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The Chronological Distribution of Enamel Hypoplasias From Prehistoric Dickson Mounds Populations

ALAN H. GOODMAN, GEORGE J. ARMELAGOS, AND JEROME C. ROSE
Department of Orthodontics University of Connecticut Health Center, Farmington, Connecticut 06032 (A.H.G.), Department of Anthropology, University of Massachusetts, Amherst, Massachusetts 01003 (G.J.A.), and Department of Anthropology, University of Arkansas, Fayetteville, Arkansas 72701 (J.C.R.)

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ABSTRACT The chronological distributions of enamel hypoplasias (indicators of nonspecific stress) are assessed for 111 individuals from two prehistoric populations from Dickson Mounds, Lewiston, Illinois. The earlier population (circa A.D. 950–1150) involves a transition from an indigenous gathering-hunting tradition to increasing adoption of Mississippian lifeways. The later population (circa A.D. 1150–1300) is fully Mississippian (MM). Based on the occurrence of hypoplasias on all permanent teeth except third molars, 14 half-year periods from birth to 7.0 years are graded for evidence of hypoplasia-stress. Both populations have a low frequency of hypoplasia which occur before 2 years of age and after 4 years of age. A common peak frequency of hypoplasias between 2.0 and 4.0 years is suggestive of an elevated degree of stress at weaning. The peak frequency of hypoplasias is earlier in the MM (2.5–3.0 years versus 3.0–3.5 years in the pre-Mississippian population). In addition, the rise to and decline from peak frequency occurs approximately 0.5 years earlier in the MM. The earlier and sharper rise to peak frequency suggests earlier and more severe weanling-related stress. Hypoplasias chronologies are undoubtedly influenced by age-related host resistance factors (Sarnat and Schour, 1941). Nevertheless, these data demonstrate that populations may vary in their chronological distribution of hypoplasias and that these variations may provide useful information on age-related patterns of exposure to environmental stressors.

Enamel hypoplasia is a deficiency in enamel thickness resulting from a disruption in amelogenesis (Sarnat and Schour, 1941). This condition is readily observed in erupted teeth as a circumferential line, band, or pitting of decreased enamel thickness (Fig. 1) (Goodman et al., 1980).

Since the introduction of the term in 1893 by Zsigmondy (Sarnat and Schour, 1941:1991), many investigators have studied the etiology of this developmental defect. Enamel hypoplasias have been associated with a wide variety of disease and nutritional deficiencies, including vitamin A deficiency (Wolbach, 1947), vitamin D deficiency (Nikiforouk and Fraser, 1981), fever (Kreshover and Clough, 1953), maternal diabetes (Grahnén and Edlund, 1967), neonatal asphyxia (Grahnén et al., 1969), neonatal jaundice (Watson, 1964), nephrotic syndrome (Schusterman and Fellers, 1969), and gastroenteritis (Smith and Miller, 1979). In his summary of results from clinical and experimental studies, Kreshover states: "... developmental tooth disturbances are generally nonspecific in nature and can be related to a wide variety of systemic disturbances ..." (1960:166). Any systemic disturbance may, therefore, lead to an enamel defect if it is sufficiently severe and long lasting to disturb

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amelogenesis (cf. Kreshover, 1960; Pindborg, 1970, 1982; Yaeger, 1980).

Because many different conditions may result in an enamel hypoplasia, it is difficult to determine their exact cause (Pindborg, 1982). However, since hypoplasias provide a nearly permanent record of stresses occurring during enamel development, they may provide a general index of infant-childhood health (Sarnat and Schour, 1941). The frequency of enamel hypoplasias has been successfully used as an indicator of general health in contemporary (Infante and Gillespie, 1974; Sweeney et al., 1969) and prehistoric (Cook and Buikstra, 1979, Swärdstedt, 1966) populations. For examples, Sweeney and co-workers (1969) found an association between the occurrence of hypoplasias and infection in Guatemalan children, and Swärdstedt (1966) found an increased frequency of hypoplasias in the lower classes of a medieval Swedish population.

Since the age of individuals at the time of the development of hypoplasias can be determined, it is possible to reconstruct the chronology of occurrence of hypoplasias for individuals and populations (Sarnat and Schour, 1941). An analyses of the chronological pattern of hypoplasias should add a useful dimension to epidemiological studies which include hypoplasia data. However, while there is general acceptance that the frequency of hypoplasias may provide an indication of general health status in populations, interpretation of the chronological distribution of hypoplasias is more controversial.

Sarnat and Schour (1941) constructed a frequency distribution for enamel hypoplasias by age of occurrence for sixty hypoplastic individuals from the Chicago area. They found that most hypoplasias (67%) occurred during the first year. Nearly another third occurred during the next 2 years and less than 2% occurred after this time. This chronology remains the only one for an industrial or contemporary population.

The sparcity of further studies on the chronology of hypoplasias may be due to Sarnat and Schour's conclusions that the distribution of hypoplasias is due to the child's biologically determined developmental changes in susceptibility to stressors (Sarnat and Schour, 1942:69; also see Massler et al., 1941:42). Since these age changes in susceptibility to stressors are assumed to be a relatively constant biological phenomena, this position implies that the distribution of hypoplasias should be similar in all populations. Furthermore, it follows that if hypoplasia distributions are similar, they will not provide information on differences in the chronology of environmental events which are causative of hypoplasia-stress.

Giro (1947) supports the view that it is not the environmental event which is important in the determination of a hypoplasia but strictly the age of the individual at the time of the event. He argues for the universality of Sarnat and Schour's (1941) results in stating: "It is a known fact that at least 68% of hypoplasias reported have occurred during the first year of life" (1947:313). Furthermore, he contends that potential causes of hypoplasias, which are not active during this first year, can essentially be eliminated because of this "fact" that hypoplasias invariably occur during the first year (1947:313). More recent dental references continue to accept the relative universality of the Sarnat and Schour enamel hypoplasia chronology (cf. Pinborg, 1970:91–92; Spouge, 1973:152; Yaeger, 1980:100).

In this paper we present chronological distributions of hypoplasias for time-successive populations from the Dickson Mounds, Lewiston, Illinois (A.D. 950–1300). The distribution of hypoplasias in the Dickson populations are compared with reference to the changing pattern of environmental stressor potentially resulting from the transition from an indigenous hunting and gathering tradition to increased use of maize agriculture and involvement in Mississippian lifeways. Our purposes are to demonstrate that the chronological distribution of enamel-hypoplasias in populations is not invariable and that chronological differences reflect differences in age-related patterns of exposure to environmental stressors.

ARCHEOLOGICAL CONTEXT

The Dickson Mounds site is located near the confluence of the Illinois and Spoon Rivers, 4.8 km Southwest of Lewiston, Illinois. The site includes both habitation areas and burial mounds. The environment is one of great diversity and potential for aboriginal exploitation (Harn, 1971).

During the approximately 350 years in which the burial mounds were used, the Dickson population underwent a great change in lifeways. During the first century of occupation (A.D. 950–1050) the burial mounds were used by indigenous gatherer-

hunters of the Late Woodland (LW) tradition (Conrad and Harn, 1972). Habitation sites appear to have been seasonally occupied by a relatively small (75–125) group. During this time and into the next century, contact increased with Mississippian cultures to the south, which had developed in the American Bottom (Fowler, 1978). The period of greatest transition (circa 1050–1150 A.D.) is often referred to as Mississippian Acculturated Late Woodland (MALW) (Goodman et al., 1984). At this time there is evidence for long-distance trade and some use of maize agriculture.

By around 1150 A.D. the Dickson Mounds inhabitants had become completely Mississippianized (MM) (Conrad and Harn, 1972). The settlement pattern consists of hamlets with surrounding camps. Large quantities of trade items are found at both the habitation and burial sites. A population size of between 600 and 1170 has been suggested by Harn (1978:251). The transition from Late Woodland to Mississippian may be summarized as including 1) an increased, though not exclusive, reliance on maize agriculture, 2) increased population density and sedentarism, and 3) extension and intensification of trade with increased involvement with Middle Mississippian centers such as Cahokia in the American Bottom. Although cultural change is great, an analysis of highly heritable dental traits strongly suggests biological continuity for the Dickson populations (Cohen, 1974).

A wide variety of studies have reported on changes in health at Dickson. Of ten indicators of stress, morbidity, and mortality, eight increase through time at Dickson (Goodman et al., 1984). This increase is most evident in the subadult segment of the population. During the Mississippian period there is a fourfold increase in the frequency of individuals with porotic hyperostosis (16 to 64%; Lallo et al., 1977) and a doubling of the frequency of individuals with enamel hypoplasias (0.90 to 1.86 per individual with complete measurements; Goodman et al., 1980) and enamel microdefects (21.4 to 40.0%; Rose et al., 1978). Long-bone growth rates are slowed (Goodman et al., 1984; Lallo, 1973) and the probability of dying increased (Lallo et al., 1980).

Analysis of the chronological pattern of hypoplasias may aid in the determination of the causes of the increase in infant-childhood stress. The well-documented and rapid cultural change, combined with a large body of data on health, makes Dickson an excellent population for an ecological study of the chronology of enamel hypoplasias.

METHODOLOGY

The sample used in this analysis consists of 111 adults and adolescents (Goodman et al., 1980). This includes all of the available Dickson skeletons with complete or near complete permanent dentition.

Of these 111 individuals, 48 are affiliated with either the Late Woodland or Mississippian Acculturated Late Woodland horizons (circa 950–1150 A.D.). These individuals occupy the premound cemetery or the earliest constructed burial mounds (A–E) and comprise an earlier, pre-Mississippian sample (Harn, personal communication). Sixty-one individuals, who occupy the latter built mounds (F–L) comprise a Middle Mississippian sample (circa A.D. 1150–1300) (Harn, personal communication). The remaining two individuals could not be classified due to grave disturbance. The two samples do not significantly differ in their mean ages or sex distribution. The mean age of the total sample is 33.3 years, with a range of 12 to 65 years. There are 50 females, 50 males, and 11 adolescents of indeterminate sex.

Enamel hypoplasias are operationally defined as transverse lines, bands, or pittings of decreased enamel thickness (see Fig. 1) (Goodman et al., 1980). Since hypoplasias are the result of a disruption during the matrix apposition stage of enamel development, the thickness of enamel is affected (Yaeger, 1980). These developmental defects are easily discerned and are not readily confused with other enamel surface irregularities. Confirmation of hypoplasias is aided by use of a dental probe and binocular microscope.

Enamel hypoplasias were recorded on all teeth except third molars. The distance of hypoplasias from the cemento-enamel junction was recorded in order to determine the age of individuals at the time of hypoplasias development. This determination was made with reference to the chronology of enamel development (Massler et al., 1941:48), and use of a chart for conversion of distance from the cemento-enamel junction to age at development (Goodman et al., 1980:520; Swärdstedt, 1966:38). Hypoplasias were assigned to 14 half-year intervals from birth–0.5 years to 6.5–7.0 years.

In order to determine the pattern of stress for individuals, hypoplasias on different teeth of the same individual were "matched" to

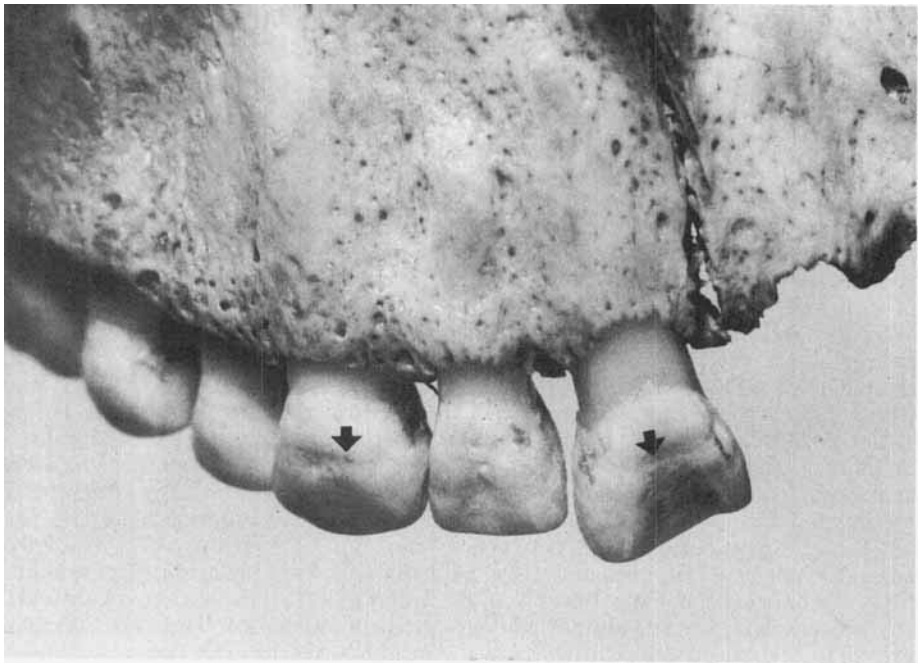

Fig. 1. Enamel hypoplasias in a prehistoric Amerindian (note arrows). The location of these hypoplasias, near the middle of the crown of the maxillary canine and near the cemento-enamel junction of the maxillary central incisor, is suggestive of their being the result of a common stressor occurring near a developmental age of 3.5 years.

determine if they developed during the same half-year period (Goodman et al., 1980). A period was recorded as "positive for an episode of stress" if two or more teeth had hypoplasias which developed during that period. A period was recorded as "negative for stress" if at least four teeth could be evaluated for the presence of a hypoplasia during that period and no more than one had a hypoplasia at this time. Finally, a period was recorded as "undetermined stress level" if four teeth could not be graded for the occurrence of hypoplasias and less than two were hypoplastic during that period.

RESULTS

The chronological distribution of hypoplasias-stress is presented in Figure 2 for the pre-Mississippian (combined Late Woodland/ Mississippian Acculturated Late Woodland) and the Middle Mississippian samples. The chronology for the pre-Mississippian period approximates a normally shaped curve with a distinct single peak near the median. The frequency of hypoplasias-stress is low before 2 years of age and after 4 years of age (under 10% per half-year period). In contrast, the frequency of hypoplasias-stress is always greater than 10% per half-year period between the ages of 2 and 4. The mode occurs at 3.0–3.5 years. More than one-fifth of individuals (22.9%) have evidence of stress during this half-year period.

The chronological distribution of hypoplasias-stress during the Mississippian period (Fig. 2) is similar to that for the pre-Mississippian period. The frequency of hypoplasias is lowest before 1.5 years and after 3.5 years and greatest between these ages. The Mississippian distribution differs from that of the earlier period by having 1) an earlier and sharper rise to peak frequency, 2) an earlier and sharper decline from peak frequency, and 3) a less regular shape (not clearly single peaked). At 1.5–2.0 years, the frequency of hypoplasias-stress in the Mississippian has risen to 28.0% as compared to 8.8% in the earlier period. The frequency of hypoplasias-stress in the Mississippian is maintained at over 20% per half-year period until the 3.5–4.0 half-year period. At this time, the frequency of hypoplasias sharply drops to 3.3%,

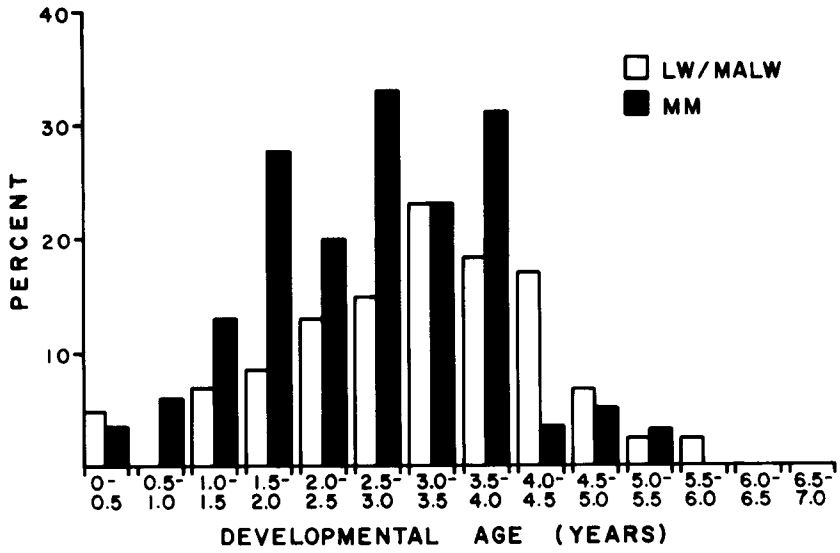

Fig. 2. Frequency distribution of enamel hypoplasias by half-year periods in two Dickson Mounds populations. MM, Middle Mississippian; LW/MALW, Late Woodland/Mississippian Acculturated Late Woodland.

TABLE 1. Frequency of enamel hypoplasias stress by half-year periods[1]

	Stress episode/observations			
	LW/MALW	MM	χ^{2*}	P(2-tail)
Birth–0.5	1/22	1/28	0.03	ns
0.5–1.0	0/22	2/34	1.34	ns
1.0–1.5	2/29	6/45	0.75	ns
1.5–2.0	3/34	14/50	3.50	.061
2.0–2.5	5/38	11/50	0.33	ns
2.5–3.0	6/40	19/58	3.05	.081
3.0–3.5	11/48	14/61	0.01	ns
3.5–4.0	9/48	19/61	1.56	ns
4.0–4.5	8/48	2/61	4.28	.038
4.5–5.0	3/48	3/61	0.02	ns
5.0–5.5	1/48	2/61	0.01	ns
5.5–6.0	1/48	0/61	0.14	ns
6.0–6.5	0/48	0/61	0.00	ns
6.5–7.0	0/48	0/61	0.00	ns

[1]LW/MALW, Late Woodland/Mississippian Acculturated Late Woodland; MM, Middle Mississippian.
*χ^2 values computed with correction for df = 1.

much below the 16.7% frequency of the pre-Mississippian period. The highest frequency of hypoplasias in the Mississippian occurs during the 2.5–3.0 half-year period (32.8%).

The differences between the two distributions are primarily due to the earlier manifestation of hypoplasias-stress in the Mississippian. The rise to and decline from peak frequency occur 0.5 years earlier in the Mississippian. Also, the peak frequency is 0.5 years earlier. The earlier occurrence of hypoplasias in the Mississippian is most clearly seen by comparison of the cumulative frequency distributions (Fig. 3). By the 1.5–2.0 half-year period, 24.7% of the 93 episodes of stress in the Mississippian have occurred as compared to 12.0% of the 50 episodes of stress in the earlier period. The cumulative frequency difference is greatest by 2.5–3.0 years (57.0 to 34.0%).

The differences in distribution are supported by a comparison of the significance of the hypoplasia frequency differences by half-year periods (Table 1). There is a higher frequency of hypoplasias in the Mississippian during all seven half-year periods between the ages of 0.5 and 4.0 years. The difference approaches significance ($p < .10$) between the ages of 1.5–2.0 years and 2.5–3.0 years. Conversely, the frequency of hypoplasias-stress is greater in the earlier period for three of four half-year periods between the ages of 4.0 and 6.0 years. The frequency of hypoplasias is significantly greater ($p < .05$) in the pre-Mississippian period for the 4.0–4.5 half-year period.

DISCUSSION

The chronological distribution of hypoplasias is similar for Dickson Mounds samples.

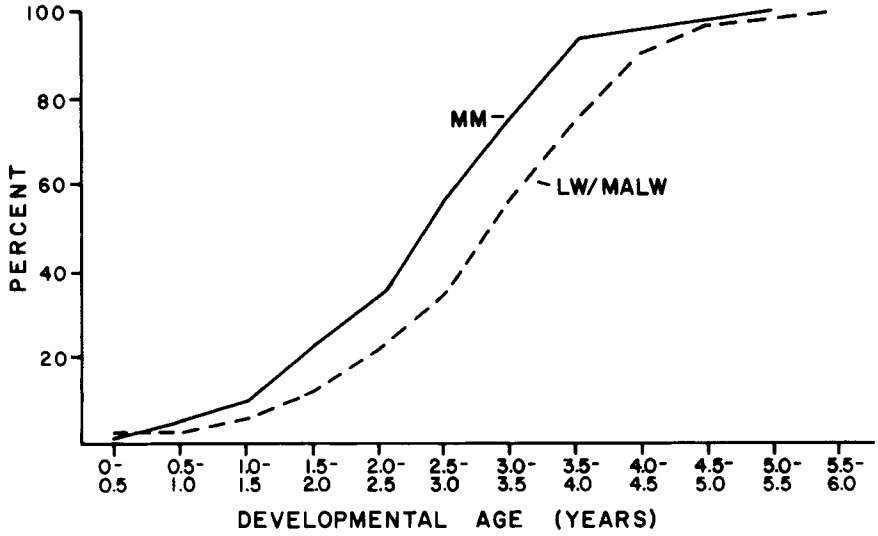

Fig. 3. The cumulative frequency of enamel hypoplasias in two Dickson Mounds populations. MM, Middle Mississippian; LW/MALW, Late Woodland/Mississippian Acculturated Late Woodland.

Both distributions reflect considerable stress between the ages of 2 and 4. A likely cause of this stress may be the weanling diet. Cook (1971:2), in a study of human growth and subsistence during the Woodland, argues that a weanling diet such as maize, which is high in carbohydrate and low in protein, is likely to result in considerable nutritional stress. She predicts that this stress should result in enamel hypoplasias.

The results from Dickson are consistent with Cook's predictions. While the 2- to 4-year period is a time of considerable stress for the pre-Mississippian population, this pattern is magnified during the Mississippian period. This populational difference is consistent with the increased rate of mortality before 5 years of age in the Mississippian (25.9 to 37.5%; Lallo et al., 1978; 21–22). The earlier and higher peak frequency of hypoplasias in the Mississippian may be a reflection an earlier and greater use of an insufficient weanling diet and, in particular, one which is high in use of cereal gruel.

The Dickson chronologies are relatively similar to all other published hypoplasia chronologies which we are aware of for non-industrial populations. The peak frequency of hypoplasias from Westerhus, a medieval Swedish population, is between 2.0 and 4.0 years (Swärdstedt, 1966:89), while the peak frequency of hypoplasias in a prehistoric California series is delayed by approximately 1 to 2 years, as compared to the Swedish and Dickson chronologies (Schulz and McHenry, 1975:913). The relative similarities in these chronologies may reflect similar chronologies of environmental stress. The delayed peak in the California series may be due to the exclusive use of the mandibular canine, a tooth which is more susceptible to hypoplasias at later ages (Goodman, 1984; Goodman and Armelagos, 1980).

All of these chronologies are relatively different from the original Chicago modern chronology (Sarnat and Schour, 1941:1992). By the time of peak occurrence of hypoplasias in any of the other populations, the frequency of hypoplasias in the Chicago population is near zero.

The differences between the Chicago chronology and the prehistoric ones have not been adequately explained. While Swärdstedt (1966) acknowledges that the Chicago and Swedish chronologies are very different, he does not comment on the difference. Schulz and McHenry (1975) attribute the differences between the California prehistoric and Chicago modern chronologies to increased mortality during the first year in the prehistoric group. They contend that individuals who died during the first year would have developed hypoplasias, had they survived the stress. This explanation may account for

some of the lowered frequency of hypoplasias in the prehistoric population in the first year. However, it can not account for peak frequencies after the age of one.

We believe that the best explanation for the variations the distribution of hypoplasias in the different populations is that they reflect variations in the chronological occurrence of stress. Data from the prehistoric populations do not provide any support for the view that the chronology of hypoplasias reflects the underlying chronology of biological susceptibility of stress, unless this pattern is radically different from that which has been proposed by Sarnat and Schour (1941).

CONCLUSIONS

The frequency of hypoplasias by half-year period from birth–0.5 to 6.5–7.0 years has been presented for two time-successive prehistoric populations from Dickson Mounds, Illinois. Both populations have a low frequency of hypoplasias in the first 1.5 years and after the age of 4 and the highest frequency of hypoplasias between the ages of 2 and 4. The chronological pattern of hypoplasias suggests an insufficient weanling diet. Furthermore, an earlier and sharper peak during the Mississippian period may be due to an earlier and increased use of maize in the weanling diet at this time. We conclude that there is variability in hypoplasia chronologies between populations which are closely matched and comparably studied and that these differences reflect differences in the age-related pattern of exposure to environmental stressors.

A comparison of the Dickson hypoplasia chronologies with three other published chronologies provides additional support for this conclusion. Contrary to the prediction of Sarnat and Schour (1941), there is great variability between populations in their chronological distribution of hypoplasias. While host susceptibility may be an important determinant of the chronological distribution of hypoplasias, its role is certainly different from that which had been proposed by Sarnat and Schour (1941). Despite the selection of surviving individuals, low hypoplasia frequencies in the first year of life in preindustrial populations call into question the contention that individuals are most susceptible to hypoplasias during the first year of life. Further research is needed in order to determine the role of general host- and tooth-dependent factors in the development of enamel hypoplasias.

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