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Reevaluating Harris Lines – A Comparison Between Harris Lines and Enamel Hypoplasia

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A B S T R A C T

This study examines the association between Harris lines and enamel hypoplasia. This association is analyzed in terms of: 1) presence/absence of these markers in each individual, and 2) age of the individuals at the time of Harris lines and enamel hypoplasias formation. Data from two archaeological groups (Azapa-71 and Azapa-140) from northern Chile were analyzed. The results indicate Harris lines and enamel hypoplasias are not associated in terms of presence/absence. Moreover, the estimated age of the individuals at the time of Harris lines and enamel hypoplasia formation shows that these two markers have a very different distribution. While enamel hypoplasias clustered between ages 3 and 5, Harris lines were more commonly formed during the first year of life, as well as during adolescence, which are the periods of most accelerated growth. We propose that Harris lines are a result of a normal, rather than abnormal, saltatory growth process.

Key words: *Harris lines, enamel hypoplasia, saltatory growth*

Introduction

In order to survive, an organism must be able to respond to changing environmental conditions. If an environmental change takes place while an individual is growing and developing, phenotypic changes can occur in response to the stimuli[1]. Among these phenotypic changes, enamel hypoplasias and Harris lines are usually recognized as markers that result from stressful conditions[2–9]. These two markers are of special importance in paleopathological studies because they provide information about the health status of individuals over the course of their development.

There is no agreement, however, about the etiology of Harris lines[10–16]. Since Harris lines are not the inevitable consequence of health impairments or the necessary response to nutritional deprivation[17, 18] their interpretation as a stress indicator, and therefore utility in the reconstruction of populations' health status, remains debatable. Indeed, some studies found no correspondence between Harris lines and illness[14,15,17,19], and others indicate that Harris lines result from growth rate regulation at the epiphyseal cartilage plate, which seems to be associated with growth velocity and not necessarily with stressful conditions[12].

Despite these findings, Harris lines have often been interpreted as indicators of stress episodes, such as nutritional deficiencies and infectious diseases, that slow or stop growth[2,3,9,16,20–23]. This interpretation derives primarily from the traditional view of growth as a continuous process, in which interruptions are considered pathological[24]. New data, however, indicate that prenatal and postnatal human growth is neither smooth nor continuous, but a saltatory process, where increases in body size are the result of time-constrained growth episodes that occur intermittently[25–29] and not continuously[30,31]. Normal growth, then, occurs by »saltation and stasis« and can be characterized as a non-linear dynamic process characterized by a two-phase sequence: 1) The first phase, known as stasis or growth suppressed phase, is controlled by growth inhibitory proteins and defined by the absence of significant incremental growth; 2) the following phase, or saltation, is characterized by a discrete growth event. Periods of growth saltation are punctuated by period of stasis that range from 1 to more than 60 days during which no growth occurs. The time intervals between these discrete periods of growth vary both within and between individuals[29].

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Although the »saltation and stasis« pattern seems to be common to all individuals, the amount of growth at each pulse, or pulse amplitude, is variable within and between individuals, and fluctuates with age[25–27,29,32]. In fact, infancy and adolescence, the periods of most accelerated growth in humans[33], have more frequent and/or higher amplitude episodes of growth saltations[28,29].

Enamel hypoplasias result from developmental disturbances of the enamel and as such they record the interacting stresses of nutritional deficiencies and illnesses that occurred while enamel was being deposited[8]. During dental development, a variety of systemic stressors related to nutrition and infectious diseases can produce abnormal enamel growth patterns[4,6,23,34–39]. The interpretation of enamel hypoplasias as a stress marker, however, is problematic because sensitivity to their formation is inter-tooth specific, and their visibility varies with the morphology of the tooth. It is possible that this differential sensitivity biases the appearance of enamel hypoplasias in the dental record[5,8,40–42], and as a result, their interpretation.

The purpose of this study is to determine if an association between enamel hypoplasias and Harris lines exists, and thus to establish whether they respond to the same stressful events. The association will be tested at two levels: 1) degree of concordance in terms of presence or absence of these indicators in each individual; and 2) association between the age of the individual at the time of enamel hypoplasia and Harris line formation. Each marker will be assessed separately, and then compared with several statistical analyses to determine any association between them.

Tibial growth and Harris lines

Tibial growth in humans follows the general velocity growth curve. The tibia, therefore, grows at a rapid rate during the first year of life, after which growth decelerates[33] until the age of 9 when the rate of growth increases again. After that age, growth reaches a maximum velocity by 10–12 years of age in girls and by 12 –14 years in boys. Following this period of accelerated growth, growth velocity decreases rapidly until it ceases altogether around the age of 18 (Figure 1)[33,43,44].

Longitudinal bone growth is achieved by the coordinated recruitment, proliferation, differentiation, maturation and eventual death of the cells of the growth plate[45,46]. The basic zones of the cartilage plate are established during the third trimester *in utero* and are well defined at birth[47,48]. Harris lines, then, can appear from birth up to 15–16 years of age[47]. The lines result from the decoupling of osteoblastic and chondroblastic activity, where the former continues and the later stops or slows[12,49,50].

Several hormones and growth factors influence the process of bone growth, including glucocorticoids (GC), insulin, thyroxine, sex hormones, growth hormone (GH), and growth factors (IGF-I, IGF-II, IGF-ß)[51,52]. Of these, GH and insulin-like growth factor-I (IGF-I) are the most important ones. Growth hormone stimulates osteoblastic

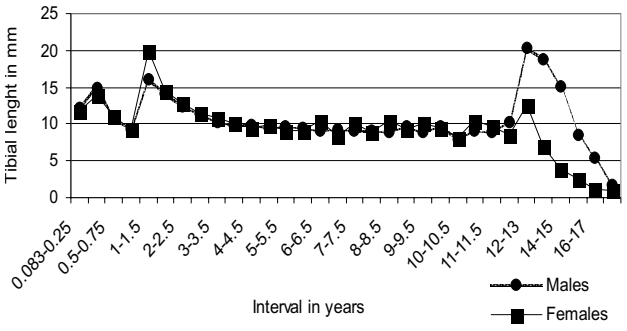

Fig. 1. Mean interval increments in millimeters of tibial length in males and females age one month through 18 years (data from Gindhart[44]).

activity and expands the pool of progenitor chondrocytes[11,53–56]. Growth hormone receptor (GHR) stimulates IGF-I expression[57] and promotes bone formation[58]. However, insulin-like growth factor-I and II (IGF-I and -II) decrease GHR secretion, suggesting the existence of a feedback mechanism in the GH/IGF axis at the local tissue level[55]. IGF-I stimulates pre-osteoblasts mitosis, chondrocyte hypertrophy[59–61] and mineralization. All chondrocyte layers produce IGF-I, but the one produced by the hypertrophic chondrocyte zone targets osteoblasts and, thus, is involved in bone deposition[62].

During growth, any overproduction of GH circulating levels must be countered by a diminished production of IGF-I in order to avoid chondrocyte mitosis running out of control. Because these mechanisms do not necessarily act simultaneously, Harris lines may form as osteoblasts continue to deposit bone, while cartilage growth has stopped or slowed[12]. Thus, it is possible that Harris lines are a normal outcome of the growth process, since growth is characterized by »stops and starts«[25–29].

Structurally, Harris lines are strata of denser, thicker, and transversely oriented bony trabeculae, that contrast with the normal composition and structure of bone. The lines are radiographically visible due to the increased mineralization that results from the irregular mineral deposition in the trabeculae (5–15% above normal)[2,7,14,16,20,22,23,47]. Yet it is possible for Harris lines to disappear as a result of subperiosteal apposition and endosteal surface resorption[3,14] which creates problems in the interpretation of these lines. Indeed, severe inconsistencies in both the sectional and radiographic records and the morbidity index values have been found and seem to be the result of the natural resorption of the lines[63].

Dental enamel deposition and enamel hypoplasia

Enamel forms through appositional growth that results in the regular deposition of layers. Since the deposition of these layers is constant, their chronology can be determined. As teeth form by continuous incremental growth, any metabolic disturbance, to which the ameloblasts are sensitive, will result in the formation of an

abnormal tissue layer[39]. Thus, developmental enamel defects, including enamel hypoplasias, are markers of childhood morbidity that can be timed in terms of the age of the individual at the time of their occurence[65–70], although some studies have contended the possibility of timing these events[71].

Deciduous enamel formation is largely an intrauterine process that continues for several months after birth[34,70], whereas the dentinogenesis of permanent teeth does not start until after birth[72]. Enamel hypoplasias, then, register developmental alterations that occurred between the 5th month *in utero* and up to 15 years of age. However, they are rarely identifiable after the age of 7, since all crown development, except for the 3rd molar, has been completed by that age[73,74]. Because enamel is not subject to remodeling, defects in dental formation remain throughout the life of the tooth, which contrasts with the resorptive process that affects Harris lines all through the life of the individual.

Sample

The sample analyzed came from two archaeological sites located in Northern Chile; Azapa-71 (AZ-71; juveniles) and Azapa-140 (AZ-140; juveniles and adults; Figure 2). A large sample of juveniles was necessary since Harris lines may disappear due to resorption; a process to which individuals who died before adulthood was reached were exposed for a shorter period of time. Thus, the inclusion of a large number of juveniles was considered necessary in order to increase the probability of identifying any association between these two markers.

Az-71 and Az-140 groups inhabited the Azapa valley during two archaeological periods: 1) Formative period (1700 B.C.–400 A.D.); and, 2) Middle Horizon (400–1000 A.D.) respectively. Both samples correspond to individuals from settled, agricultural communities, with similar diets, and economic activities[75–83].

Fig. 2. Map with the location of the Azapa Valley, Northern Chile.

TABLE 1
DESCRIPTION OF THE SAMPLE

Age/Sex	Undetermined	Female	Male	Total
0–3.0	28			28
3.1–7.0	18			18
7.1–12.0	14			14
12.1–19.0	27			27
19.1–30.0		11	6	17
30.1–40.0		9	7	16
>40		12	4	16
Total	87	32	17	136

Individuals included in this study have: 1) at least one suitable tooth for the analysis of enamel hypoplasia. Suitable teeth are those with an attrition lower or equal to 2°[84]. Teeth with a higher degree of attrition were included only if they presented an enamel defect; and, 2, at least one complete tibia. A total of 136 individuals fulfilled the criteria of this study (Table 1).

Adult individuals were sexed considering the morphology of the: 1) ventral arc; 2) subpubic concavity; 3) ischiopubic ramus ridge; 4) greater sciatic notch; 5) nuchal crest; 6) mastoid process; 7) supra-orbital margin; 8) glabella; and, 9) mental eminence[85, 86]. No juvenile individuals were sexed since most methods show a low degree of accuracy, at least in these groups[87].

Adult individuals were aged according to the morphology of the: 1) pubic symphysis, which was analyzed with two methods[88–90]; 2) auricular surface[91]; and 3) lateral-anterior cranial sutures[92]. Subadult individuals were aged considering: 1) dental maturation[93]; 2) presence of the primary ossification centers[94]; and, 3) union of epiphyses[93,95–97].

Methods

Harris lines were studied using radiographs from the tibia (preferentially the left one) taken at the bio-archaeology laboratory in the »Museo San Miguel de Azapa,« University of Tarapacá, Chile. The tibia was selected for the identification of Harris lines because: 1) it has proven to be one of the most reliable bones for the detection of Harris lines; 2) it shows minimal fading of Harris lines[14,15]; 3) it is the most commonly used bone, which allows for comparisons with other studies[16,63,98–102]; and 3) it presents nearly horizontal non-convoluted epiphyses whose shape do not significantly distort the geometry of a transverse line in relation to the plane of the X-ray.

If both tibiae were present, only the left one was considered, since it has been shown to present a greater number of lines, in comparison with the right one[103,104]. When the left tibia was absent the right one was radio-

graphed; thus, over 83% of the radiographs taken correspond to left tibiae, while the remaining 16% were taken from the right tibiae.

The radiographs were obtained using 85–90 volt and 2–3 mA settings. Adult tibiae were exposed for 3–4 seconds, while in juveniles the exposure time varied between 2–3 seconds. All tibiae were radiographed in antero-posterior view. Tibial growth, in this study, is described in percentages, so no correction was necessary for the small distortion due to focal-film distance. These percentages were calculated considering average tibial length at each age in relation to average adult tibial length (length at age 16–19).

A radiopaque line in the radiograph was recorded as a Harris line only when it covered at least 30% of the shaft width, and when its angle was greater than 45° and less than 135°[103]. Both the proximal and distal length were calculated considering that 43% of the diaphyseal growth occurs towards distal.

Several methods have been developed for the estimation of the age of the individual at the time of Harris line formation using the tibia[99,101,105]. The methods utilized in this study were Byers'[106] for adults, and Hummert and Van Gerven's[16] for both adults and juveniles. For the application of both methods, tibial length was directly measured from the radiographs.

Byers' method[106] was chosen because it offers a series of advantages including: 1) a table of percentage of growth per year; 2) it does not assume an average tibial length at birth; 3) it differentiates between males and females; 4) it considers the different rates of growth for proximal (57%) and distal (43%) segments; and, 5) it is case specific since it considers each tibia's length in the calculation of the individual's age at the time the line was deposited.

The age of the individual at the time of line formation using the Byers' method[106] was calculated by: 1) measuring the total length of the tibia in the radiograph; 2) identifying the lines as proximal or distal according to their location in the diaphysis; 3) calculating the distance between the radiopaque line and the epiphyseal end (proximal or distal), to which the identified line is most closely located; 4) calculating the percent of bone formed at the time the line was deposited (Pct; see Table 2); and, 5) comparing the results obtained for Pct with the chronology of tibial growth for males and females as described by Byers[106] (Figure 3).

However, Byers' method[106] cannot be applied to juveniles because their epiphyses are not yet fused to the diaphysis. Hummert and Van Gerven[16] developed a method than can be applied to both adults and juveniles. This is highly advantageous considering that Harris lines are subject to resorption, so that if only adults are analyzed an absence of association between Harris lines and enamel hypoplasia could potentially be the result of the resorptive process.

To apply Hummert and Van Gerven's method[16] the developmental age of the subadults was determined by estimating the degree of dental maturity[93]. Hummert and Van Gerven's[16] method can also be applied to adults; in their case the total diaphyseal length is estimated by subtracting the length of the epiphyses from the total length of the tibia. Hummert and Van Gerven's method[16] can be divided in two steps: 1) determination of the tibial growth pattern for the sample under study; and, 2) estimation of the age of the individual at the time of the line formation. The tibial growth pattern was established by: 1) calculating the total diaphyseal length; 2) determining the location of the primary center of ossification, considering that 43% of the tibia's shaft growth is distal; 2) calculating the percentage of distal (43%) and proximal (57%) growth increment, in mm, for each age; 3) converting the obtained values into percentages of annual growth for each age. These percentages were calculated by dividing the average length

TABLE 2
FORMULA FOR CALCULATING PERCENTAGE OF BONE LENGTH (PCT) AT THE TIME OF RADIOPAQUE LINE FORMATION (AFTER BYERS[106])

Bone	Harris line closest to	Formulae
Tibia	Proximal	Pct =1.15 (T–1.75P) x 100/T
Tibia	Distal	Pct= 1.15 (T–2.33D) x 100/T

T – total length of the tibia

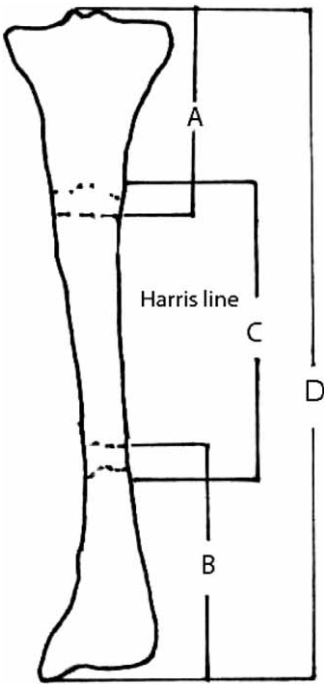

Fig. 3. Diagram illustrating the measurements taken when Byers'[106] method was applied. A – distance between Harris line and proximal epiphyseal end. B – distance between Harris line and distal epiphyseal end. C – length of the diaphysis at the time of Harris line deposition. D – total tibial length as measured in the radiographs.

TABLE 3
CHRONOLOGY OF PROXIMAL AND DISTAL TIBIAL GROWTH AS MEASURED IN THE RADIOGRAPHS IN PERCENTAGES

	UB	Birth	6m	9m	1	1.5	2	3	4	5	6	8	9	10	12	14	15	16–19
UB	100	90.8	82.6	75.1	65.8	62.0	51.0	41.9	40.0	36.1	31.9	29.9	27.4	24.9	24.8	24.7	21.9	21.2
Birth		100	90.9	82.7	72.5	68.3	56.2	46.2	44.0	39.8	35.2	32.9	30.2	27.4	27.3	27.3	24.2	23.3
6m			100	91.0	79.8	75.1	61.8	50.8	48.4	43.7	38.7	36.2	33.2	30.1	30.0	30.0	26.6	25.6
9m				100	87.7	82.6	67.9	55.8	53.2	48.1	42.5	39.8	36.5	33.1	33.0	32.9	29.2	28.2
1					100	94.2	77.4	63.7	60.7	54.8	48.5	45.4	41.6	37.8	37.6	37.6	33.3	32.1
1.5						100	82.3	67.6	64.5	58.2	51.5	48.2	44.2	40.1	40.0	39.9	35.4	34.1
2							100	82.3	78.4	70.8	62.6	58.6	53.7	48.8	48.6	48.5	43.1	41.5
3								100	95.3	86.1	76.1	71.2	65.3	59.3	59.1	59.0	52.4	50.5
4									100	90.3	79.9	74.8	68.5	62.3	62.0	61.9	54.9	52.9
5										100	88.4	82.7	75.8	68.9	68.7	68.5	60.8	58.6
6											100	93.6	85.8	77.9	77.7	77.5	68.8	66.3
8												100	91.7	83.3	83.0	82.8	73.5	70.8
9													100	90.9	90.5	90.3	80.2	77.3
10														100	99.6	99.4	88.2	85.1
12															100	99.8	88.6	85.4
14																100	88.7	85.5
15																	100	96.4
16–19																		100

UB – unborn

of the shaft (distal or proximal) at a specific age (e.g. 6 years) by the length of the shaft (distal or proximal) at age 16–19; and 4) multiplying the results by 100.

Eighty-seven tibiae from different individuals of these two groups, ranging between unborn and 19 years of age were used to construct a table for tibial growth (Table 3). In some of these individuals no teeth suitable for enamel hypoplasia analysis were available; those cases were not included in the analysis of Harris lines and enamel hypoplasias. However, they were utilized in the estimation of tibial growth so that this information would be as complete as possible. The sample, nevertheless, was missing individuals of ages 7, 11 and 13, and thus those ages had to be omitted from Table 3. In order to construct this table, the diaphyseal length of the tibia was measured directly from the radiographs.

Estimation of age of the individual at the time of Harris line formation was calculated by: 1) measuring the distance between the primary ossification center and the transverse line; 2) estimating the percentage of growth in the shaft (distal or proximal) completed at the time of line deposition by dividing the distance between the line and the primary ossification center by the corresponding diaphyseal length of the tibia's shaft segment (distal or proximal depending on the location of the line); and 3) comparing the results obtained with the chronology of tibial growth obtained for this sample (Table 3, Figure 4).

The method, when applied to adults, requires the subtraction of the epiphyseal contribution to the total tibial length, and the following calculation of the location of the primary ossification center in the tibia. As with subadults, all measurements of lines are made from this point. Hummert and Van Gerven[16] applied their method to the distal end of the tibia only. In this study, however, radiographs showed that some individuals, especially juveniles, have Harris lines either only, or

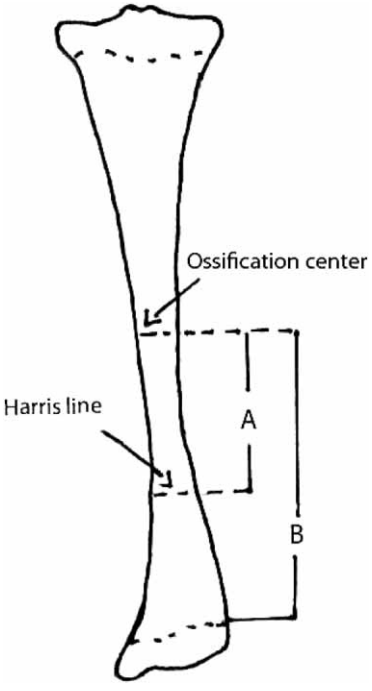

Fig. 4. Diagram illustrating the measurements taken when Hummert and Van Gerven's[16] method was applied. A – distance between Harris line and ossification center. B – total distal length of the diaphysis.

TABLE 4
GOODMAN AND ROSE MODIFIED FORMULAS FOR PERMANENT DENTITION

	Crown height		Developmental age in years			Regression equation		
	X	SD	N	At cusp	At CEJ	Duration	Average	Equation
Maxilla								
I1	11.209	0.675	18	1.0	4.5	3.5	0.312	=–(Ht*0.312)+4.5
I2	9.729	0.764	18	2.0	4.5	2.5	0.257	=–(Ht*0.257)+4.5
C	10.972	1.062	17	1.0	6.5	5.5	0.501	=–(Ht*0.501)+6.5
PM1	8.197	0.863	33	3.0	6.0	3.0	0.366	=–(Ht*0.366)+6.0
PM2	7.174	0.819	34	3.5	6.0	2.5	0.348	=–(Ht*0.348)+6.0
M1	7.439	0.579	21	1.0	3.5	2.5	0.336	=–(Ht*0.336)+3.5
M2	7.452	0.570	32	4.0	7.5	3.5	0.470	=–(Ht*0.470)+7.5
M3	6.258	0.549	21					
Mandible								
I1	9.417	0.568	16	1.0	4.0	3.0	0.319	=–(Ht*0.319)+4.0
I2	9.660	0.527	20	1.0	4.0	3.0	0.311	=–(Ht*0.311)+4.0
C	11.367	1.307	16	1.5	4.5	3.0	0.264	=–(Ht*0.2.64)+4.5
PM1	8.326	0.979	31	2.0	6.0	4.0	0.480	=–(Ht*0.480)+6.0
PM2	7.290	0.791	28	3,0	7.0	4.0	0.549	=–(Ht*0.549)+7.0
M1	7.826	0.516	14	1,0	3.5	2.5	0.319	=–(Ht*0.319)+3.5
M2	7.472	0.700	18	4,0	7.0	3.0	0.401	=–(Ht*0.401)+7.0
M3	6.451	0.755	15					

At Cusp – beginning of enamel development, At CEJ – time at which enamel deposition is completed (cement-enamel-junction formation), Ht – distance between the enamel defect and the cement-enamel junction

mostly, on the proximal end, and thus the analysis included both the proximal and distal segments of the tibia.

For enamel hypoplasia, each tooth in each individual's dentition was examined. A total of 2,467 teeth were analyzed; of these 650 (26.3%) were deciduous, while the remaining 1,817 (73.7%) were permanent teeth. The hypoplastic defects were identified and the distance between the defect and the cement-enamel junction (CEJ) measured. A mean crown height was established for each tooth type, for both deciduous and permanent teeth. These averages were calculated by measuring teeth that did not present attrition[84]. These measurements were taken following Rogers's method[107]. The average crown height was then divided into the numbers of months (deciduous teeth) or years (permanent teeth) that it takes for the crown to develop (see Tables 4 and 5). These calculations were necessary for the estimation of age of the individuals at the time of enamel hypoplasia formation.

The regression equations of Goodman and Rose[5] were applied to permanent teeth. However, considering that variation in crown height may impact the estimation of age at formation of enamel hypoplasia[108] the Goodman and Rose' method[5] was modified, following Wright's[8] recommendations, for the average crown height per tooth-type in the sample. Therefore, a second set of equations was applied to enamel hypoplasias present in permanent teeth. These linear regression equations were calculated assuming a constant rate of enamel growth[5,8]. This method will be referred to as the »Modified Goodman and Rose method« (Table 4). In deciduous teeth, the method of Blakey and Armelagos'[66] was applied (Table 5). This method considers that enamel in deciduous teeth is deposited between the 5th prenatal month and the 10–12th postnatal month, in a regular manner, so that each transverse segment of enamel represents a specific period in the development of the individual[66]. To apply this method an average tooth crown height for each tooth type was estimated in suitable deciduous teeth, and the formulas adjusted to these values (Table 5). Suitable deciduous teeth were those which degree of wear was $\leq 2^{\circ}$[84].

The age at formation of both Harris lines and enamel hypoplasias is expressed in years. The first year of life (<1), referred to here as »during the first year«, was described separately since at this time humans show such a rapid growth rate[33], that this growth period needs to be differentiated from subsequent years. Since most enamel hypoplasias are formed after birth, the few that were formed during the last trimester *in utero* were collapsed in this category (during the first year of age; <1).

Statistical analyses

Frequencies of enamel hypoplasia and Harris lines were calculated in order to determine the degree of association (χ^2) between these two indicators. This association was first estimated in terms of presence/absence of the two markers in each individual, regardless of the age at which Harris lines and/or enamel hypoplasia occurred (Table 6). In addition, a loglinear model[109] was

TABLE 5
BLAKEY AND ARMELAGOS[66], DECIDUOUS DENTITION ADJUSTED FORMULAS

	Crown height		Developmental age (in months)			Regression equation		
	X	SD	N	At cusp (prenatal)	At CEJ (postnatal)	Duration	Average	Equation
Maxilla								
i1	6.630	1.889	14	5	4	9	0.737	–(Ht/0.737)+4
i2	5.828	0.392	14	5	5	10	0.583	–(Ht/0.583)+5
c	6.661	0.595	15	6	9	13	0.512	–(Ht/0.512)+9
m1	5.837	0.361	31	5	6	11	0.531	–(Ht/0.531)+6
m2	6.404	0.500	24	6	12	15	0.427	–(Ht/0.427)+12
Mandible								
i1	5.624	1.052	17	5	4	9	0.625	–(Ht/0.625)+4
i2	5.941	0.332	18	5	5	10	0.594	–(Ht/0.594)+5
c	6.862	0.558	22	6	9	13	0.528	–(Ht/0.528)+9
m1	6.732	0.422	30	5	6	11	0.612	–(Ht/0.612)+6
m2	6.578	0.591	19	6	12	15	0.439	–(Ht/0.439)+12

At Cusp – beginning of enamel development, At CEJ – time at which enamel deposition is completed (cement-enamel-junction formation), Ht – distance between the enamel defect and the cement-enamel junction

TABLE 6
PREVALENCE OF HARRIS LINES AND ENAMEL HYPOPLASIA AMONG THE INDIVIDUALS ANALYZED

	Harris lines									Enamel hypoplasia								
	Undetermined			Female			Male			Undetermined			Female			Male		
Age at death	n1	N	%	n1	N	%	n1	N	%	n2	N	%	n2	N	%	n2	N	%
0–3.0	18	28	64.3							12	28	42.9						
3.1–7.0	16	18	88.9							12	18	66.7						
7.1–12.0	10	14	71.4							13	14	92.9						
12.1–19.0	15	27	55.6							26	27	96.3						
19.1–30.0				8	11	72.7	5	6	83.3				10	11	90.9	5	6	83.3
30.1–40.0				5	9	55.6	3	7	42.9				9	9	100.0	7	7	100.0
>40				7	12	58.3	2	4	50.0				10	12	83.3	4	4	100.0
Total	59	87	67.8	20	32	62.5	10	17	58.8	63	87	72.4	29	32	90.6	16	17	94.1

n1 – number of individuals with at least one Harris lines, n2 – number of individuals with at least one hypoplastic defect

applied in order to assess the possible association between age, enamel hypoplasia, and Harris lines.

The ages of the individuals at the time of enamel hypoplasia and Harris line formation were calculated and the results, expressed in one-year range, were then compared. The ages at which enamel hypoplasia formation occurred in deciduous teeth, obtained with the Blakey and Armelagos[66] method, were first combined with the results obtained from permanent teeth with the Goodman and Rose's method[5], and in a second instance with the results obtained with the Modified Goodman and Rose method. Therefore, the results obtained in deciduous and permanent teeth are presented together. The results for enamel hypoplasia were combined in this manner because the Blakey and Armelagos[66] method can only be used in deciduous teeth, while the Goodman and Rose's[5] and the Modified Goodman and Rose methods are exclusively applicable to permanent teeth. Thus, the results obtained with these methods account for different (although sometimes overlapping) periods in the life cycle of an individual, whereas the tibia, used to examine Harris lines formation, accounts for the entire period of growth and development.

Since Harris lines and enamel hypoplasia have a different time span during which they can be formed, later comparisons between the two markers were limited to Harris lines and enamel hypoplasia formed between ages 1–7 (Tables 10–17). In order to avoid bias due to inter-tooth differential sensitivity and intra-tooth differential visibility[41], the average number of enamel hypoplasias formed at each year of age, in the individuals examined, was subtracted from the total number of enamel hypoplasias formed at the corresponding age in each individual (Table 15). Thus, the term »remaining enamel hypoplasias« refers to the number of enamel defects left after the average number of enamel hypoplasias formed per year was subtracted (Table 15).

TABLE 7
CHI-SQUARE VALUES FOR ENAMEL HYPOPLASIA/HARRIS LINES PRESENCE/ABSENCE AMONG THE INDIVIDUALS ANALYZED

Age at death	EH–/HL–	EH+/HL–	EH–/HL+	EH+/HL+	χ^2	df	p
General	12	37	16	71	0.71	1	1
0–3.0	8	2	8	10	3.31	1	0.1
3.1–7.0	0	2	6	10	1.125	1	0.1
7.1–12.0	1	3	0	10	2.69	1	0.2
12.1–19.0	0	12	1	14	3.84	1	1
19.1–30.0 F	1	2	0	8	2.93	1	0.1
19.1–30.0 M	0	1	1	4	0.24	1	1
30.1–40.0 F	0	4	0	5	–	–	–
30.1–40.0 M	0	4	0	3	–	–	–
>40 F	2	5	0	5	1.71	1	0.20
>40 M	0	2	0	2	–	–	–

EH– – enamel hypoplasia absent, HL– – Harris lines absent, EH+ – enamel hypoplasia present, HL+ – Harris lines present, DF – degrees of freedom, F – female, M – male

Likewise, due to the possibility that some Harris lines are the result of growth instead of stress, the average number of Harris lines formed at each year of age by the individuals examined, was subtracted from the number of Harris lines observed in each individual at the corresponding age. The resulting number of Harris lines is termed »remaining Harris lines« (Table 16).

Three correction factors (0.1, 0.2, 0.3) were added to the remaining Harris lines. The value of these factors was determined considering that 0.3 is estimated as the maximum number that can be added to a result without fundamentally altering it. The correction factors were added in order to increase the likelihood that Harris lines would match with enamel hypoplasias. These factors were added, exclusively, to individuals that presented both Harris lines and enamel hypoplasias that were formed between ages 1–7 years.

The remaining Harris lines, once the correction factors were added, were matched each time with the remaining enamel hypoplasia. Remaining Harris lines were compared with remaining enamel hypoplasias but not reverse, due to the possibility that the absence of Harris lines was a consequence of the resorption process. The percentages of matches and disagreements were then compared.

The rather meticulous methodological process, described above, was performed in order to determine, with the greatest possible accuracy, any association between Harris lines and enamel hypoplasias, and to account for any variables that might mask such association.

Results and Discussion

Of the 136 individuals, a total of 86 (63.2%) had Harris lines. The analysis showed that 67.8% of the juveniles, 62.5 % females, and 58.8% males had Harris lines (Table 6). Likewise, 79.4% of the sample had at least one tooth with a hypoplastic defect. Indeed, 90.6% of females, 94.1% males, and 72.4% of juveniles presented enamel hypoplasia (Table 6).

When the entire sample is considered there is no identifiable association between these two indicators of stress (χ^2= 0.71; p(a)= 1; Table 7). Similarly, when the sample is divided according to the age-sex categories, none of them showed any significant association between enamel hypoplasias and Harris lines (Table 7). The highest degree of association was found in the age segments 0–3.0, 3.1–7.0, and 19.1–30.0 females. All of them show χ^2 values (p= 0.1; Table 7), which indicate that there is some association between these two markers, although not a significant one.

A loglinear model was used to assess the association between age, enamel hypoplasia, and Harris lines. None of these factors caused significant deviation from the expected frequencies, indicating that these three factors are not associated (likelihood ratio: ns).

A total of 263 Harris lines were identified; 31.9% located in the proximal half and 68.1% in the distal half of the tibia. Byers'[106] method is only applicable to individuals with fused epiphyses. Thus, 37 individuals were analyzed with this method, while the Hummert and Van Gerven's method[16] was applied to the entire sample.

Among the 37 individuals with fused epiphyses 81 lines were identified. Using the Byers'[106] method, the results show that the highest peak for age at the time of Harris line formation in males (28.0%) and females (16.1%) occurs during the first year of age (<1; Table 8). A second peak can be identified, for both males and females, at the time of adolescence. In females, this second peak in frequency takes place at the age of 11 (14.3%), while in males it takes place at the age of 13 (12.0%; Table 8).

Using the Hummert and Van Gerven's[16] method, the greatest frequency of Harris lines occurs at the age of 2 years (30.8%; Table 9). This distribution is skewed due to the inclusion of juveniles in the sample. Since these individuals died prematurely, their remains only account for the earliest years of development. Among adult individuals Harris lines present three main peaks in frequency; the first peak occurs during the first year

TABLE 8
AGE AT THE TIME OF HARRIS LINE FORMATION

Age	Byers'[106] method						Hummert-Van Gerven's[16] method					
	Female		Male		Total		Female		Male		Total	
	N	%	n	%	N	%	N	%	N	%	N	%
<1	9	16.1	7	28.0	16	19.8	7	16.3	2	11.8	9	15.0
1	5	8.9	1	4.0	4	4.9	1	2.3	3	17.7	4	6.7
2	4	7.1	0	0.0	4	4.9	3	6.9	3	17.7	6	10.0
3	4	7.1	4	16.0	6	7.4	2	4.7	0	0.0	2	3.3
4	3	5.4	1	4.0	4	4.9	0	0.0	0	0.0	0	0.0
5	2	3.6	0	0.0	2	2.5	2	4.7	2	11.8	4	6.7
6	3	5.4	2	8.0	4	4.9	2	4.7	2	11.8	4	6.7
7	2	3.6	1	4.0	3	3.7	0	0.0	0	0.0	0	0.0
8	3	5.4	1	4.0	4	4.9	3	6.9	1	5.9	4	6.7
9	5	8.9	2	8.0	7	8.6	7	16.3	2	11.8	9	15.0
10	4	7.1	1	4.0	8	9.9	2	4.7	0	0.0	2	3.3
11	8	14.3	0	0.0	12	14.8	0	0.0	0	0.0	0	0.0
12	1	1.8	1	4.0	3	3.7	0	0.0	0	0.0	0	0.0
13	2	3.6	3	12.0	3	3.7	0	0.0	0	0.0	0	0.0
14	1	1.8	1	4.0	1	1.2	3	6.9	1	5.9	4	6.7
15	0	0.0	0	0.0	0	0.0	11	25.6	1	5.9	12	20.0
16	0	0.0	0	0.0	0	0.00	0	0.00	0	0.0	0	0.0
17	0	0.0	0	0.0	0	0.00	0	0.00	0	0.0	0	0.0
Total	56	100	25	100	81	100.00	43	100.00	17	100	60	100

Byers'[106] method – can only be used in individuals with fused epiphyses

of age (<1; 15%), the second at age 9 (15%), and the third at age 15 (20%; Table 8). Notice that no lines were identified as formed at ages 7, 11, or 13, with the Hummert and Van Gerven's[16] method; this is due to the fact that no individuals of those ages were available when the tibial growth table was constructed (Table 3).

A total of 1,026 hypoplastic defects were identified; 37 (3.6%) of them in deciduous teeth and 989 (96.4%) in permanent teeth. In terms of age at the time of enamel hypoplasia formation, the combined results obtained with the Blakely and Armelagos'[5] and Goodman and Rose's[5] methods showed a peak for enamel hypoplasia formation at the age of 3 in the permanent teeth of individuals aged between 7.1 and 30.0 years of age. For individuals whose age at death was above 30 years, the peak occurred between the ages of 3 and 5 (Table 10).

Results attained with the Modified Goodman and Rose method show a similar distribution compared to the ones obtained with the original method. Among juveniles the peak age for enamel hypoplasia formation was 3.0 years. Similarly, in adults, the results obtained with this method show the highest frequency of enamel hypoplasia at age 4 (Table 11).

Enamel hypoplasia does show a clear difference in frequency in deciduous as compared to permanent teeth, where deciduous teeth show not only a significantly lower percentage of teeth affected by enamel hypoplasia (4.9% vs. 34.8%), but also a significantly lower average number of defects per tooth affected (1.16 vs. 1.56; τ=5.9; df=18; $p<0.05$). Other studies have also shown a low frequency of enamel hypoplasia in deciduous teeth[110,111]. Enamel hypoplasia then, seems to be more common after the first year of life and not before. Al-

TABLE 9
AGE AT THE TIME OF HARRIS LINES FORMATION. ALL LINES CONSIDERED HUMMERT AND VAN GERVEN'S METHOD[16]

Age at time of HL deposition	N	%
<1	33	12.6
1	24	9.1
2	81	30.8
3	25	9.5
4	9	3.4
5	19	7.2
6	17	6.5
8	11	4.2
9	16	6.1
10	5	1.9
12	0	0.0
14	9	3.4
15	14	5.3
Total	263	100

N – number of Harris lines formed at that age

TABLE 10
AGE AT TIME OF ENAMEL HYPOPLASIA FORMATION: BLAKEY AND ARMELAGOS[66], GOODMAN AND ROSE[5] COMBINED

Age at death	0–3.0		3.1–7.0		7.1–12.0		12.1–19.0		19.1–30.0 Female		19.1–30.0 Male		30.1–40.0 Female		30.1–40.0 Male		>40 Female		>40 Male	
Age EH	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
<1	28	100.0	9	30.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
1	0	0.0	0	0.00	1	0.5	0	0.0	1	1.2	3	2.7	1	1.2	1	1.2	0	0.0	0	0.0
2	0	0.0	2	6.7	14	7.3	41	11.6	8	9.4	25	22.5	7	8.1	8	9.4	5	7.7	4	13.8
3	0	0.0	17	56.7	81	42.4	139	39.3	21	24.7	39	35.1	24	27.9	21	24.7	16	24.6	4	13.8
4	0	0.0	2	6.7	57	29.8	85	24.0	17	20.0	22	19.8	24	27.9	17	20.0	13	20.0	5	17.2
5	0	0.0	0	0.0	21	10.9	58	16.4	22	25.9	17	15.3	24	27.9	22	25.9	17	26.2	9	31.0
6	0	0.0	0	0.0	15	7.9	21	5.9	14	16.5	5	4.5	6	6.9	14	16.5	12	18.5	6	20.6
7	0	0.0	0	0.0	2	1.1	10	2.8	2	2.4	0	0.0	0	0.0	2	2.4	2	3.1	1	3.5
Total	28	100	30	100	191	100	354	100	85	100	111	100	86	100	85	100	65	100	29	100

Age EH – age at time of enamel hypoplasia formation

TABLE 11
AGE AT TIME OF ENAMEL HYPOPLASIA FORMATION: BLAKEY AND ARMELAGOS[66], MODIFIED GOODMAN AND ROSE COMBINED

Age at death	0–3.0 Undetermined		3.1–7.0 Undet		7.1–12.0 Undetermined		12.1–19.0 Undetermined		19.1–30.0 Female		19.1–30.0 Male		30.1–40.0 Female		30.1–40.0 Male		>40 Female		>40 Male	
Age EH	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
<1	28	100.0	9	30.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
1	0	0.0	0	0	1	0.5	0	0.0	1	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
2	0	0.0	2	6.7	14	7.3	41	11.6	11	9.9	3	3.5	4	4.7	2	5.4	2	3.1	2	6.9
3	0	0.0	17	56.7	81	42.4	139	39.3	38	34.3	21	24.4	21	24.7	6	16.2	20	30.8	6	20.7
4	0	0.0	2	6.7	57	29.8	85	24.0	36	32.4	27	31.4	28	32.9	17	45.9	25	38.5	3	10.3
5	0	0.0	0	0	21	10.9	58	16.4	20	18.0	26	30.2	14	16.5	9	24.3	7	10.8	7	24.1
6	0	0.0	0	0.0	15	7.9	21	5.9	6	5.4	9	10.5	15	17.7	2	5.4	9	13.9	9	31.0
7	0	0.0	0	0.0	2	1.1	10	2.8	0	0.0	0	0.0	3	3.5	1	2.7	2	3.1	2	6.9
Total	28	100	31	100	191	100	354	100	112	100	86	100	85	100	37	100	65	100.0	29	100

though this pattern is the one usually found, this distribution is exactly the opposite to the one observed with Harris lines, which were more frequently formed during the first year of age (<1; Tables 7–10). The difference, though, may be due to the fact that enamel defects form primarily between <1–7 years of age while Harris lines form between <1–16 years of age. Thus, the comparison between these two markers will provide misleading results if Harris lines formed after the age of 7 are included, given that teeth whose crowns are being formed at that time, are the least susceptible to this type of defect[72,73].

Since individuals who died at an early age did not live long enough to continue forming Harris lines or enamel hypoplasias, their inclusion in the analysis would result in skewed distributions for these markers[112]. Even individuals whose age at death range between 7 and 19 years of age must be excluded since any teeth affected by enamel hypoplasia might be unerupted and therefore unobservable. Thus, the subsequent analyses considered Harris lines and enamel hypoplasias only in adults. Harris lines formed during the 1st year of age were omitted since there would be no deciduous teeth available in an adult with which to make a comparison, and because enamel defects formed during the 1st year of life in permanent teeth can only be detected histologically[108]. Therefore, in the analysis of adults, only Harris lines formed between the ages of 1–7 years were considered. The results obtained with the Byers' method[106] show that most of the lines (58.3%) are formed at the age of 1 (Table 12). After the age of 1, line formation decreases markedly. Using the Hummert and Van Gerven's method[16], the results are slightly different and show that 60% of Harris lines were formed between the ages 1 and 2, after which age, Harris line formation decreases noticeably (Table 12).

When enamel hypoplastic defects are analyzed for the adult population only, the results contrast significantly with those found for Harris lines. When the Goodman and Rose's[5] method is considered, the majority of enamel defects (72.6%) cluster between ages 3–5 years (Table 13). The results obtained with the Modified Goodman and Rose method show that 80.2% of the defects formed between the ages 3–5 years (Table 13).

TABLE 12
HARRIS LINES FORMED BETWEEN AGES 1–7 IN ADULT INDIVIDUALS*

	Byers'[106] method						Hummert and Van Gerven's[16] method					
	Female		Male		Total		Female		Male		Total	
Age	N	%	N	%	N	%	N	%	N	%	N	%
1	13	86.7	1	11.1	14	58.3	2	11.8	4	30.8	6	20.0
2	1	6.7	0	0.0	1	4.2	7	41.2	5	38.5	12	40.0
3	0	0.0	4	44.4	4	16.7	2	11.8	0	0.0	2	6.7
4	0	0.0	1	11.1	1	4.2	1	5.9	0	0.0	1	3.3
5	0	0.0	0	0.0	0	0.0	3	17.7	2	15.4	5	16.7
6	1	6.7	2	22.2	3	12.5	2	11.8	2	15.4	4	13.3
7	0	0.0	1	11.1	1	4.2	–	–	–	–	–	–
Total	15	100	9	100	24	100	17	100	13	100	30	100

* The total number of Harris Lines considered with each method varies due to the fact that the total number of Harris lines formed prior to the age of 1, which were subtracted, varied with the method applied

TABLE 13
ENAMEL HYPOPLASIA IN ADULTS AT AGE OF FORMATION*

	Goodman and Rose[5]						Goodman and Rose modified					
	Female		Male		Total		Female		Male		Total	
Age	N	%	N	%	N	%	N	%	N	%	N	%
1	5	2.2	0	0.0	5	1.4	0	0.0	0	0.0	0	0.0
2	36	15.5	7	5.8	43	12.2	17	6.5	7	4.6	24	5.8
3	55	23.6	33	27.3	88	24.9	79	30.3	33	21.7	112	27.1
4	61	26.2	19	15.7	80	22.6	89	34.1	47	30.9	136	32.9
5	53	22.8	36	29.8	89	25.1	41	15.7	42	27.6	83	20.1
6	22	9.4	22	18.2	44	12.4	30	11.5	20	13.2	50	12.1
7	1	0.4	4	3.3	5	1.4	5	1.9	3	1.9	8	1.9
Total	233	100	121	100	354	100	261	100	152	100	413	100

*The total number of hypoplastic defects considered with each method varies due to the fact that the total number of defects formed during the age of 1 (<1) and after the age of 7, which are not considered here, differed according to the calculations of each method

These combined results show that, even if the sample is reduced to avoid the biases already identified, enamel defects and Harris lines show a very different distribution by age. In fact, the distribution of Harris lines (Tables 7, 8 and 11) is similar to the tibial growth pattern (Figure 1).

Considering these results, it is possible to question the status of Harris lines as a stress indicator, or even as evidence of arrested growth. If most Harris lines are the result of normal growth[12], but some Harris lines form as the result of stress, it becomes necessary to remove from consideration those lines that are the result of growth, and match the residual ones with enamel hypoplastic defects to see if they coincide. Since it is not possible to determine which lines are the result of stress and which the result of growth it is assumed that the average number of Harris lines formed at each year of life is the result of normal growth. This specific number of lines, therefore, should not be considered in the analysis of association between Harris lines and enamel hypoplasias when these two markers are being evaluated as indicators of stress. With this in mind, the specific average number of Harris lines (Table 14) formed at each age was subtracted from the corresponding number of Harris lines formed by each individual at each age. The result obtained after this subtraction is termed »remaining Harris lines«.

To further complicate things, enamel defect distribution may be the result of a higher sensitivity and higher visibility of defects formed at a specific age due to crown morphology, rather than the sole product of stress[68]. In order to avoid this problem and considering that both sensitivity and visibility of enamel hypoplasia may be specific to each year of life, it was assumed that the average number of enamel hypoplasias formed during each year of life in the individuals analyzed, is the result of specific stress sensitivity/visibility at that age. Therefore, the age-specific average of enamel hypoplasias was subtracted from the total number of enamel defects formed at each specific year of age by each individual

TABLE 14
AVERAGE NUMBER OF HARRIS LINES BY AGE

	Byer's[106] method			Hummert and Van Gerven's[16] method		
Age	NHL	N individuals	F (HL-By)	NHL	N individuals	F (HL-HVg)
1	4	4	1.0	33	26	1.3
2	4	4	1.0	24	21	1.1
3	6	5	1.5	78	39	2.0
4	4	3	1.3	75	17	4.4
5	2	2	1	9	6	1.5
6	4	4	1	19	12	1.6
7	3	3	1	16	10	1.6

NHL – number of Harris lines, F(HL-By) – average number of Harris lines per year of age obtained with the Byers'[106] method, F(HL-HVg) – average number of Harris lines per year of age obtained with the Hummert and Van Gerven's[16] method, N – individuals. Notice that the total number of individuals for both methods varies because Byers'[106] can only be applied to adults, while Hummert and Van Gerven's [16] can be applied to both adults and subadults. In addition, due to the fact that both methods give different estimations for age of the individuals at the time of Harris line formation, the number of lines (NHL) varied

(Table 15). The resulting number of enamel hypoplasias is termed »remaining enamel hypoplasias«.

The remaining Harris lines were then assessed against the remaining enamel hypoplasias. Only presence/absence of Harris lines and enamel hypoplasia was analyzed in the matching process, and not the total number of lines or defects present. Therefore, the number of »possible matches« is lower than the number of remaining lines because an individual may have formed more than one line at a certain age, but all those lines would account for only one »possible match«.

Harris lines were matched with enamel hypoplasia but not the other way around, because the number of enamel defects is larger than the number of Harris lines. Thus, by comparing Harris lines with enamel hypoplasia the probability of finding a match was increased. In addition, this avoided bias due to the possibility that the absence of Harris lines was a result of resorption. The percentages of matches and disagreements were then compared.

Although 70 individuals present both Harris lines and enamel hypoplasias, the year-by-year analysis showed that 7 of them did not have Harris lines formed within the range of 1–7 years of age. Thus, only 63 individuals were suitable for this particular analysis.

Several correction factors (0.1–0.3) were added to the remaining Harris lines. This step was included to increase the possibility of finding matches between these two markers. In other words, we would rather err on the side of caution than risk spurious results. These correction factors are somewhat arbitrary because it is impossible to determine, with the data available, whether or not the average number of Harris lines subtracted contained any lines that were the result of stress. The results obtained show that with or without the correction factors the majority of Harris lines disappeared (100%–79.3%; Table 16).

The remaining lines were then matched with the remaining enamel hypoplasias. Since after the subtractions all Harris lines of the Byer's[106] method disappeared, the following analyzes were only applicable to the results obtained with the Hummert and Van Gerven's[16] method. Matches are defined here as those cases in which Harris lines and enamel hypoplasias coincide in each specific individual, in terms of age at the time of formation. The comparison did not consider the cases in which Harris lines were absent and enamel hypoplasias present because the absence of Harris lines might be the result of resorption. If a significantly large number of Harris lines resulted from stress and not from growth, we should expect to find a greater proportion of matches than mismatches. The numbers of possible matches along with the number of matches found were compared for the Hummert and Van Gerven's[16] method used in Harris lines, and for all methods used in enamel hypoplasia (Table 17).

TABLE 15
AVERAGE NUMBER OF ENAMEL HYPOPLASIA BY YEAR

	Goodman and Rose's (1990) method			Goodman and Rose modified method		
Age	NEH	N individuals	F (EH-GR)	NEH	N individuals	F (EH-GRmod)
1	31	23	1.4	1	1	1.0
2	157	49	3.2	81	36	2.3
3	306	68	4.5	351	73	4.8
4	203	63	3.2	281	75	3.8
5	182	62	2.9	162	55	2.9
6	84	38	2.2	86	34	2.5
7	13	8	1.6	20	13	1.5

NEH – number of enamel hypoplasias, F(EH-GR) – average number of Enamel hypoplasia per year of age calculated with the Goodman and Rose (1990) method, F(EH-GRmod) – average number of enamel hypoplasia per year of age calculated with the Goodman and Rose Modified method

TABLE 16
REMAINING HARRIS LINES AFTER SUBTRACTION OF THE AVERAGE NUMBER OF HARRIS LINES AND ENAMEL HYPOPLASIA

μ_s	HL	HL-F (HL-By)	% Eliminated lines	HL	HL-F (HL-HVg)	% Eliminated lines
0.0	23	0	100	116	22	81.0
0.1	23	0	100	116	24	79.3
0.2	23	0	100	116	24	79.3
0.3	23	0	100	116	24	79.3

μ – correction factors added to the remaining Harris lines, HL-F(HL-By) – total number of Harris lines minus the average number of Harris lines formed at each specific age obtained by using the Byer[106] method, HL-F(HL-HVg) – total number of Harris lines minus the average number of Harris lines at each specific obtained by using the Hummert and Van Gerven's[16] method

The results show that the number of matches are much less than the number of mismatches (Table 17), regardless of what method is used or what correction factor added. This is interesting because, if there is a direct relationship between the formation of enamel hypoplasias and Harris lines we would expect a higher frequency of matches. Since this is not the case, we suspect that the matches identified may be the result of chance alone. In other words, there appears to be little evidence to indicate a direct relationship between the formation of enamel hypoplasias and Harris lines.

Overview

The results obtained in this study showed no association between Harris lines and enamel hypoplasia. This has been noted in other studies[100,113–115]. Our analysis of the presence/absence of these indicators showed no association when the sample was considered as a whole or when each age-sex group was considered individually. χ^2 was not applicable to the 30.1–40.0 years old female and male segments, nor to the >40 male group (Table 7). However, this is not relevant since the results of this test showed no significant association between Harris lines and enamel hypoplasia among juveniles, who have the highest probability of presenting an association considering that the time of exposure to the resorptive process has been shorter than in adults. In addition, the application of a loglinear model showed that age, enamel hypoplasias and Harris lines are not statistically associated.

When the age of the individuals at the time of Harris line and enamel hypoplasia formation was analyzed, the two indicators behaved very differently. While Harris lines showed a high rate of formation during the first year of age (>1) and a second peak at adolescence, enamel hypoplasias showed their highest frequencies between the ages of 3 and 5. We acknowledge that this discrepancy between the two markers might have been the result of the differential range of years in which enamel hypoplasia (>1–7) and Harris lines (<1–16) can be formed, and the fact that almost 64% of the sample was composed of juveniles, which might have skewed the distribution of Harris lines towards younger ages. The use of Harris lines formed during the first year of age might be questioned because enamel hypoplasias formed at this time rarely appear in permanent teeth. However, the fact that there is a very low percentage of deciduous teeth with enamel defects (4.9%), and that the few deciduous teeth that present them show a low average number of defects per affected tooth (1.2) cannot be overlooked. The results obtained showed that while Harris lines were formed at a high rate during, and at, the first year of age, enamel hypoplasias were not. This, however, may be either the result of lower sensitivity in deciduous teeth to environmental factors, or it may be due to the possibility that individuals stressed during infancy did not survive long enough to erupt their secondary dentition[66].

When only Harris lines and enamel hypoplasias present in adults and formed between the ages 1–7 were considered, the distribution of the two indicators continued to be very different. Harris lines showed a high rate of formation at ages 1 and 2, and a very low frequency after that age. The distribution of enamel hypoplasias, in contrasts, shows that the majority of the enamel defects were formed between the ages of 3 and 5.

The results reveal that, regardless of the various ways the data were treated, Harris lines repeatedly showed a high peak during or at the first year of age.

TABLE 17
NUMBER AND PERCENTAGES OF MATCHES OBTAINED AFTER THE AVERAGE NUMBER OF HARRIS LINES AND ENAMEL HYPOPLASIA WERE SUBTRACTED AND THE DIFFERENT CORRECTION FACTORS WERE ADDED

μ	HVg-GRBA			HVg-Grmod		
	Number of possible matches	%Mat	% Mis	Number of possible matches	% Mat	% Mis
0.0	10	20.0	80.0	10	20.0	80.0
0.1	12	16.7	83.3	12	16.7	83.3
0.2	12	16.7	83.3	12	16.7	83.3
0.3	12	16.7	83.3	12	16.7	83.3

HVg – Hummert and Van Gerven's[16], GRBA – Goodman and Rose[5] and Blakely and Armelagos[66], Grmod – Modified Goodman and Rose, μ – correction factors corresponding to the possible number of HL formed each year due to stress. These factors were added to the remaining HL, % Mat – % of matches, % Mis – % of mismatches

Moreover, Harris lines are very common during the first year of age and not as common at older ages. In contrast, enamel hypoplasias show the opposite pattern; they are rarely formed during the first year of age and appear more commonly after the age of 2.

It is known that *intra uterine* environment and lactation provide the fetus and the neonate with protection against multiple stressors[116–119]. Accordingly, the low frequency of enamel hypoplasias at this age has usually been attributed to the protection conferred by both the *uterine* environment and maternal milk[38,120–122].

Taking this into consideration, we should expect to find a low prevalence of stress markers during the first year of age, as shown by enamel hypoplasias in this study. The high incidence of Harris lines during age 1, however, does not fit this model. If Harris lines were an indicator of stress, we should expect to find a low frequency of Harris lines during the first year of age and a high incidence after this period. Moreover, if Harris lines are the consequence of stressors that results in slowed or stopped growth, we should expect to find them, not only in association with other stress markers (such as enamel hypoplasia), but also at times when growth is decelerated.

The results obtained here do not fit with any of these expectations. Harris lines are not associated with enamel hypoplasia. Instead, Harris lines show a distribution pattern that is similar to the growth curve of the tibia (see Figure 1[44]), which is analogous to the velocity growth curve in humans[33]. Other studies have had similar results, and this study provides additional support for a relationship between growth velocity and transverse line formation[12].

Since our results suggest the possibility that Harris lines are not necessarily pathological, it is possible that they might, instead, be a consequence of normal growth, especially since normal growth is characterized by the presence of stasis periods, or pauses in growth[25–29]. Hence, the interpretation of Harris lines as a stress indicator, as well as their usefulness in the reconstruction of past and present population's heath status needs to be reconsidered.

Finally, none of the growth factors and hormones that influence growth, per se, can explain the increased bone deposition, vascular invasion, and decreased mitosis of the progenitor chondrocytes associated with the appearance of Harris lines. Glucocorticoids (GC), however, might be involved in Harris line formation since they stimulate osteoblastic activity and the subsequent bone deposition. Although it is apparent that IGF-I and GH might mediate some of its actions, GC stimulates insensitivity to GH and IGF-I, and thus reduces their expression[56]. Secretion of GC may increase with stress, but studies in rats have shown no differences in GC levels among rats exposed to stress and non-stressed controls[59]. Although GC might be involved in the appearance of transverse lines by stimulating osteoblastic activity and reducing GH sensitivity, and thus mitosis in the reserve layer, this does not mean that GC is the only factor involved in the formation of Harris lines, especially considering that even in the presence of GC bone growth still occurs[123].

As long as bone growth at the endocrinological level is not fully understood, it will not be possible to determine the endocrinological pathway of Harris lines formation. The fact that Harris lines are more common during critical periods of growth[124] demands further examination. Although normal growth is characterized by periods of stasis, it is also known that growth events divert the normal physiology of an organism, at least momentarily. Growth then, may be a costly, although normal, biological perturbation for the organism[125] that may result in Harris lines formation.

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PREISPITIVANJE HARRISOVIH LINIJA – USPOREDBA HARRISOVIH LINIJA I HIPOPLAZIJE ENAMELA

S A Ž E T A K

Ova studija istražuje povezanost između Harrisovih linija i hipoplazije enamela. Ova je povezanost analizirana u odnosu na: 1) prisustvo/odsustvo ovih biljega u svake osobe, i 2) dobi osoba u vrijeme formacije Harrisovih linija i hipoplazije enamela. Analizirani su podaci dviju arheoloških skupina (Azapa-71 i Azapa-140) iz sjevernog Čilea. Ovi rezultati indiciraju da Harrisove linije nisu povezane s hipoplazijom enamela u smislu prisustva/odsustva. Štoviše, procijenjena dob osoba u vrijeme razvoja Harrisovih linija i hipoplazije enamela pokazuje da ova dva biljega imaju vrlo različitu raspodjelu. Dok hipoplazija enamela je grupirana u dobi od 3 do 5 godina, Harrisove linije se najčešće formiraju tijekom prve godine života te tijekom adolescencije, što su razdoblja najubrzanijeg rasta. Stoga smatramo da su Harrisove linije rezultat normalnog skokovitog procesa rasta.