

## RESEARCH ARTICLE

# Linear enamel hypoplasia in *Homo naledi* reappraised in light of new Retzius periodicities

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## Funding information

Connor Family Faculty Foundation; Wenner-Gren Foundation; Royal Society, Grant/Award Number: RG110435; The Leverhulme Trust, Grant/Award Number: RPG-2018-226; Evolutionary Studies Institute at the University of Witwatersrand; European Union's Horizon 2020 Research and Innovation Programme, Grant/Award Number: 819960; Office of Research and Development at the University of Arkansas

## Abstract

**Objectives:** Among low-latitude apes, developmental defects of enamel often recur twice yearly, linkable to environmental cycles. Surprisingly, teeth of *Homo naledi* from Rising Star in South Africa (241–335 kya), a higher latitude site with today a single rainy season, also exhibit bimodally distributed hypoplastic enamel defects, but with uncertain timing and etiology. Newly determined Retzius periodicities for enamel formation in this taxon enable a reconstruction of the temporal patterning of childhood stress.

**Methods:** Using high resolution casts of 31 isolated anterior teeth from *H. naledi*, 82 enamel defects (linear enamel hypoplasia [LEH]) were identified. Seventeen teeth are assigned to three individuals. Perikymata in the occlusal wall of enamel furrows and between the onsets of successive LEH were visualized with scanning electron microscopy and counted. Defects were measured with an optical scanner. Conversion of perikymata counts to estimates of LEH duration and inter-LEH interval draws upon Retzius periodicities of 9 and 11 days.

**Results:** Anterior teeth record more than a year of developmental distress, expressed as two asymmetric intervals centered on 4.5 and 7.5 months bounded by three LEH. Durations, also, show bimodal distributions, lasting 3 or 12 weeks. Short duration LEH are more severe than long duration. Relative incisor/canine rates of formation are indistinguishable from modern humans.

**Discussion:** We invoke a disease and dearth model, with short episodes of distress reflecting onset of disease in young infants, lasting about 3 weeks, followed by a season of undernutrition, possibly intensified by secondary plant compounds, spanning about 12 weeks, inferably coincident with austral winter.

## KEYWORDS

developmental stress, Middle Pleistocene, ontogeny, Rising Star, seasonality

## 1 | INTRODUCTION

Recent human ontogeny is remarkable among mammals for delayed maturation and prolonged childhood (Bogin, 1998). Characterizing

growth in fossil hominins, in the absence of known ages, is challenging (Bolter & Cameron, 2020). A first step towards capturing growth rates is to identify time markers in fossil hard tissues. Paleoanthropological growth studies draw fundamentally on time markers preserved in

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enamel (Bailey & Hublin, 2007; Bromage, 1987; Dean, 1987; Guatelli-Steinberg, 2004; Hillson, 2014; Lacruz et al., 2008; Smith et al., 2015). The outer lateral enamel surface of hominoid teeth preserves two time markers: perikymata (PK), which are the surface expression of Retzius layers deposited with a precise but variable endogenous rhythm measured in days (FitzGerald, 1998; McFarlane et al., 2021); and linear enamel hypoplasia (LEH), an adventitious exogenous record of developmental stress measured in seasons and years, usually attributed to disease or under-nutrition (Bacon et al., 2020; Brunet et al., 2002; Chollet & Teaford, 2010; Franz-Odenaal et al., 2004; Goodman & Armelagos, 1985; Guatelli-Steinberg & Skinner, 2000; Skinner, 1986; Skinner & Skinner, 2017). Since these enamel features may be observed without invasive imagery, together they can disclose epidemiological patterns of the timing and impact of environmental stressors that would not be evident from studying just one or a few individuals.

Disease and undernutrition often show seasonality but may differ in seasonal drivers; that is, biotic and abiotic forces (Elliot et al., 2002; Kumar & Kumar, 2018), which can act singly or synchronously. Biotic factors such as acute disease come on suddenly and usually last, at most, a few weeks (de Benedictis & Bush, 2018; Long, 2005; O'Grady et al., 2017) while abiotic factors, including cycles of temperature and rainfall, typically last much longer since they are controlled by orbital mechanics (Kingston, 2005). Distinguishing between biotic and abiotic forces from their effect on enamel formation may not always be possible. Nevertheless, a host species will conceivably evolve adaptive responses (e.g., phenotypic and behavioral) to recurrent abiotic forces, such as seasonal variation in moisture and temperature acting over geological time, likely lessening their impact. Biotic agents, such as bacteria and viruses, can evolve faster than their hosts, creating an ongoing competition between immune response and evolving pathogens (Bonneaud et al., 2011) and increasing their impact on enamel-secreting ameloblasts. Thus, we suggest that disease is more likely to invoke an acute response of short duration while seasonal undernutrition or cold distress (Skinner, 2021) is more likely to create wider and shallower LEH. We define a cycle as an interval over which a phenomenon recurs. Most cycles in nature (including seasons) are nonperiodic, that is, somewhat variable in timing and amplitude (Briens & Briens, 2002). Therefore, we do not look upon repetitive LEH (rLEH) as metronomic but simply showing average timings consistent with being seasonally cued.

A previous study of LEH created during crown formation in *Homo naledi* reconstructed the recurrence of LEH based on Retzius periodicities (RP) ranging from 6 to 10 days per perikyma concluding that, on average, modes of developmental stress occurred at 1.6–2.8 months (lasting about 2 weeks) and 4.3–7.6 months (lasting about 11 weeks) (Skinner, 2019); that is, developmental stress was episodic, not chronic. The pattern of discrete bimodal durations and intervals was tentatively ascribed to two annual independent stressors such as disease and undernutrition. An unresolved issue in the earlier study was that the reconstructed recurrences (ca. 2 and 6 months) did not constitute an annual cycle of 12 months, allowing the inference that a

semiannual stressor, like that ascribed to some low-latitude apes (Skinner, 2021), might still prevail.

The advent of newly-determined RP for *H. naledi* (Mahoney et al., 2024) enables a reappraisal of the timing and duration of developmental defects of enamel in this taxon and provides a glimpse into the timing of early childhood experiences. Retzius periodicity has now been observed among three teeth from *H. naledi*: two canines with a RP of 11 days and a molar with a RP of 9 days (Mahoney et al., 2024). The canines are not antimeres. Only the specific tooth identified as U.W. 101-886 (see Delezene et al., 2023) from their study appears in our research. Regrettably, at the time of our examination and taking of molds, the labial surface of this tooth was partially obscured by glue, precluding direct counting of perikymata. These RP values are relatively high for *Homo* (see discussion in [Mahoney et al., 2024]), resulting in significantly increased estimates of duration and recurrence of developmental stress in this taxon, necessitating reconsideration of potential etiologies. In addition, a new catalogue of dental remains ('canonical tooth identifications') from the Rising Star assemblage has recently appeared (Delezene et al., 2023), which cautiously attributes some of the teeth to individuals designated as "associations." This latter study has enabled us to correct many side and jaw attributions for the teeth (11 out of 31 teeth) as well as to classify those canonically identified teeth with clear glue on their labial surfaces. Adherent glue was judged not to affect LEH counts but could affect measurements of LEH width and depth prompting their removal (9 out of 78 measurements). In short, the current study aims to reconstruct the duration and recurrence of developmental stress among *H. naledi* based on an improved sample and actual RP. We do not seek to find the prevalence of LEH in *H. naledi*, we focus solely on the problem of the timing of LEH which in our opinion, affords the best clue as to etiology. Overall prevalence of LEH among *H. naledi* (21/142 permanent teeth affected) has been reported (Towle & Irish, 2020). A report and illustration of only a single instance of pit-type enamel hypoplasia among 142 observable permanent teeth, including cheek teeth, from *H. naledi* has been provided by Towle and Irish (2019, 2020).

## 1.1 | PREDICTIONS

While yearly cycles of disease and undernutrition could both be dependent on a synchronous environmental cue, here we examine the possibility that cycles of stressful conditions experienced by *H. naledi* are independent, thereby creating subannual and/or asynchronous intervals of uneven length between successive LEH furrows, which themselves show contrasting salience.

### 1.1.1 | Null hypotheses

H<sub>01</sub>-Timing of successive LEH is randomly distributed.

**TABLE 1** *Homo naledi* anterior teeth selected for casting and assessment of linear enamel hypoplasia.

Canonical specimen number U.W. 101-	Canonical ID (2023) <sup>a</sup>	LEH per tooth type (assessed in isolation from other teeth)		Associations <sup>a</sup>
		Incisor	Canine	
0038	URI1	2		
0039	LoRI1	3		
0073	URI2	3		Possible antimere of 1588
0335	LoRI2	3 (glue obscured)		
0337	URC		1	
0339	LoRC		5	
1014-0377	LoRC		2	2
0412	ULC		1	Possible antimere of 0908
0501	ULC		2	
0591	ULI1	1		
0706	ULC		2	3, antimere of 0816
0709	URI2	2		3
0816	URC		2	3
0886	LoRC		2 (glue obscured)	4, antimere of 1126
0908	URC		3	Possible antimere of 0412
0931	ULI1	5		3, antimere of 1012
0932	ULI2	3		3, antimere of 0709
0952	ULI2	3		
0985	LoLC		4	
0998	LoLI2	4		2, antimere of 1005C
1005B	LoRI1	4		2
1005C	LoRI2	4		2
1012	URI1	3		3
1075	LoRI2	3		4, antimere of 1131
1076	LoLC		4	2, antimere of 0377
1126	LoLC		2 (glue obscured)	4
1131	LoLI2	2		4
1132	LoLI1	3 (glue obscured)		4, antimere of 1133
1133	LoRI1	1 (glue obscured)		4
1556	ULC		1	6
1588	ULI2	2		Possible antimere of 0073
Mean+/-SD		2.8 ± 1.0	2.4 ± 1.3	
N teeth		18	13	
N LEH		51	31	

Abbreviations: L, left; LEH, linear enamel hypoplasia; Lo, lower; U, upper; R, right.

<sup>a</sup>(Deleze et al., 2023).

H<sub>02</sub>-If not random, perikymata counts for both durations of LEH and interval between successive LEH are only unimodally distributed.

H<sub>03</sub>-LEH do not differ in intensity or severity (as measured by onset angle and daily deficit of enamel, respectively) among teeth, individuals or other groupings.

### 1.1.2 | Alternative hypotheses

H<sub>1</sub>-LEH show bimodal durations and intervals between LEH (short and long).

H<sub>2</sub>-Grouped LEH (modes, individuals) show contrasting intensity and/or severity.

Acceptance of the null hypotheses (nonrandom) would suggest only a single stressor type while rejection would suggest that *H. naledi* experienced different classes of stressor.

## 2 | MATERIALS AND METHODS

### 2.1 | Sample selection

The Dinaledi Chamber within the Rising Star cave system dates from ca. 241 to 335 kya (Robbins et al., 2021). The dental sample on which this study is based, and their methods of analysis, have been previously described (Skinner, 2019). Teeth were selected for casting if any LEH were clearly observed and perikymata possibly countable; that is, we selected relatively unworn teeth since our goal is not to try to determine prevalence but to find, if possible, the time within and between episodes of developmental stress preserved as LEH. Molds were created by Mark Skinner and Debbie Guatelli-Steinberg (details in Skinner et al. (2019)). Our tooth identifications follow those in Delezene et al. (2023). Table 1 includes the associations of teeth for individuals proposed by these authors. In the present study, there are four designated “individuals” (2, 3, 4, 6) plus two more, based on duplicated teeth; that is, six individuals, at least (Table 1) out of 9 sets of reasonably associated teeth from the site (Delezene et al., 2023). Each discrete LEH on a tooth is considered independent of another on the same tooth and forms the unit of analysis. There are 82 LEH among 31 identified teeth. Each tooth was assessed for LEH, initially in isolation, and only later grouped by the newly realized associations.

The presence and decile location of each LEH defect were assessed through images created by Keyence digital and JEOL JSM-6490LV scanning electron microscopy (SEM) (see Data S1). The entire labial surface from cusp tip to cervix was examined. The number of perikymata within the occlusal wall and between onsets of successive LEH was counted, where possible. In those instances where perikymata counts had to be predicted rather than directly observed, allowances were made for the fact that perikyma generally decrease in width and increase in number progressively from occlusal margin to cervix, a phenomenon especially marked in *H. naledi* (Guatelli-Steinberg et al., 2018). For example, where labial wear precluded direct perikyma counts, measured LEH occlusal wall widths were divided by specific tooth (incisor/canine) mean perikyma width for that decile. Similarly, where perikymata counts between successive LEH could not be made, the distance, measured on scaled composite SEM images showing the LEH interval, was divided by tooth specific mean perikyma width to predict inter-LEH perikymata counts (details in Skinner, 2019).

Based on the fact that not all the teeth can be reliably associated into discrete individuals, we adopt two analytical approaches. Initially, all teeth are aggregated for analysis but separated simply into tooth types (incisors and canines). Raw counts of perikymata (predicted or observed) in the occlusal walls show “duration” of developmental stress while counts of perikymata between onsets of successive LEH

show inter-LEH interval (data available in Table S1). In other words, an interval spans from onset to onset of two LEH and includes the first furrow within the interval. Secondly, in order to benefit from the association of teeth into individuals, for particular analyses, all observations are converted to z-scores of perikymata counts based on each grouping of tooth type and association; for example, incisors from association 2, combined with canines from association 2, added to incisors from association 3, and so forth, up to and including a final grouping each of unassociated incisors and canines. Associated teeth, in the present study, account for 17 teeth out of 31, slightly more than half the sample.

### 2.2 | Subjectivity of scoring presence/absence of LEH

When antimeres are present in intact jaws, it has been customary in earlier studies to score LEH as “present” only if a defect is visible to the naked eye on both teeth; see discussion in Guatelli-Steinberg and Benderlioglu (2006). The consequence of this conservative approach is an increase in replicability but also an increase in the threshold for detection. In our study of isolated teeth, all LEH on each tooth were evaluated through comparison of visibility among naked eye, digital photographs, and, the final arbiter, highly magnified and detailed images obtained through SEM. This approach should ensure that, within a single tooth, reasonable consistency in evaluating enamel topography is maintained and that comparatively lesser defects are not missed; the disadvantage of this approach is that salience of a particular LEH affects how the others on the tooth are perceived. SEM images of each tooth with LEH indicated are shown in the accompanying Data S1, which compares identified LEH among groupings of associated anterior teeth. We acknowledge the challenge of correctly homologizing LEH among teeth, later brought into association as possibly being from a single individual. We revisit this issue in the discussion; but emphasize that since our focus is not on prevalence but interval between successive LEH on a tooth, any tendency to preclude an LEH would simply be a more conservative approach to our study of the phenomenon.

### 2.3 | Renumbered LEH

LEH were numbered from occlusal tooth tip to cervix for each tooth separately since at the time of our data collection we did not know which teeth might be associated. In light of our later ability to assign some teeth to individuals, it became possible to homologize LEH among the anterior teeth with heightened confidence and accuracy (see Data S1). However, a pair of LEH shared between two teeth might, on occasion, be separated on one of the teeth of the pair by an additional LEH not discerned on the other at the time of our initial examination, necessitating the renumbering of LEH to be as consistent as possible among all associated teeth. Consequently, we have provisionally renumbered the LEH as shown in Table 2. There are

**TABLE 2** Proposed renumbering of LEH on “associated” teeth (see also Data S1).

Association	Tooth identification	Originally assigned LEH number	Proposed numbering	Effect
2	1005B LoRI1	1	1	
		2Reg <sup>a</sup>	2	
		3	3	
		4	4	
	0998 LoLI2	1	1	
		2Reg	2	
		3	3	
		4	4	
	1005C LoRI2	1Reg	1	
		2	2	
		3	3	
		4	4	
	1076 LoLC	1Reg	2	Change
		2	3	Change
3		4	Change	
4		5	New	
377-1014 LoRC	1	3	Change	
	2	5	Change	
3	0931 ULI1	1	1	
		2	2	
		3	3	
		4Reg	4	
		5	5	
	1012 URI1	1	2	Change
		2Reg	4	Change
		3	5	Change
	0932 ULI2	1	1	
		2	2	
		3	4	Change
	0709 URI2	1	2	Change
		2Reg	4	Change
	0706 ULC	1	4	Change
		2Reg	6	Change
	0816 URC	1	4	Change
		2Reg	6	Change
4	1132 LoLI1	1	1	
		2Reg	2	
		3	3	
	1133 LoRI1	1Reg	2	Change
	1131 LoLI2	1Reg	2	Change
	1075 LoRI2	2	3	Change
		1	1	
		2Reg	2	
	1126 LoLC	3	3	
		1Reg	2	Change
2		4	New	

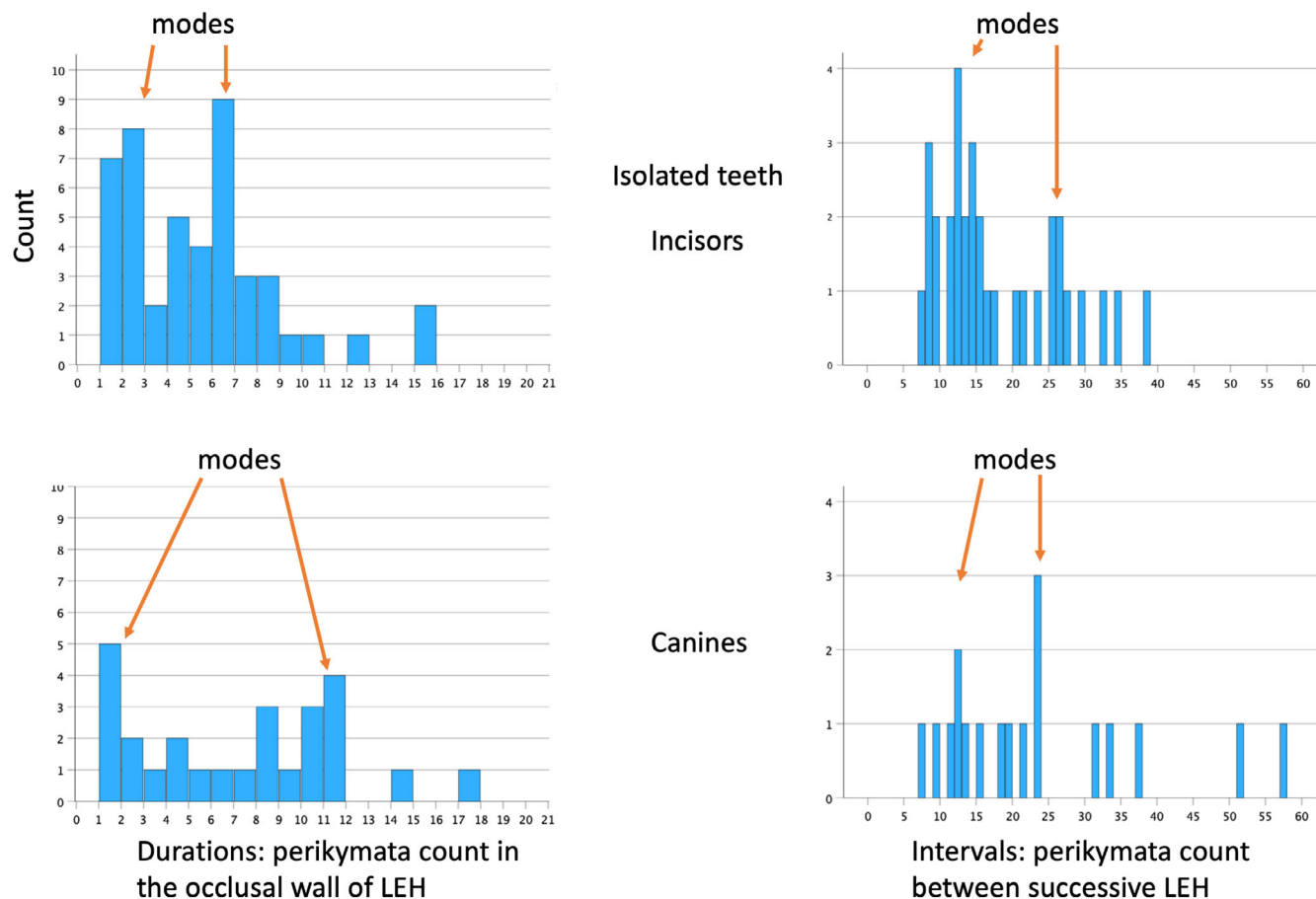
(Continues)

TABLE 2 (Continued)

Association	Tooth identification	Originally assigned LEH number	Proposed numbering	Effect
	0886 LoRC	1Reg	2	Change
		2	4	Change

Abbreviation: LEH, linear enamel hypoplasia.

<sup>a</sup>Reg(istration) indicates a specific LEH judged to be discernible on all associated anterior teeth (see Data S1).



**FIGURE 1** Distribution of counts of perikymata for linear enamel hypoplasia (LEH) durations and intervals separated by tooth type. Apparent modes are indicated.

23 changed numbers out of 48 LEH with an increase of two new events.

The frequency distribution of perikymata counts between and within LEH (Figure 1) shows apparent peaks, which may signal repetitive events. Such peakedness, however, can be due to chance, multiple RP, cycles in the etiological stressor, unequal cycles, and skipped cycles. The study of rLEH is not for the unwary or faint of heart.

## 2.4 | An evaluation of potential randomness of LEH data

To test for random occurrence and distribution of LEH, we invoke a hypothetical tooth with just over 100 perikymata (based on

perikymata counts for U.W. 101-0932 [URI2] and U.W. 101-0337 [URC]). In order to obtain an interval between two LEH, we assigned onset of both LEH by a perikyma number between 2 and 99 perikyma taken from a random number generator; the only proviso being that the two LEH had to be at least three perikymata apart. The number of perikymata between adjacent LEH is counted.

## 2.5 | The problem of potential sample differences in perikymata formation rates

The reconstruction of time within and between LEH defects relies on the periodicity with which perikymata are formed. Two RPs have been reported for *H. naledi*: 9 and 11 days per perikyma (Mahoney

et al., 2024). If both periodicities are present in our sample, they may create bimodalities in reconstructed duration and interval that obscure any potential patterning of recurrence and inhibit our ability to seek the etiology of stress in *H. naledi*. We employ two strategies for removing the effects of differences in RP: ratio analysis and z-score transformation.

### 2.5.1 | Ratio analysis

In the current study, in addition to aggregated perikymata counts, we include the analysis of repetitive intervals (or durations) within single teeth by comparing the ratio of perikymata count for a particular interval (or duration) divided by the perikymata count in the preceding interval (or duration) on the same tooth. The advantage of this novel technique, which relies on the reasonable assumption that RP remains constant within a tooth (Dean, 1987; FitzGerald, 1998), is that the potential for different RP, among aggregated teeth, is obviated. This method can show unequivocally the cyclic nature of stressful events if the variation around a ratio of 1.0 is significantly less than expected by chance. Additionally, in the case of durations, handled the same way, this technique can show whether there is evidence of bimodally distributed durations suggestive of more than one class of stressor. Similarly, if episodes of LEH recur bimodally, but at unequal intervals within what is construed to be a yearly cycle, without any additional evidence of twice-yearly recurring stressors in the environment, then we can infer that there are two independent annual classes of stressor which together then create two LEH per year.

One has to be careful in the interpretation of the distribution of ratios. In that the distributions of ratios (e.g., of successive intervals or durations) are inherently skewed, ratios are converted to natural logarithms, which aids in the visual assessment of potential bimodalities. If symmetries in length (or duration) pertain, then there should be a lot of 1.0 ratios. If, instead, asymmetries in stress interval (or duration) are the case (due, for example, to two classes of stressor), then, with successive events compared along a tooth, there should be a lot of ratios that are <1.0 and >1.0, with few or no ratios of 1.0. Skipped intervals (due, for example to no disease or an unusually mild winter) will increase the ratios in both directions, depending on the degree of asymmetry in interval lengths or durations.

### 2.5.2 | z-score analysis

Conversion of measures to z-scores (standardized deviations) enables combining variables with different units. In our case, the proxy for time is the periodicity of Retzius layers expressed on the outer enamel surface as perikymata. Retzius periodicity varies among individuals, populations, and taxa but is thought to be almost invariant within the individual (FitzGerald, 1998) but see (McFarlane et al., 2021). Consequently, expressing deviation of a particular measure, say perikymata count between LEH, from the mean measure for that variable for associated teeth, allows for differences in RP between individuals, which enables the pooling of associated teeth for analysis.

## 2.6 | Metrical analysis

Renumbered LEH are used for analyses of defect salience. Measurements of discrete enamel furrows were taken with a "µsurf Mobile Plus" optical scanner (details in Skinner et al., 2019) and analyzed with µsoft Analysis Premium 6.2 software from NanoFocus® AG (Oberhausen, Germany). Defect salience is measured fundamentally by angle of onset into the enamel furrow and by the daily deficit in enamel (Skinner, 2019, 2023). We reason that an elevated response to stress is reflected in a steeper entrance angle into the defect, as ameloblast secretion is reduced. This we term "intensity." Longer lasting defects get naturally deeper (Guatelli-Steinberg, 2003); therefore average daily enamel deficit takes into account how long the stress response lasts; we term this "severity."

## 3 | RESULTS

Our first task is to examine whether observed inter-LEH perikymata counts in the sample could be generated simply by chance. This potentiality has been recently examined in evaluation of LEH among orangutan teeth concluding that the frequency distributions were not random (Skinner & Ji, in review). As described above, here we created randomly spaced intervals on a tooth composed of about 100 perikymata in the imbricational enamel. This was repeated 200 times for comparison with actual data observed in this study (Table 3).

Evidently, the distribution of intervals between LEH differs from random significantly; intervals for *H. naledi* span far fewer PK (and hence time) and show a lower coefficient of variation.

### 3.1 | Evidence of bimodalities in the dataset

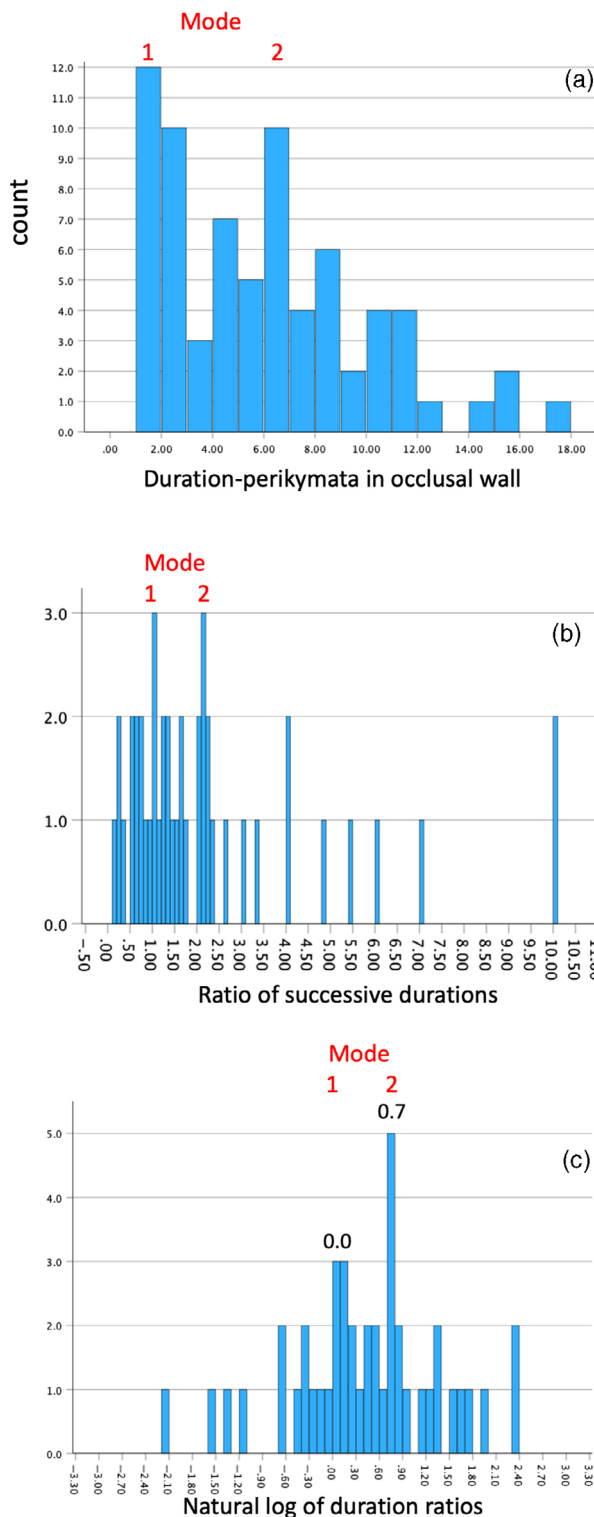
Figure 1 shows unconverted perikymata counts for the whole sample (ignoring associations of teeth) separated into tooth types (incisors

**TABLE 3** Comparison of perikymata counts between observed and randomly created LEH.

Measure	Inter-LEH perikymata count (n)	
	Observed (51)	Randomly spaced (200)
Median <sup>a</sup>	15.9	25.0
Interquartile range	13.8	32.8
Mean ± SD	19.8 ± 10.9	32.3 ± 21.8
Skewness	1.46	0.76
Kurtosis	2.48	-0.39
Range	50.0	88.0
Coefficient of variation (%)	55.1	67.5

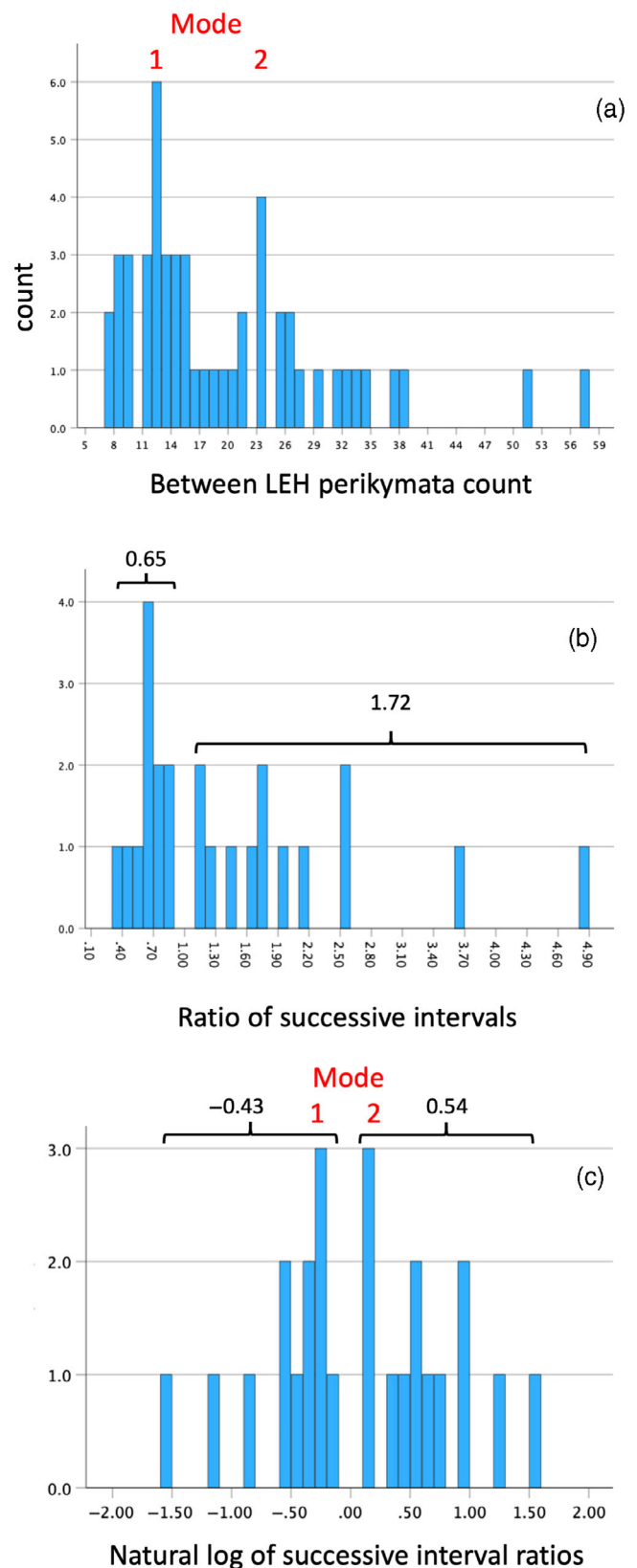
Abbreviation: LEH, linear enamel hypoplasia.

<sup>a</sup>Mann-Whitney test;  $z = -3.651$ ,  $p < 0.001$ .



**FIGURE 2** Evidence for two durations of stressor: (a) distribution of number of perikymata in the occlusal wall of defects, (b) successive ratios of durations (count of occlusal wall perikymata) to previous count on the same tooth, and (c) natural logs of duration ratios shown in “(b)” (to remove skewing).

and canines) for both durations and intervals. There are apparent modes in frequency, shown in Figure 1, for both durations and intervals. However, bimodality can be created by a mix of RP or by



**FIGURE 3** Evidence for two intervals of stressor: (a) distribution of number of perikymata between onset of defects, (b) successive ratios of an interval to previous interval on the same tooth, and (c) natural logs of interval ratios above. The indicated clusters of panel “(b)” are the median antilog values shown in panel “(c).” LEH, linear enamel hypoplasia.



**TABLE 4** Idealized frequency peaks of within-tooth ratios of successive inter-LEH perikymata counts to previous interval (constrained to sum to 12 months).

	Symmetric intervals		Asymmetric intervals <sup>a</sup>	
	Semiannual stressors		Twice-yearly stressors	
	6:6 months		5:7 months ( $\pm 17\%$ difference)	4:8 months ( $\pm 33\%$ difference)
Ratios if no skipped intervals	1.00	0.71/1.40 <sup>b</sup>	0.50/2.00 <sup>b</sup>	
Ratio if shorter interval occasionally skipped	Additional ratio 0.50	Additional ratios 0.42/1.71	Additional ratios 0.33/1.5	
Ratio if longer interval occasionally skipped	Additional ratio 0.50	Additional ratios 0.58/2.40	Additional ratios 0.67/3.00	

Abbreviation: LEH, linear enamel hypoplasia.

<sup>a</sup>Ratios of recurrent asymmetric intervals are right skewed but can be normalized by conversion to natural logs (Figure 3).

<sup>b</sup>There will be relatively few ratios of 1.0.

asymmetric intervals. These possibilities are examined through application of the following methods.

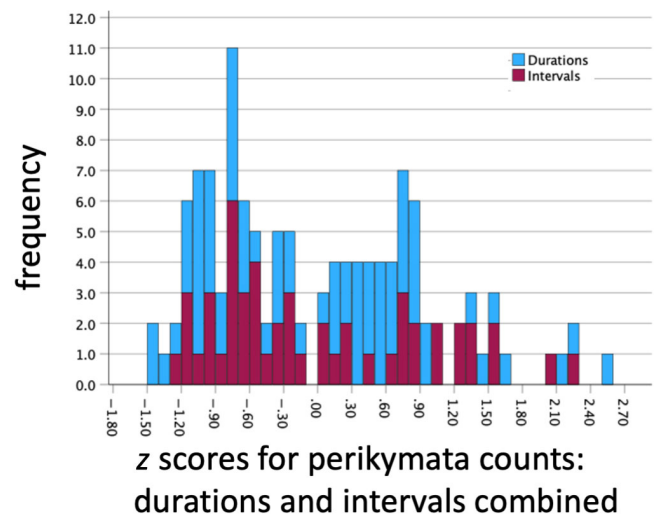
### 3.1.1 | Ratio analysis

To examine whether bimodalities are created by different RP within the sample, we turn now to ratio analysis in which successive measures are divided by previous measures on the same tooth. The results, for duration analysis, are shown in Figure 2 (tooth types combined).

With tooth types combined, there are apparently two modes of duration centered on about 1–2 and 6–7 perikymata (panel “a” in Figure 2). It seems unlikely that differences in Retzius periodicity could account for such a large difference. Indeed, the ratios of successive measures to previous on the same tooth, shown in panels “b” and “c” (converted to natural logs to minimize skewing), show two modes, of which the second is twice as long in duration as the first. The first mode around ratio 1.0 reflects that many successive episodes are of the same duration while the second mode reflects those stressful episodes of longer duration. If one splits the durations into two groups (<5 PK, 5 + PK), the median duration of each group is 2.0 and 8.0 perikymata, respectively.

Turning now to the interval between successive LEH, Figure 3 shows that, as for durations, there are apparently two modes. If one splits the bimodally distributed intervals shown in Figure 3a into two groups above and below the median interval (<16 PK, 16 + PK), the median interval of each separate group is 12 and 25 PK, respectively. This large difference may, of course, be exaggerated by differences in Retzius periodicity. However, ratio analysis (which removes the effect of RP) shown next in Figure 3b,c shows a dearth of naturally logged ratio values at 0.0 indicating the presence of asymmetry in interval lengths in the data. If one then splits the interval data into two groups above and below 0.0, the median antilog ratio value for each group becomes 0.65 and 1.72, respectively.

As may be seen in Table 4, which presents a table of idealized ratios of shorter and longer (asymmetric) intervals in months (summing to 12 months), the intervals in our study conform fairly closely to a 5 month: 7-month asymmetry, including skipped shorter intervals.



**FIGURE 4** Bimodal distribution of perikymata counts in occlusal wall of linear enamel hypoplasia defects and between defects (based on tooth type and associated teeth, where known).

If, on the other hand, one assumes that there are no skipped intervals, and that the two intervals must sum to a 12-month span, the best fit would be a 4.5 and 7.5-month pair of intervals (yielding ratios of 0.60 and 1.67; cf. the observed median ratios noted above of 0.65 and 1.72).

### 3.1.2 | z-score analysis

The impression that durations and intervals both show bimodality should not be surprising if underlying etiologies are shared between the two variables. In terms of the goal of elucidating etiology, we consider the inference important. A test of this idea can be created by combining them into a single analysis. This is accomplished by first grouping the teeth into associations (2, 3, 4, and unassociated) identified in Delezenne et al. (2023) where the value of a perikymata count is compared with the mean for a grouping of tooth type and association and converted to a z-score. An advantage of converting perikymata

Tooth	Measure	Duration		Interval	
		Short ( $z < 0$ )	Long ( $z \geq 0$ )	Short ( $z < 0$ )	Long ( $z \geq 0$ )
Combined tooth types	<i>n</i>	35	37	29	22
	Mean $\pm$ SD	2.8 $\pm$ 2.0	8.8 $\pm$ 3.1	13.6 $\pm$ 7.9	27.8 $\pm$ 8.9
	Median	2.1	8.0	12.6	25.9

Abbreviation: LEH, linear enamel hypoplasia.

counts into z-scores, beyond enabling the combination of teeth into groups thereby increasing sample size, is that, by doing so, potential differences in Retzius periodicity between individuals and tooth types are obviated. The results of this analysis are shown in Figure 4.

There is clear evidence of bimodality in the z-score data on combined durations and intervals between LEH. In other words, there are two different durations and two different intervals between LEH. We label these temporal groups into “short” and “long”, and will now consider each separately to see whether their properties support the inference of two different classes of stressor.

### 3.2 | Durations of developmental stress

Initially, we measure duration of developmental stress simply in terms of the number of perikymata in the occlusal wall. We divide these bimodally distributed data (Figure 1) into whether their z-scores, based on tooth type and “association”, are above or below zero (see analysis above). Table 5 shows that the longer duration mode is about 4 $\times$  the shorter mode.

### 3.3 | Intervals between LEH

Similarly, we measure intervals between bouts of developmental stress by inter-LEH perikymata counts. We divide these bimodally distributed data (Figure 1) into whether their z-scores were above or below zero (see analysis above). Table 5 shows that the longer interval mode is about 2 $\times$  the shorter mode.

### 3.4 | Within tooth variation

It is now possible to compare the distribution of LEH along the developing crown from tip to cervix with more confidence (Table 6).

Quite clearly, there is a strong statistical tendency for the earliest numbered episodes of developmental stress to be of short duration (combined tooth types, chi square value = 17.806, df = 5,  $p = 0.003$ ).

We now examine the metrical differences in LEH salience both between short and long duration defects and during crown formation (Table 7).

**TABLE 5** Average perikymata counts of bimodally distributed LEH durations and intervals, for “associated” teeth, separated into modes by z-scores (see Figure 4).

Other than a tendency for LEH 1 to have a low angle of onset, there are no apparent trends of increase or decrease in LEH measurements during crown formation for either duration mode (short vs. long) as shown by LEH number. In this analysis, when all LEH are combined, those of short duration are significantly shallower with a more gradual slope; that is, they seem less salient. However, this is misleading since it is well known that, due to the way in which enamel layers are deposited, longer lasting LEH become naturally deeper as well (Guatelli-Steinberg, 2003). To avoid this problem, “daily enamel deficit” (last column in Table 7) is calculated (Skinner, 2023) by dividing depth by the number of days over which an LEH formed (PK count in occlusal wall  $\times$  Retzius periodicity: 9 or 11 [Mahoney et al., 2024]). Indeed, as shown in Table 7, the daily enamel deficit is significantly greater among short duration LEH. Thus, despite short duration defects being relatively shallow and showing a less steep angle of descent into the enamel furrow (i.e., showing less “intensity”), they show more “severity” than do long duration defects.

### 3.5 | Estimates of duration and interval in real time

Our emphasis now shifts from the anatomical distribution of LEH among anterior teeth from *H. naledi* to their timing. Because earlier analysis (Skinner, 2019) had suggested that LEH intervals were sub-annual, we turn to the conversion of LEH durations and intervals into estimates of the time involved for these events, drawing upon newly obtained RP for *H. naledi*. We consider RP of both 9 and 11 days (Table 8), although only the latter is reported for canine teeth; there are no RPs for incisors.

Several remarks may be made about the values in Table 8. Comparison of LEH among “associated” teeth (see Data S1) suggests that not all teeth from an individual show all LEH, that is, there are skipped episodes (creating very widely separated intervals); this phenomenon is considered in the footnotes to Table 8. Keeping sample sizes in mind, there is no apparent trend along the crown for durations or intervals to change. Consequently, accepting the combined values as generally indicative, short durations last about 3 weeks while long durations last about 12 weeks, that is, 4 $\times$  as long. Short intervals straddle 4 months while long intervals straddle 8 months, that is, a combined span (allowing for skipped

**TABLE 6** Distribution of short and long duration LEH along the developing tooth crown.

Renumbered LEH	Incisor		Canine		Decile	Proportion (%) of short durations	
	Short	Long	Short	Long		Incisor	Canine
1	11	1	5	1	4	100	
2	6	9	1	4	5	100	100
3	3	7	1	4	6	73	60
4	2	5	3	3	7	40	60
5	1	1	1	2	8	29	33
6			1		9	11	14
					10	100 <sup>a</sup>	100 <sup>a</sup>
Total	23	23	12	14			

Abbreviation: LEH, linear enamel hypoplasia.

<sup>a</sup>Preponderance of short duration defects in decile 10 is probably an artifact of small sample size ( $n = 5$ ) and enamel geometry.

**TABLE 7** Difference in LEH median depths, angles of onset, and daily enamel deficit compared between those of short and long duration (values for separate and combined LEH are shown) (LEH are renumbered according to associations in Delezene et al. (2023))<sup>a</sup>.

Duration mode	Renumbered LEH	$n$	Depth ( $\mu\text{m}$ )	Onset angle ( $^{\circ}$ )	Enamel deficit per day ( $\mu\text{m}$ )	
					If RP = 9	If RP = 11
Short	1	16	10.0	2.04	0.50	0.41
	2	7	9.90	3.03	0.48	0.39
	3	4	8.76	2.52	0.49	0.40
	4	4	13.00	2.78	0.35	0.29
	5–6	3	14.91	3.16	0.86	0.71
Kruskall–Wallis all 6 LEH	df = 5	$H$	5.126	4.817	2.462	
		$p$	0.401	0.439	0.782	
Long	1	2	18.54	2.44	0.19	0.15
	2	13	17.21	3.76	0.32	0.26
	3	11	23.47	3.52	0.26	0.21
	4	7	15.13	3.38	0.24	0.20
	5	3	25.11	3.83	0.40	0.33
K–W	df = 4	$H$	1.962	2.164	2.107	
		$p$	0.743	0.706	0.716	
Combined LEH						
Short		34	10.0	2.35	0.40	0.38
Long		36	19.9	3.48	0.30	0.24
Mann–Whitney		$z$	–5.041	–2.256	–2.797	
		$p$	<0.001	0.024	0.005	

Abbreviations: LEH, linear enamel hypoplasia; PK, perikymata; RP, Retzius periodicity.

<sup>a</sup>These new data on antimeres showed that one canine's measurements of depth and angle (U.W. 101-0706) were clearly in error (allowing their exclusion from this analysis).

LEH) with a mean of 1.04 years. Considering the crudity of the methods and the many analytical steps that went into their creation, we think that it is safe to conclude that, in the environment of *H. naledi*, there are twice-yearly episodes of stress, asymmetrically timed in an annual cycle (at ca. 4–8-month intervals; cf., 4.5–7.5-month, based on ratio analysis [see earlier]).

### 3.6 | Ontogenetic timing

A sequence of any three LEH will likely span about a year. Just to be clear, one is not including LEH3 except as a demarcator of the endpoint of the preceding second interval. Rather, LEH3 denotes the start point of the next interval. Notably, renumbered

**TABLE 8** Conversion of median perikymata counts to estimates of time for LEH durations and intervals between LEH.

Measure	Renumbered LEH	n	Short mode			n	Long mode		
			PK count	If RP 9–11			PK count	If RP 9–11	
				Days	Months			Days	Months
Durations	1	16	1.9	17–21		2	12.5	113–138	
	2	7	2.9	26–32		13	6.8	61–75	
	3	4	2.5	23–28		11	8.9	80–98	
	4	5	4.0	36–44		8	7.0	63–77	
	5	2	2.2	20–24		3	8.4	76–92	
	6	1	11.2	101–123					
	All		35	2.1	19–23		37	8.0	72–88
Intervals	1–2	14	14.4	130–158	4.3–5.2	12	24.3	219–267	7.2–8.8
	2–3	10	12.3	111–135	3.6–4.4	6	25.9	233–285	7.7–9.4
	3–4	3	9.4	85–103	2.8–3.4	4	33.0	297–363	9.8–11.9
	4–5	2	10.4	94–114	3.1–3.7				
	All		29	12.6 <sup>a</sup>	113–139	3.7–4.6	22	25.9 <sup>b</sup>	233–285

Abbreviations: LEH, linear enamel hypoplasia; PK, perikymata; RP, Retzius periodicity.

<sup>a</sup>If 4 intervals that include a skipped LEH are excluded, mean perikymata count for short intervals becomes 12.0 (converting to 108–132 days, mean = 120 days).

<sup>b</sup>If 3 intervals that include a skipped LEH are excluded, mean perikymata count for long intervals becomes 26.0 (converting to 234–286 days, mean = 260 days).

**TABLE 9** Distribution of LEH occurrence along the tooth crown.

Decile	Incisor (n = 18)				Canine (n = 13)				
	Number of LEH	PK per decile	RP9-11	Yearly LEH acquisition rate <sup>a</sup>	Number of LEH	PK per decile	RP9-11	Yearly LEH acquisition rate	
				If RP = 9				If RP = 9	
1	0	3.00	3	27.0–33.0	0				
2	0	5.00	9	45.0–55.0	0	5.00	3	45.0–55.0	
3	0	5.31	13	47.8–58.4	0	6.67	6	60.0–73.4	
4	2	5.47	15	49.2–60.2	0.99	10.38	8	93.4–114.2	
5	3	6.50	16	58.5–71.5	1.17	10.50	6	94.5–115.5	1.93
6	11	8.00	13	72.0–88.0	4.29	14.00	5	126.0–154.0	2.90
7	10	12.00	9	108.0–132.0	3.76	13.20	5	118.8–145.2	3.69
8	7	15.80	5	142.2–173.8	3.60	21.4	5	192.6–235.4	2.65
9	9	21.14	7	190.3–232.5	2.47	25.33	6	228.0–278.6	1.87
10	3	12.29	7	110.6–135.2	1.40	10.8	5	97.2–118.8	1.50
Total	45	94.5			Tooth mean = 2.53	30	117.3 <sup>b</sup>		Tooth mean = 2.42

Abbreviations: LEH, linear enamel hypoplasia; PK, perikymata; RP, Retzius periodicities.

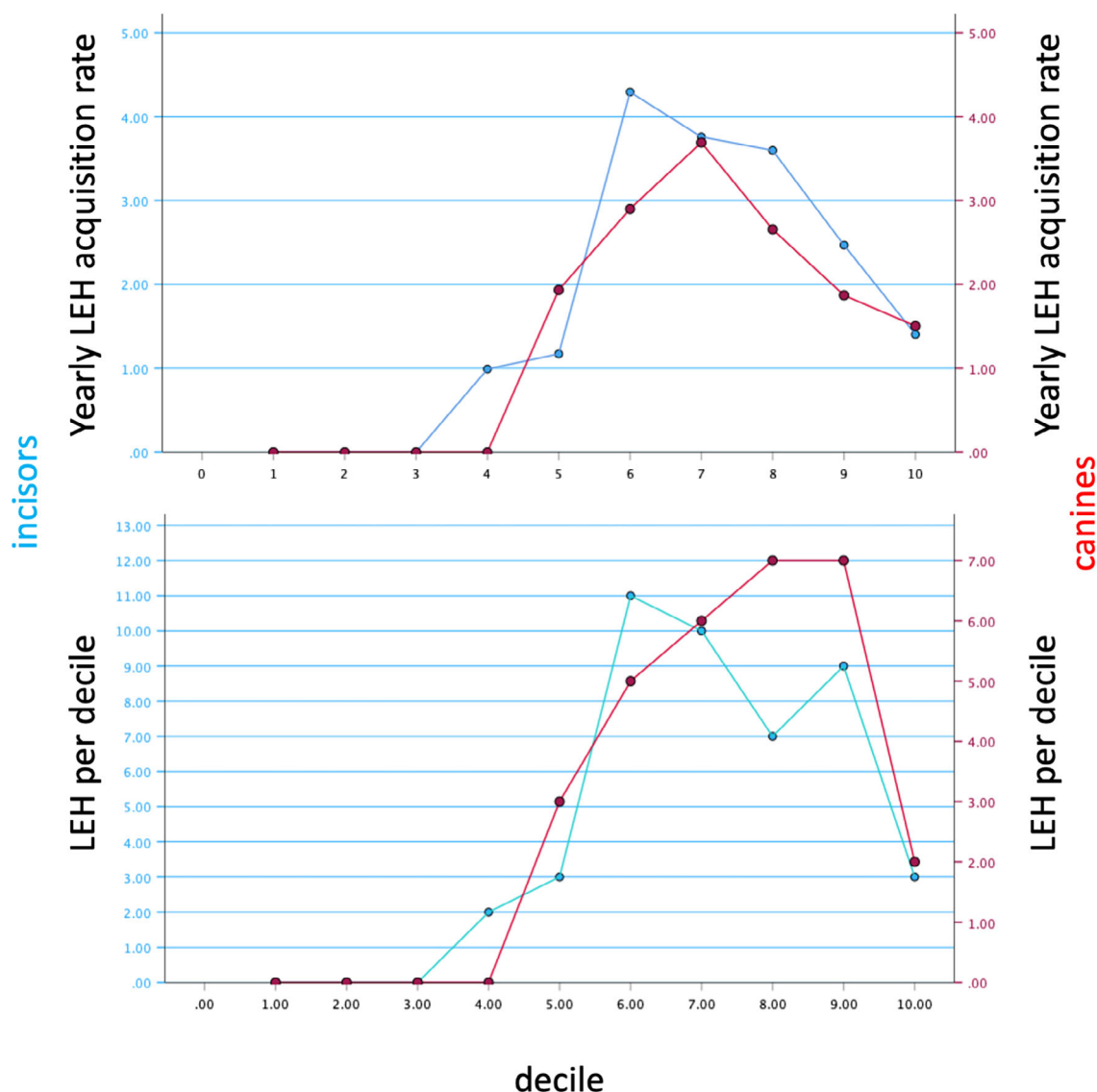
<sup>a</sup> $365.25 \times ((\text{Number of LEH in a decile}/n)/[\text{PK per decile} \times \text{RP}])$ .

<sup>b</sup>Excludes decile one.

LEH1 through LEH3 account for 74% of all LEH in our data. In other words, most anterior teeth capture only about 1 year of developmental stress despite some crowns taking as long as 5 years to form (Mahoney et al., 2024) presumably because

developmental stress only commences relatively late in crown formation (Table 9).

The rates of LEH acquisition per decile for each tooth type are shown in Table 9 and Figure 5. While the mean yearly rates of LEH



**FIGURE 5** Evidence for delayed canine crown formation relative to incisors in the aggregated sample: upper panel-yearly rate of linear enamel hypoplasia (LEH) acquisition per decile for separated tooth types; lower LEH per decile of crown height.

acquisition are much the same in both types (incisors 2.5, canines 2.4; and, for combined tooth types, 2.1 and 2.0, if  $RP = 9$  or 11, respectively), it may be noted that LEH acquisition starts and peaks later in canines. The bottom panel of Figure 5 shows that LEH per decile peaks in deciles 6 and 7 for incisors versus deciles 8 and 9 for canines. Among modern human children decile formation takes on average  $1.29\times$  as long for canines as incisors (Reid & Dean, 2000). Among *H. naledi* we calculate, from Table 9, the ratio is 1.33, an insignificant difference ( $t = -0.477$ ,  $df = 17$ ,  $p = 0.639$ ). This delay is also shown in the “registration LEH” used in matching LEH among associated teeth (Data S1). In other words, the rate of canine tooth formation relative to the incisors is the same in both taxa of *Homo*.

## 4 | DISCUSSION

### 4.1 | Findings

LEH was recorded in 31 isolated anterior teeth (18 incisors, 13 canines with a total of 82 defects) attributed to *H. naledi*. The most important observation of our study is that durations of LEH and the interval between successive defects are both bimodally distributed. We attribute this pattern to the existence in the environment of these hominins of two classes of stressor. This inference is supported by the following observations:

- LEH are of short (ca. 3 weeks) and long duration (ca. 12 weeks);

- b. Short duration defects have, in comparison with long duration defects, shallow entrance angles but a higher rate of daily enamel deficit;
- c. First episodes of developmental stress are usually of short, rather than long duration;
- d. Intervals between defects are of unequal length: ca. 4.5 versus 7.5 months on average.

We propose that the results are best explained as the consequence of separate annual cycles of disease and dearth and that our findings, based on newly derived RP, make ecological sense.

## 4.2 | Caveats and considerations

In studies of any mortality cohort, one must acknowledge that the observed characteristics of a sample may not be representative of the living assemblage (Wood et al., 1992). For example, while we found that short duration defects were more severe, we must also countenance the possibility that those individuals who experienced prolonged and more severe stress had already died and are not represented in our sample. One advantage of studying developmental defects formed in infancy, but observed in erupted teeth from older individuals, is that we know they survived to at least the age of reproduction and, to a limited extent perhaps, reflects the important effects of natural selection. Another consideration is that LEH furrows are naturally less salient in more occlusally forming enamel due to the shallow angle with which enamel layers are deposited initially in imbricational enamel (McGrath et al., 2019). We feel this problem is less acute in our particular sample where most of the defects, as we have shown, are located in the cervical half of the dental crowns.

### 4.2.1 | Disease as putative stressor

As noted in the introduction, biotic stressors, such as certain disease processes can be severe but relatively brief immunologically. Our model of “two classes of stressor with asymmetric incidence,” to explain the patterning of LEH in *H. naledi*, is consistent with recent observations of disease occurrence in chimpanzees and modern humans. For example, viruses are among the most important agents of morbidity and mortality among apes (Negrey et al., 2022). Kanyawara (Uganda) chimpanzees who live in an environment with two rainy peaks per year show only a single annual peak in respiratory disease (Emery Thompson et al., 2018). Among humans, acute coughs due to respiratory disease resolve within 25 days, the common cold by 15 days and nonspecific respiratory symptoms by 16 days (de Benedictis & Bush, 2018). These figures are consistent with the median duration of short episodes of LEH in *H. naledi* (19–23 days).

If the long duration defects relate to a time of stress that is coincident with a lean austral winter then, under our model, another stressor would have to have occurred about 4 months earlier or later, that is, February/March or October/November. Enamel hypoplasias

are a common sequelae of respiratory tract infections (Alaki et al., 2008). In South Africa today, the peak of respiratory syncytial virus (RSV) is from February to May (Kyeyagalire et al., 2014). Furthermore, RSV is a ubiquitous disease among infants around the world and easily infects chimpanzees. Initially, maternal antibodies to RSV are protective, delaying developmental stress. Primary infections are symptomatic and do not confer immediate immunity, while incidence peaks in the wintertime (Ogra, 2000). The features of this disease process are consistent with the patterning of LEH in *H. naledi* and provide one potential underlying cause of the LEH pattern that we observed.

### 4.2.2 | Dearth as putative stressor

As noted in the introduction, we expect abiotic stressors to be relatively mild but of longer duration as they are controlled by orbital forces (Kingston, 2005). Phenophases of leafing and fruiting, for example, are cued by insolation (Borchert et al., 2015; Rivera et al., 2002). Dearth may connote simple undernutrition but in the case of apes and their kin usually refers to a seasonal decline in preferred foods, especially fruit, which are more or less successfully replaced or augmented with fallback foods (Harrison & Marshall, 2011; Marshall et al., 2009). A lean season can be offset through nutrient switching (e.g., Marshall et al., 2009) but may expose herbivores to antifeedants in secondary plant compounds (Cooper & Owen-Smith, 1985). Baboons in the Drakensburg mountains of South Africa consume a narrowed range of plants, rich in tannins, during a late winter period lasting about 6 weeks (Byrne et al., 1993). Infant baboons, and presumably infant hominins who feed from their mother's dietary residue, or who self-forage, will likely ingest seasonally high secondary plant compounds (Altmann, 2009).

In the context of seasonal shortages and/or substitutions, it is worth emphasizing that the reconstructed median duration of the long episodes of enamel hypoplasia among *H. naledi* is almost twice as long as any hitherto reported median durations among apes: chimpanzees (7 weeks), bonobos (9 weeks), and orangutans (6–8 weeks) (Skinner, 2021; Skinner & Skinner, 2017). Interestingly, the median number of perikymata in the occlusal wall of enamel furrow defects is quite similar across fossil hominin taxa: Krapina Neandertals-5.5 (range, 2–12) (Guatelli-Steinberg, 2008); *Australopithecus africanus*-6.0 (range, 5–12) (Skinner, 2019); East African *Australopithecus*-7.5 (range, 4–14); *Paranthropus* 5.8 (range, 5.0–6.5) (Guatelli-Steinberg, 2004); *H. naledi*-8 (range, 5–17) (“long” defects). It is the relatively high RP of *H. naledi* combined with slightly higher perikymata counts that translate into very long duration of some episodes of developmental stress for this taxon. Collectively, these taxa inhabited a large range of habitats to which each was adapted, but whether they are comparable in terms of stress duration as measured by perikymata counts has not yet been determined. This does not imply that cold distress is, for example, less in Neanderthals than in *H. naledi*. We are exploring cold distress and LEH in snow monkeys from northern Japan as a potential guide to such considerations (Skinner et al., in preparation).

While the two classes of stressor we have considered (disease and under nutrition) may explain the pattern of rLEH among *H. naledi*, there are clearly other potential stressors. Thermal distress is another possible stressor in the South African Highveld that is marked today by stark seasonality and a long cold dry season in the austral winter, with occasional below freezing temperatures (Paine et al., 2019; Skinner, 2021). However, undernutrition and hypothermia are both metabolic in nature, synchronous, and likely indistinguishable in terms of their effect of enamel formation. Currently, we are unable to distinguish between metabolic stress due to undernutrition and that due to seasonal cold, nor need they be mutually exclusive. However, given the long duration of the stress (10–13 weeks) among *H. naledi*, we think sustained food shortage is probably the more potent “long duration” stressor. Nevertheless, the potential role of secondary plant compounds as a factor to explain rLEH among low-latitude apes should be explored in future studies.

### 4.3 | Meaning and significance

Recently, an index termed enamel deficit ratio (EDR) has been proposed that allows meaningful comparison of samples despite potentially differing daily secretion rates (DSR) and differing daily deficits in enamel deposition (the LEH furrow) (Skinner, 2023). Drawing upon DSRs for canine outer lateral enamel reported in Mahoney et al. (2024), the EDR for short and long duration events in *H. naledi* is 9.4% and 5.7%, respectively. These values are low in comparison with those reported for fossil apes from South East Asia by (Skinner, 2023) and for Japanese macaques (Skinner et al., in preparation) (there are as yet no published EDRs for extant apes) indicating that the impact of developmental stress during infancy and childhood may be comparatively low for *H. naledi*.

In order to reconstruct the quality of early life experiences among *H. naledi*, we must keep in mind the infant's reliance, especially at first, on maternal milk for nourishment and passive immunity. Apart from fairly regular episodes of developmental stress of short and long duration, enamel surfaces in *H. naledi* are normal (see [Towle & Irish, 2019, 2020] for a minor exception) with no hint of what is thought to be weaning-related coronal waisting observed in some chimpanzees (Skinner et al., 2012). We have not attempted to reconstruct the age of weaning in *H. naledi* as we lack recourse in our data to the neonatal line. However, the concentration of enamel defects in the cervical half of anterior teeth could reflect the weaning process and increasing pressure on the infant to survive nutritionally on its own (Dettwyler, 1995) with, possibly, increased exposure to secondary plant compounds (Lambert, 2009).

It does not necessarily follow that the mild degree of developmental stress we describe for *H. naledi* should manifest in the postcranial skeleton. Generally, LEH is deemed a negative phenomenon that is typically related to stress. If, however, LEH is linked to the seasonal ingestion of secondary plant compounds inimical to matrix secretion during amelogenesis, but signaling recourse to a fallback food that

enables the animal to weather a lean season, then enamel hypoplasia is a positive sign, an example, perhaps, of the osteological paradox (Wood et al., 1992). Indeed, there is evidence, provided from pig husbandry, that enamel thinning as a response to developmental stress (probably fever) links to prolonged survivorship (Skinner et al., 2019). In that study, we concluded that each of the major oral tissues (enamel, dentin, and bone) respond to the same stressors in their own specific way. Consequently, we would not necessarily expect the mild degree of developmental stress, we describe here for *H. naledi* to be expressed as postcranial skeletal defects. Moreover, the late secretory ameloblast, that responsible for LEH, is nearing the end of its lifespan and may be exquisitely sensitive to quite mild stress not recorded by other tissues with higher thresholds of response to stressors (Witzel et al., 2008). The relative vigor of the young ameloblast may explain why other scholars cannot detect seasonality of pathological striae deeper within ape enamel (Smith et al., 2010).

#### 4.3.1 | Sample and methods

There are several reasons why our results must be considered tentative and that undue significance should not be placed upon them. Our choice of sample is biased towards fairly unworn teeth; consequently, our data are based on a cohort that died relatively young. Also, our samples are small; we have not been able to remove completely the statistical redundancy inherent in a mixed sample of isolated teeth, even when trying to separate them into individuals. Moreover, we do not include later-forming second and third molar crowns; additional study on postcanine teeth is recommended to obtain a more complete picture of developmental stress throughout *H. naledi* ontogeny. Finally, our finding of bimodalities in both interval and duration are without precedent and require refutation or confirmation by others in subsequent studies. Despite the generally informative results of this analysis, it is clear that a better way has to be found to assess enamel topography so that isolated isomeres and antimeres from individuals match more closely. Particularly, we need to assess the whole labial/buccal surface at a sub-perikyma resolution that still permits the visualization of the much coarser furrow form defects. We think that this subjectivity problem is particularly suited to solution in future studies by recourse to machine learning coupled with enhanced scanning instruments.

#### AUTHOR CONTRIBUTIONS

**Mark Fretson Skinner:** Conceptualization (equal); data curation (equal); formal analysis (equal); investigation (equal); methodology (equal); project administration (equal); resources (equal); writing – original draft (equal); writing – review and editing (equal).

**Lucas Kyle Delezene:** Data curation (equal); funding acquisition (equal); writing – review and editing (equal). **Matthew M. Skinner:** Funding acquisition (equal); writing – review and editing (supporting).

**Patrick Mahoney:** Funding acquisition (equal); writing – review and editing (equal).

## ACKNOWLEDGMENTS

The authors are grateful to John Hawks for thoughtful comments on an earlier draft of this manuscript. Our research was supported by a workshop grant from the Wenner-Gren Foundation to L.K.D. and M.M.S. that funded data collection for M.F.S. and the Evolutionary Studies Institute at the University of Witwatersrand. Matt Skinner has also received funding from the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation programme (grant agreement No. 819960). L.K.D. thanks the Office of Research and Development at the University of Arkansas and the Connor Family Faculty Foundation for providing funding during the period of data collection. Access to specimens and technical assistance were provided by Bernard Zipfel and Lee Berger. P.M. acknowledges support from The Royal Society and The Leverhulme Trust (grant numbers RG110435 and RPG-2018-226).

## DATA AVAILABILITY STATEMENT

Raw data and scanning electron microscopy images are provided as Online Supplementary files with this contribution.

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**How to cite this article:** Skinner, M. F., Delezene, L. K., Skinner, M. M., & Mahoney, P. (2024). Linear enamel hypoplasia in *Homo naledi* reappraised in light of new Retzius periodicities. *American Journal of Biological Anthropology*, e24927. <https://doi.org/10.1002/ajpa.24927>