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Childhood Stress and Decreased Longevity in a Prehistoric Population

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Early childhood is a period of increased risk for individuals in a variety of human populations (Wood 1983). With the classic synergy of undernutrition and increased susceptibility to infection, this age is one of increased morbidity and mortality (Preston 1980). In fact, the majority of public health efforts to increase survivorship in developing countries today focus on increasing survival to age five years (Mosley 1983).

A less well considered aspect of the dynamics of this age period concerns the long-term consequences of these early stresses. Infrequently addressed, for example, are long-term health and mortality patterns following *survivorship* of early stress. A noteworthy exception is Meindl and Swedlund, who study the effect of "autumnal fever" (1977:410) on mortality patterns in a historic New England cohort. These authors find that the mean longevity of the cohort "exposed" and surviving the early infectious disease stress is greater than a later unexposed cohort. Meindl and Swedlund propose a genotype screening mechanism to account for this pattern. Unfortunately, however, these authors are unable to note submortality variations in response to the epidemic, as there is no window into these individuals' response differences.

One possible biological window into observing long-term consequences of early stress is dental enamel hypoplasia, deficiency in enamel thickness resulting from a disruption in ameloblastic matrix formation (Sarnat and Schour 1941). Unlike most biological tissues, enamel does not incorporate the ability to remodel: once enamel matures its structure is unalterable by internal biological events. Additionally, since enamel is secreted in a regular and ring-like fashion, the tooth's enamel crown provides a permanent chronological record of metabolic disruptions that occurred during its time of development (Kreshover 1960; Sarnat and Schour 1941; Via and Churchill 1959).

Enamel defects may be due to hereditary conditions, localized trauma, or systemic disruption (stress) (Pindborg 1970, 1982). Hypoplasias that are due to systemic disruptions (chronologic enamel hypoplasias) may be distinguished from those that are the result of other factors (Yaeger 1980). Systemic disruptions are likely to affect more than one tooth, and the location of the defect on these teeth will reflect the relative completeness of crown development at the time of the stress (Sarnat and Schour 1941; Yaeger 1980; also see Figure 1). This analysis includes only those defects that are the result of systemic disruptions (stress-hypoplasias).

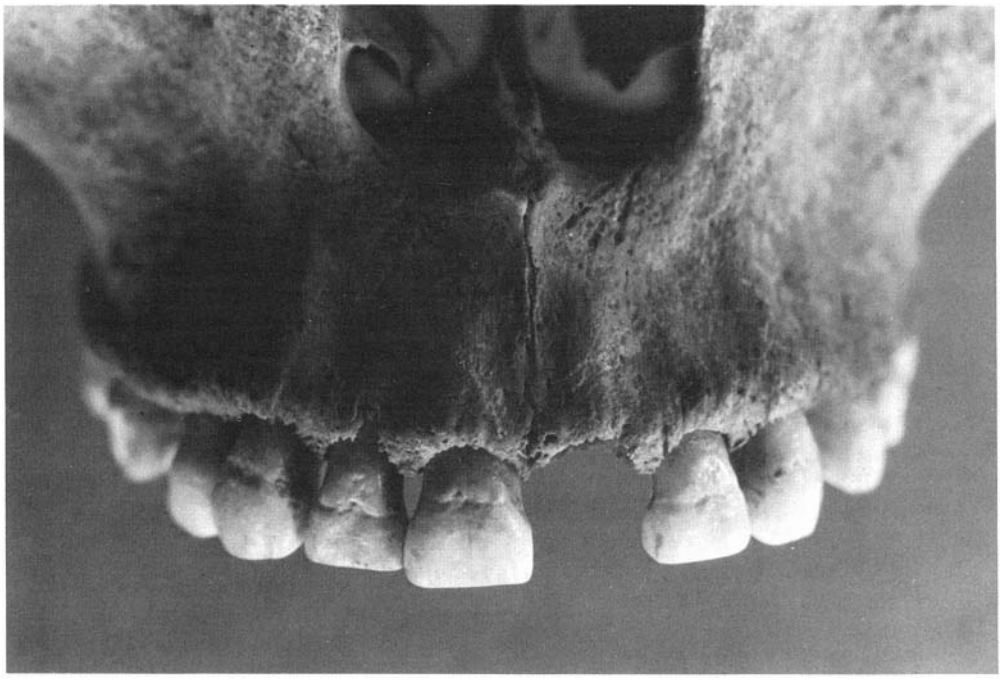

Figure 1
Chronologic enamel hypoplasias (stress-hypoplasias) on anterior, maxillary permanent teeth. Hypoplasias are observable on the right central incisor, the right and left lateral incisors, and the right and left canines. All hypoplasias occur between 3.0–3.5 years developmental age and appear to be the result of the same systemic disruption (stress).

The following is an analysis of the association between longevity and stresses occurring at 3.5 to 7.0 years of age, as indicated by the position of hypoplasias on permanent tooth crowns in individuals from the Dickson Mounds, Illinois, archeological populations (A.D. 1000–1350). Our specific objectives are to provide evidence for the association between stress during childhood and longevity, to suggest explanatory mechanisms for this association, and to demonstrate the potential use of hypoplasias in studying the long-term effects of stress in prehistoric *and* contemporary populations.

Methods and Materials

The sample consists of 111 adults and adolescents (50 males, 50 females, and 11 adolescents of undetermined sex) from the Dickson Mounds, a multicomponent habitation-burial complex located in Lewiston, Illinois (Goodman, Armelagos, and Rose 1980, 1984). The burials have been divided into three cultural horizons: Late Woodland (LW), Mississippian Acculturated Late Woodland (MALW), and an early and late Middle Mississippian (MM) (Harn 1971, 1978, 1980). During the Late Woodland period (ca. A.D. 1000–1100) the area was occupied by a relatively small (75–125) and semi-sedentary hunting and gathering population with seasonal camp sites and an economy directed toward the use of a broad spectrum of local fauna and flora. The MALW (ca. A.D. 1050–1175) is a transitional period, possibly overlapping for some time with the LW, during which local populations began to come under the influence of Mississippian cultures further to the south in the American Bottom (Harn 1978, 1986). During the MM

period (ca. A.D. 1175–1350) the Mississippianization of local populations became complete with the culmination of trends toward extended and intensified trade networks, increased population density, size, and sedentarism, and possibly greater reliance on maize agriculture (Harn 1978).

These changes have been associated with an increase in nutritional and infectious pathologies and a decrease in life expectancy (Goodman, Lallo, Armelagos, and Rose 1984). Porotic hyperostosis, an indication of iron deficiency anemia, is four times as prevalent among MM subadults as compared to LW subadults (52% to 13%) (Lallo, Armelagos, and Mensforth 1977). Periosteal infections increase from 27% in the combined LW plus MALW to 67% in the MM (Lallo, Armelagos, and Rose 1978), and the frequency of enamel hypoplasias doubles in adults and adolescents (Goodman, Armelagos, and Rose 1980). Life expectancy is lower at all age intervals in the MM when compared to the combined LW and MALW samples (Moore, Swedlund, and Armelagos 1975).

Enamel hypoplasias were recorded on all permanent teeth except third molars. Hypoplasias are easily identified and were operationally defined as circumferential lines, bands, or pitting of decreased enamel thickness (Goodman, Armelagos, and Rose 1980, 1984; see Figure 1). The distance of the hypoplasia from the cemento-enamel junction was measured to one-tenth mm using a thin-tipped caliper. This distance measure was converted to the individual's dental age when the disruption occurred, based on the developmental standard of Massler, Schour, and Sarnat (1941).

Each half-year period between birth and 7.0 years was rated as either stress-positive, stress-negative, or undetermined, based on the following criteria: (1) a half-year period was rated as stress-positive if there were two or more teeth with hypoplasias occurring during the half-year period; (2) a half-year period was rated as stress-negative if less than two hypoplasias occurred during this period and at least four tooth crowns were available for scoring whose development includes this period; and (3) a half-year period was rated as undetermined if one or no hypoplasias occurred during this period and there were less than four tooth crowns available for scoring whose development included this period. By using this method, a chronology of stress by half-year periods was developed for each individual from birth to seven years of age (Goodman, Armelagos, and Rose 1980, 1984).

This study is based on the evidence for stress between 3.5 and 7.0 years of age. The extensive dental attrition characteristic of the Dickson series limited our ability to observe the enamel record of stress from birth to 3.5 years. Due mainly to occlusal surface attrition, many individuals have a series of undetermined periods starting at birth–0.5 years and extending as far as the 3.0–3.5 year period (Goodman, Armelagos, and Rose 1984). However, all individuals yielded a complete record of stress-hypoplasias for the seven half-year periods from 3.5–7.0 years.

Results

We compared the mean age at death of individuals in the three cultural horizons and in the total sample for those individuals with no hypoplasias-stress periods (total n = 50, 45.0%), one hypoplasia-stress period (total n = 45, 40.5%), and two or more hypoplasias-stress periods (two defects: total n = 14, 12.6%; three hypoplasias: total n = 2, 1.8%) (Table 1, Figure 2). The lowest overall frequency of hypoplasias is found in the LW group. All individuals in this sample have either one or no hypoplasias-stress periods between 3.5 and 7.0 years. Individuals with one hypoplasia-stress period have a slightly greater mean age at death (34.7 years) than individuals with no hypoplasias-stress periods (31.6 years). This difference, however, is not statistically significant (Table 1; F-ratio = .35).

This association between hypoplasias-stress periods and longevity is reversed during the MALW periods. The mean age at death of individuals without hypoplasias-stress periods is 36.6 years, or

Table 1
Comparison of mean ages at death for individuals by cultural horizon and number of hypoplasias-stress periods between 3.5–7.0 years developmental age.

	Sample size	Mean	S.D.	1-way ANOVA (F-ratio)	A priori (A vs. B)	Contrasts (A vs. C)	T-values (A vs. B + C)
Late Woodland	20	33.0	11.5	.35	.55	—	.55
No hypoplasias (A)	11	31.6	10.4				
One hypoplasia (B)	9	34.7	13.0				
2–3 hypoplasias (C)	—	—	—	—			
MALW	45	33.3	13.4	1.44	1.22	1.53	1.69
No hypoplasias (A)	22	36.6	12.8				
One hypoplasia (B)	14	31.1	14.7				
2–3 hypoplasias (C)	9	28.6	11.7				
Middle Mississippian	46	31.6	11.2	6.52[c]	2.25[b]	3.50[d]	3.52[d]
No hypoplasias (A)	17	37.5	9.0				
One hypoplasia (B)	22	30.2	11.0				
2–3 hypoplasias (C)	7	21.8	8.7				
Total sample	111	32.5	12.1	4.99[c]	1.84[a]	3.04[c]	3.08[c]
No hypoplasias (A)	40	35.8	10.1				
One hypoplasia (B)	45	31.4	12.7				
2–3 hypoplasias (C)	16	25.6	10.8				

[a]2-tailed $p \leq .10$.
[b]2-tailed $p \leq .05$.
[c]2-tailed $p \leq .01$.
[d]2-tailed $p \leq .001$.

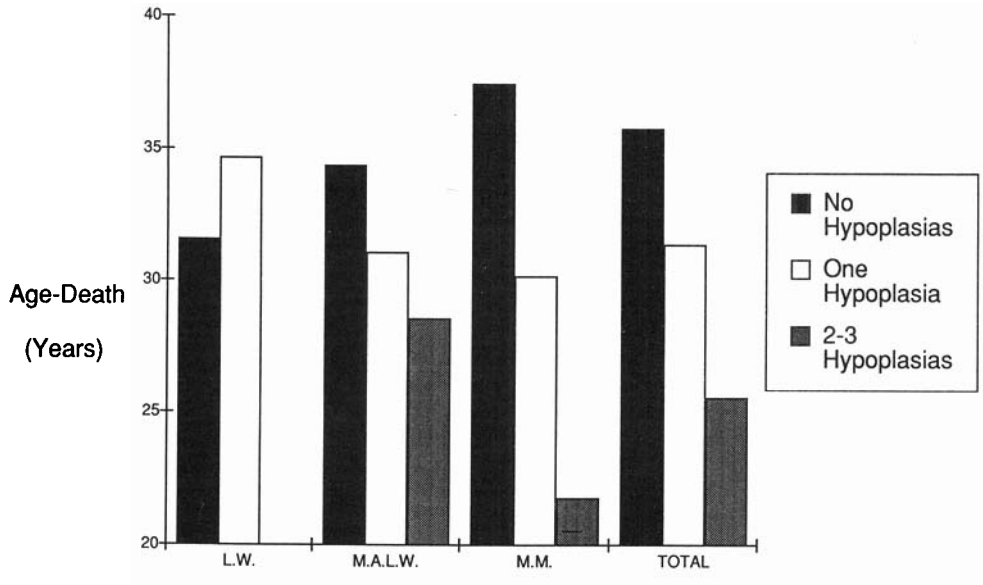

Figure 2
Mean ages at death of Dickson Mounds adolescents/adults by number of hypoplasias-stress periods between 3.5–7.0 years developmental age. LW = Late Woodland, MALW = Mississippian Acculturated Late Woodland, MM = Middle Mississippian.

5.5 years greater than those with one hypoplasia-stress period (31.1 years) and 8.0 years greater than those with two or more hypoplasias-stress periods (28.8 years) (Figure 2).

This inverse association between stress periods and mean age at death is most pronounced during the Middle Mississippian. The mean age at death of individuals without hypoplasias-stress periods is 37.5 years, or 7.3 years longer than those with one hypoplasia-stress period and 15.7 years longer than those with two or more hypoplasias-stress periods (Figure 2). A one-way ANOVA, testing for the statistical significance of differences in ages at death among hypoplasia-stress period groups (Nie et al. 1975), yielded an F-ratio of 6.52 (Table 1; $p < .01$).

Furthermore, we tested for the specificity of differences between groups with a series of a priori contrasts (see Table 1). These provide a comparison of the mean age at death in group A (no stress periods) with (1) group B (one stress period), (2) group C (two or three stress periods), and (3) group B + C combined (one or more stress periods). For the MM group, all a priori contrasts yielded statistically significant results at the .05 level of confidence. The most significant differences are found in comparing individuals without any stress periods with those with one or more or two or more stress periods ($t = 3.50$ and 3.52; $p < .001$ and $< .001$).

Finally, there is a significant decrease in longevity with childhood stress in the total sample (Table 1; Figure 2). The mean age at death of individuals without hypoplasias-stress periods is 35.8 years, or 4.4 years greater than for those with one hypoplasia-stress period (31.4 years) and 10.2 years greater than for those with two or more hypoplasia-stress periods (25.6 years). The ANOVA yields a significant F-ratio (4.99, $p < .01$), and all a priori contrasts are significant at the .10 level of confidence. Again, the most significant contrasts are between the no hypoplasias-stress periods group and the one or more hypoplasias-stress periods and two or more hypoplasias-stress pe-

riods groups (t = 3.04 and 3.08; $p < .01$, $< .01$).

Discussion

At least three prior studies of archeological groups have noted an inverse relationship between the occurrence of enamel hypoplasias and longevity. White (1978) assessed hypoplasias on permanent maxillary first molars from South African Plio-Pleistocene australopithecines (ca. 1.5–3.0 M.Y.B.P.). He noted that individuals with maxillary first molar hypoplasias from the Swartkrans site (n = 6) had "lower-than-expected" ages at death. These individuals die between four and thirteen years of age, while individuals with non-hypoplastic first molars (n = 110) die between 8 and 31 years of age. We have calculated the mean age at death of individuals in these groups as 7.8 and 19.6 years, respectively. While this study suffers from a restricted sample size and lack of precision in assigning ages at death to fragmentary paleontological materials, the data nonetheless demonstrate a decrease in fitness associated with hypoplasias.

Cook and Buikstra (1979) compare the mean age at death of infants and children with and without postnatal defects on deciduous tooth crowns from Middle and Late Woodland skeletal samples from Illinois. They conclude that postnatal dental defects are associated with decreased longevity during both the Middle and Late Woodland periods.

Rose, Armelagos, and Lallo (1978) studied areas of disturbed enamel formation visible in thin-section (Wilson bands) in Middle Woodland, Mississippian Acculturated Late Woodland, and Middle Mississippian samples from Illinois. They found that individuals with Wilson bands died at an earlier mean age at death in all samples. Overall, we calculated the average age at death of the 21 individuals with Wilson bands to be 26.7 years, or 15.4 years less than the average age of the 66 individuals without Wilson bands (42.1 years). While these results strongly support the suggestion that childhood stress reduces longevity, the study failed to control for differential availability of enamel for study. Younger individuals will have less dental attrition and therefore more enamel available for study and the observation of a dental defect. Nonetheless, these studies support our contention that dental developmental defect may predispose to an earlier age at death and that this relationship warrants further study.

Our results support the general trend noted in the previous studies. Specifically, they provide a confirmation and extension of results from the Rose, Armelagos, and Lallo study (1978) of the histological evidence of dental defects. Rose and co-workers provide data on the MALW and MM periods. The differences in mean age at death of individuals with and without Wilson bands in their MALW and MM samples is 11.8 and 12.8 years, respectively. Our mean differences for individuals with two or more versus no hypoplasias are 8.0 and 15.7 years respectively for MALW and MM samples.

There are at least three processes that may account for the association between childhood stress and decreased life expectancy. First, these data may result from differential lifelong patterns of biological susceptibility to physiological disruptions and their adverse effects. An increased susceptibility to stress may cause both an increased frequency of childhood hypoplasias and an earlier age at death. That is, individuals who are ill during childhood continue to fall ill as adults. This association is due to a "weaker constitution," and the sum effect is earlier death.

Second, individuals who were exposed to and survived a period of severe childhood stress may suffer a loss in ability to respond to other stresses. In a sense, these individuals are "biologically damaged" by the early stress. The wear and tear of stresses during development may render them less fit to respond to and survive subsequent stresses. For example, suboptimal early nutrition has been proposed as a mechanism for later immune dysfunction (Chandra 1975; Miler 1982).

Third, these data may result from differential lifelong patterns of behaviorally and culturally based exposure to stressors. An increased lifelong potential for exposure to stressors may cause both an increased frequency of childhood stress and earlier ages at death. Barker and Osmond (1986a, 1986b) have shown an association between poor nutrition and respiratory infections in childhood and coronary disease and chronic bronchitis in adulthood. They propose that this relationship is mainly due to social conditions present in childhood, which are likely to persist into adult life.

It is not possible to rule out any of these processes. All may contribute to the associations that we have observed. However, the wide variation in the degree of association between stress and longevity supports the view that the association is not solely a function of biological factors, since these samples appear to be genetically continuous (Cohen 1974; Goodman, Lallo, Armelagos, and Rose 1984). Furthermore, the greatest difference between stressed and nonstressed group mean ages at death occurs in the MM period. Since this is also the horizon in which status differences are likely to be greatest (Rothschild 1979), these data suggest that lifelong differences in social status, and therefore differential cultural buffering from stress, may be important. Unfortunately, it is difficult to assess cultural buffering in archeological populations. We have tested to see if differences in type of grave offering, an indicator of status differences, might explain the association between stress and longevity (Goodman, Rothschild, and Armelagos 1983). While individuals with no grave goods are more likely to have multiple hypoplasias (17.6%) as compared to individuals without non-utilitarian offerings (8.7%), this relationship does not explain the association between hypoplasias and age-at-death (Goodman, Rothschild, and Armelagos 1983). The inability to use grave goods to explain the observed association is probably more a function of their uncertainty as indicators of status than of the insignificance of status differences in the etiology of childhood stress and adult mortality.

Conclusions

Chronologic enamel hypoplasias, circumferential areas of decreased enamel thickness, provide a record of disturbed enamel matrix formation resulting from systemic physiological disruption (stress). Permanent dentition enamel hypoplasias were employed as retrospective "memories" of stresses occurring between 3.5 and 7.0 years developmental age. We compared the mean age at death of individuals (n = 111) from the Dickson Mounds, Illinois (A.D. 1000?–1350), with and without hypoplasias-stress periods.

Adults and adolescents with two or more hypoplasias-stress periods have a lower mean longevity (25.6 years) than those with one (31.6 years) or no hypoplasias-stress periods (35.8 years). We propose three biocultural hypotheses to explain this relationship: (1) "inherent biological susceptibility," (2) "biological damage," and (3) "lifelong differential cultural buffering."

While our data have largely been unable to distinguish among these mechanisms we suggest that a cultural buffering hypothesis is most congruent with the pattern of associations. Furthermore, this hypothesis is not exclusive of a "biological damage" mechanism. Low status during childhood may promote disease and undernutrition, which leaves individuals less able to rally from future insults.

Hypoplasias appear to be an effective tool for studying the longitudinal pattern of stress, morbidity, and mortality in prehistoric populations. Further study of the association between childhood stress and longevity in prehistoric populations is warranted. These analyses should help to establish the consistency of the association between childhood stress and adult longevity and the processes that account for this association.

Enamel hypoplasias and other biological markers may provide an alternative means for studying the long-term effects of stress in contemporary populations.

Since these markers provide a biologically unbiased "memory" of stress, they may yield more consistent patterns of effect than individuals' recall of stressful events or other retrospective measures of exposure. In combination with supplementary data on childhood illnesses and nutrition, such an analysis may provide the most direct method for understanding the processes by which childhood stress may be associated with events occurring later in life.

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Detecting Positions in Networks: A Formal Analysis of Loose Social Structure in Rural Java

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Javanese society is one of a number of Southeast Asian societies that are renowned for being loosely structured (see Foster 1984). In Java the notion of loose social structure relates to the fact that (1) large-scale kin groups are absent, (2) social class and religious groups crosscut each other, and (3) the durability of marriage and neighborhood bonds between individuals is weak. The ensuing social structure does not consist of tight corporate groups enforcing uniform obedience over the course of time; instead, actors are embedded in a social world characterized by diverse and shifting social linkages demanding loyalties of differing content and strength. In this report I address the issue of loose structure in Javanese ethnography and use my own network data on an Islamic rural elite to explore this topic and analyze it formally. As qualitative analysis is frequently unable to discover subsets of similarly related actors in weakly structured systems, the last section is devoted to an application of formal network methods appropriate to this task.

The Problem of Loose Social Structure in Java

In the Javanese bilateral system of descent, nuclear families form the building blocks of social organization. Neolocal residence is common, and larger kin groups are virtually absent. Households *(somah)* are built upon nuclear or stem families. These are the main units of economic, social, and emotional support and of decision making. Household members share resources and income, and they work together to earn a living. Beyond the household, parents and (adult) children support each other economically and socially. In the rural areas, landholding