of sheep incisor
556 teeth resulting from induced parasitism. *Archives of Oral Biology*, 31(7), 427-439.
- 557 Ten Cate, A. R., & Nanci, A. (1994). Structure of the oral tissues. In: Ten Cate, A. R. (Ed.), *Oral*
558 *Histology: Development, Structure, Function*, 45-57. St. Louis: Mosby Year Book.
- 559 Thomaz, E. B., Cangussu, M. C. T., Silva, A. A. M. D., & Assis, A. M. O. (2010). Is malnutrition
560 associated with crowding in permanent dentition? *International Journal of Environmental*
561 *Research and Public Health*, 7(9), 3531-3544.
- 562 Tobias, P. V. (1967). *Olduvai Gorge (Vol. 2)*. Cambridge: Cambridge University Press.

563 Tonge, C. H., & McCance, R. A. (1973). Normal development of the jaws and teeth in pigs,
564 and the delay and malocclusion produced by calorie deficiencies. *Journal of*
565 *Anatomy*, 115(1), 1-22.

566 Towle, I. E. (2017). Dental pathology, wear and developmental defects in South African
567 hominins (Doctoral dissertation, Liverpool John Moores University).

568 Towle, I., & Irish, J. D. (2018). A probable genetic origin for pit defects on the molars of
569 *Paranthropus robustus*. bioRxiv, 400671.

570 Towle, I., Dove, E. R., Irish, J. D., & De Groote, I. (2017). Severe plane-form enamel
571 hypoplasia in a dentition from Roman Britain. *Dental Anthropology*, 30, 16-24.

572 Towle, I., Irish, J. D., & De Groote, I. (2018). Amelogenesis imperfecta in the dentition of a
573 wild chimpanzee. *Journal of medical primatology*, 47(2), 117-119.

574 Wright, J. T., Fine, J. D., & Johnson, L. (1993). Hereditary epidermolysis bullosa: oral
575 manifestations and dental management. *Pediatric Dentistry*, 15, 242-242.

576 Xing, S., Guatelli-Steinberg, D., O'Hara, M., Li, J., Wei, P., Liu, W., & Wu, X. (2015). Micro-CT
577 imaging and analysis of enamel defects on the early late pleistocene xujiayao
578 juvenile. *International Journal of Osteoarchaeology*, 26(6), 935-946.

579 Zihlman, A. L., Bolter, D. R., & Boesch, C. (2007). Skeletal and dental growth and
580 development in chimpanzees of the Tai National Park, Côte D'Ivoire. *Journal of*
581 *Zoology*, 273(1), 63-73.

582 Jančová, M., Štelcl, J., Klíma, B. F., & Drozdová, E. (2019). Localised enamel hypoplasia of
583 human primary canines (LHPC) in the Necropolis of Great Moravia in Znojmo-Hradiště (the
584 so called Stronghold of Znojmo, 9th-10th century CE, Czech Republic) and analysis of
585 chemical elements on surface enamel and hypoplastic defect via EDX method.
586 *Anthropologischer Anzeiger; Bericht über die biologisch-anthropologische Literatur*.

587 Halcrow, S. E., & Tayles, N. (2008). Stress near the start of life? Localised enamel hypoplasia
588 of the primary canine in late prehistoric mainland Southeast Asia. *Journal of Archaeological*
589 *Science*, 35(8), 2215-2222.

590 Pramanik, S., & Saha, D. (2017). The genetic influence in fluorosis. *Environmental Toxicology*
591 *and Pharmacology*, 56, 157-162.

592 Russell, A. L. (1962). Dental fluorosis in Grand Rapids during the seventeenth year of
593 fluoridation. *The Journal of the American Dental Association*, 65(5), 608-612.

594 Musale, P. K., Soni, A. S., & Kothare, S. S. (2019). Etiology and Considerations of
595 Developmental Enamel Defects in Children: A Narrative Review.

596 Wang, J., Sun, K., Shen, Y., Xu, Y., Xie, J., Huang, R., ... & Lin, Y. (2016). DNA methylation is
597 critical for tooth agenesis: implications for sporadic non-syndromic anodontia and
598 hypodontia. *Scientific Reports*, 6, 19162.

599

600

601 Gualdi-Russo, E., Zedda, N., Esposito, V., & Masotti, S. (2017). More on molar incisor
602 hypomineralisation (MIH) and linear enamel hypoplasia (LEH) in archaeological human
603 remains. *Clinical Oral Investigations*, 21(7), 2153-2154.

604

605 Kühnisch, J., Lauenstein, A., Pitchika, V., McGlynn, G., Staskiewicz, A., Hickel, R., & Grupe, G.
606 (2016). Was molar incisor hypomineralisation (MIH) present in archaeological case series?.
607 *Clinical Oral Investigations*, 20(9), 2387-2393.

608

609 Ioannou, S., Sassani, S., Henneberg, M., & Henneberg, R. J. (2016). Diagnosing congenital
610 syphilis using H utchinson's method: Differentiating between syphilitic, mercurial, and
611 syphilitic-mercurial dental defects. *American Journal of Physical Anthropology*, 159(4), 617-
612 629.

613