New model of coronary heart disease

A new model for the causation of coronary heart disease is emerging (Barker, 1994). Under the old model an inappropriate lifestyle, including cigarette smoking and lack of exercise, leads to accelerated destruction of the body in middle and late life, including the more rapid development of atheroma, raised blood pressure, and the development of insulin resistance. Under the new model, coronary heart disease results not primarily from external forces but from the body's internal environment, homeostatic settings of enzyme activity, cell receptors, and hormone feedback, which are established in response to undernutrition *in utero* and lead eventually to premature death.

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11 *Early life stresses and adult health: insights from dental enamel development*

ALAN H. GOODMAN

Introduction

Because early life is often a stage of increased physiological stress, morbidity, and mortality, this period is a justified focus of biomedical research and public health intervention efforts (Mosley, 1983; Preston, 1980; Wood, 1983). Indeed, a widely accepted public health goal is to increase survival to age five (Mosley, 1983). In contrast, an up-to-now infrequently considered question concerns the consequences in adulthood of survived, early life stress. What happens to the survivors of undernutrition and other early perturbations? Do these individuals go on to lead healthy and high functioning lives as adults, or do they suffer illnesses more often and die younger? What biocultural processes might link early life events and stresses to later health and functioning?

Although questions pertaining to the long-term consequences of early stress are both fascinating and important, they have not been the subject of sustained and systematic investigation, most likely because of epidemiological difficulties in linking present events to events in the distant past. Thus, the main purpose of this paper is to critically evaluate potential hard tissue 'windows' or memories of early life stress. If these hard tissue measures are available in adulthood as reliable, valid, and unaltered epidemiological measures of early events, they may provide a unique means for linking early events to adult conditions. Finally, whereas this chapter focuses on applications from past populations, in fact mainly 'skeletal series', the methods are not limited to past or skeletal populations. Indeed, a main conclusion is that studies of hard tissue memories of past physiological perturbations should be undertaken with living individuals and groups, in situations in which the results can be contextualized and processes better understood. In this chapter: (i) common epidemiological

difficulties in linking early stress and adult health are briefly reviewed; (ii) various bone and tooth bioassays that might be used as measures of early life stress are compared; (iii) enamel, and specifically linear enamel hypoplasias (LEH), a developmental enamel defect, are highlighted as they seem to have the greatest potential for these types of epidemiological concerns; (iv) two examples are reviewed on the use of LEH in linking early and later health (one from Dickson Mounds, an archaeological population, and the other from Hammon-Todd, a historical skeletal series); and (v) some conclusions and implications are suggested for better determining the processes by which early and late events may be linked.

Linking early events and adult health

A prominent assumption underlying much of the literature on development and ageing is that stressful events occurring early in development may influence adult health. Freud, for example, believed that adult phobias were ultimately caused by traumas occurring during well-defined psychodevelopmental stages. Whereas it is ambiguous in many other areas, psychoanalytical theory is clear in predicting that the type and severity of mental illness is a function of the severity and timing of traumas during development.

Aetiological links between childhood stress and adult health are also prominent in the physical health literature. All but a few theories of ageing leave a place for the influence of early events. A variety of 'wear and tear' theories predict that repeated or chronic stress will predispose to an increased rate of loss of functional abilities, more illness, and early mortality. Although common, these assumptions and theories are hard to evaluate. Controlled animal experiments have provided a variety of leads (Herrenkohl, 1979), but the applicability of these studies to humans is uncertain.

Studies of the long-term consequences of early stress in humans may be divided into a few fundamental types. The most robust experimental design involves prospective studies in which individuals have been followed throughout life (Wadsworth, 1986). Unfortunately, there are few such studies: they are costly, difficult to conduct and maintain, and, by definition, they take a long time. A more common type of study is based on retrospective reporting of a stressful event during early life. Whereas these case control designs can be useful, they all suffer from the vagaries of individual memories. This recall error may be eliminated in studies of historically documented stressful events such as the Dutch Famine of 1944–45 (Stein *et al.*, 1975) and the experience of living in a concentration camp (Schmolling, 1984). These unique 'natural' experiments are likely to

continue to provide key insights into the long-term consequences of early stresses.

Other researchers have avoided the vagaries of memory by recovering records of health and development from the prenatal and neonatal period, and linking it to adult morbidity and mortality. This might be done on the level of the population or the individual. For example, in an ecological design, Barker and colleagues have frequently linked known historical socioeconomic and health conditions to geographic variation in health in more recent times (Barker & Osmond, 1986*a*,*b*, 1987), or have followed individuals with early health/nutritional status records, such as birthweights, and contrast health experiences in adulthood in relationship to the records from early life (see Barker *et al.*, 1990, 1992).

In a sense, the bioassays that are reviewed below are employed in this last way. The only difference is that a currently measurable biological characteristic replaces a written record. This is exactly what is done when adult height is employed as a measure of early life nutritional status (D'Avanzo, La Vecchia & Negri, 1994; Marmot, Shipley & Rose, 1984; Waaler, 1984). The obvious advantage of this method is that one need not depend on the availability of prior health records; early life nutrition and health status is measured at the same time that current health is assessed.

In summary, the tool kit for studying the relationship between early stress and adult health, and making sense of these relationships, is rather sparse. Methods generally trade off reduced temporal and financial costs for epidemiological soundness. Moreover, it is harder still to interpret processes that link past and present. How, for example, does one control for the socioeconomic conditions that may effect exposure to early, intermediate, and late stressors? None the less, these are interesting and important questions and with some creativity they may be studied empirically. The aim of this chapter is to suggest some further research tools to bring to light these hidden relationships.

Skeletal memories of stress

In studying health in past populations; paleoepidemiologists have only bones and teeth as guides. Although limited, a variety of skeletal measures of early life perturbation and disease have been proposed and utilized, and furthermore, the structures upon which these measures are based stabilize and may be discerned in adult skeletons (Goodman & Armelagos, 1989; Martin, Goodman & Armelagos, 1985). Common examples of these measures of childhood stress or ill-health seen in adult skeletons include porotic hyperostosis, bone infection (periostitis), and the developmental indicators of Harris lines, skull base height, vertebral neural canal (VNC)

diameters, long bone lengths and widths, and developmental enamel defects (specifically linear enamel hypoplasia) (Goodman & Armelagos, 1989; Martin *et al.*, 1985).

Porotic hyperostosis, usually interpreted to be a sign of iron-related anaemia (Martin *et al.*, 1985), and periostitis, a sign of generalized bone infection, are bone pathologies that can be observed in adulthood, and with some assurance be traced to an infant–childhood origin. Mittler and Van Gerven (1994) have recently shown that cribra orbitalia, a type of porotic hyperostosis, is linked dramatically to decreased longevity in an archaeological population from Sudanese Nubia. Unfortunately, it is not always easy to estimate the age at which a lesion developed, since bone turnover and remodelling does occur thus obscuring some lesions, and this method has little applicability to living population research because these bone pathologies are greatly obscured by skin and soft tissue layers.

The use of VNC diameters (or stenosis), skull base height, and long bone lengths and widths is based, along with the study of adult height, on the common proposition that adult size variation is attributable fundamentally to growth performance during a critical, early period. Clark *et al.* (1986), for example, argue that the VNC dimensions are stabilized by about four years of age, and thus reflect early health conditions, in fact, mirroring strongly the conditions of neural and lymphatic organ development. They have linked VNC diameters to decreased longevity in an archaeological population and have suggested its use via modern xerography as a way to predict adult morbidity and mortality in living populations. Whereas this method is promising in theory, research has not established unambiguously the time of development of VNC dimensions, the potential to change VNC dimensions after childhood, or the specificity and sensitivity of the VNC to common stressors. The same unknowns and limitations apply to skull base height and other osseous measurements. Finally, whereas a great deal is known about the sensitivity and specificity of height to undernutrition and disease stresses, there is still much debate as to the ubiquity and degree to which catch-up in height occurs (see, for contrasting views, Martorell *et al.*, 1990, and Golden, 1994), and by extension the validity of adult height as a measure of conditions during infancy and childhood.

Harris lines, or growth arrest lines, are transverse lines of increased radiopacity seen on X-rays, particularly of tibia and other long bones (Martin *et al.*, 1985). The position of the lines reflect the size of the bone shaft at the time of formation, and from this datum one can extrapolate developmental age at time of line formation (Martin *et al.*, 1985; Magennis, 1990). It was also once assumed that these lines reflected periods of relatively acute stress. If these propositions are true, and if lines infrequently remodel and resorb, then a chronological record of physiological perturbations might be obtained in an adult by locating lines on long bone radiographs. Unfortunately, Harris lines do resorb, and even more unfortunately, Harris lines do not appear to be valid measures of stress (Martin *et al.*, 1985). Magennis (1990), for example, finds little association with childhood disease, and shows that Harris lines are more (not less) frequent during periods of rapid growth in length. Thus, there are essential reasons to suggest that bone measurements, including heights, are not the clearest possible windows into past conditions. None the less, a few possibilities do exist and they should be further evaluated.

Linear enamel hypoplasias

Another method of chronologically assessing early life stress is found in the record of disturbed enamel quality and quantity. Two unambiguous and relatively unique advantages of enamel as a tissue are that it is easy to visually inspect and it does not remodel. Enamel is essentially inert once formed and calcified; 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 which occurred during its time of development (Kreshover, 1960; Sarnat & Schour, 1941; Via & Churchill, 1959).

Linear enamel hypoplasias (LEHs) are a class of developmental enamel defects (DDE). They are seen visually as circumferential areas of decreased enamel thickness (Goodman & Rose, 1990, 1991). They are the direct result of a disruption in ameloblastic matrix secretion (Goodman & Rose, 1990), a conclusion that is over a half century old (Sarnat & Schour, 1941). Enamel hypoplastic defects may be due to hereditary conditions, localized trauma, or systemic disruption (stress) (Pindborg, 1970, 1982). Hypoplastic defects which are due to systemic disruptions (chronological enamel hypoplasias) may be distinguished from those which are the result of other factors (Goodman & Rose, 1990; 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 & Schour, 1941; Yaeger, 1980; also see Figure 11.1).

The precise aetiology of enamel hypoplasias is unknown. A long list of environmental, nutritional, physiological and hormonal conditions have been aetiologically linked to LEH formation (Goodman & Rose, 1990). Work with contemporary children in Mexico, for example, shows that LEH is more common in families with less material wealth and is predictive of decreased growth status (height-for-age and weight-for-age) (Goodman *et al.*, 1992). Another study, comparing supplemented and non-supple-

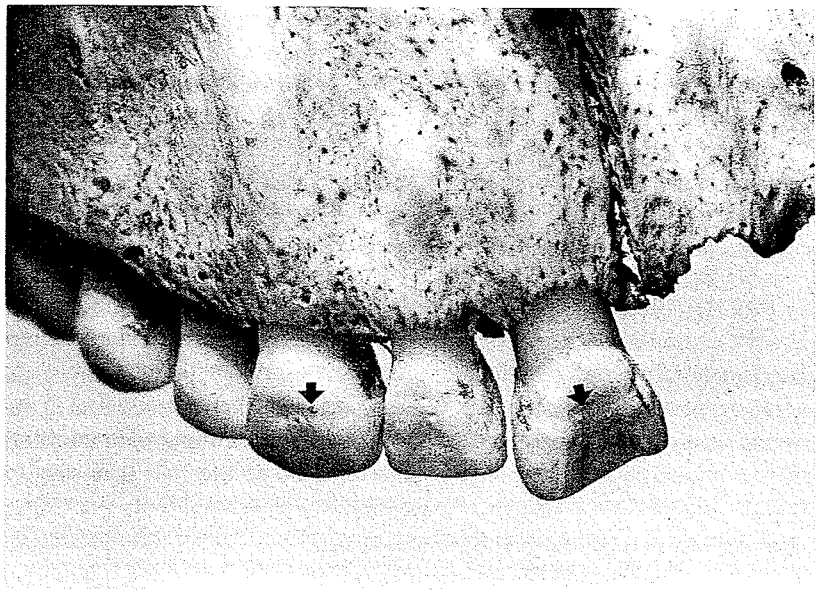

Figure 11.1. Chronologic enamel hypoplasias (stress-hypoplasias) on right maximally central incisor and canine (arrow). Both of these shallow hypoplastic bands developed around 3.5 year developmental age, based on the degree of crown completion (enamel apposition begins at the occlusal tips which are slightly worn). The common estimated age at formation suggests that these defects were the result of the same systemic physiological perturbation (stress).

mented children, found that enamel defects are nearly twice as common in non-supplemented children (Goodman, Martinez & Chavez, 1991). Thus, a working 'threshold' model has been proposed in which enamel defects formation commences when a physiological threshold is passed, and enamel matrix formation stops (Figure 11.2). The threshold is influenced by nutrient intake, concurrent morbidity, and 'unknown aetiological factors'.

The aetiology of LEH may be seen as very similar to the aetiology of linear growth retardation. The differences are that enamel disruption is more of a discontinuous, threshold variable. Another difference, of potential importance in considering long-term consequences, is that enamel is an epithelial tissue and its disruption may be concurrent with the disruption in development of other epithelial tissues. For example, Jaffe *et al.* (1985) have shown that individuals with idiopathic brain disorders are much more likely to also have disrupted enamel development than normal

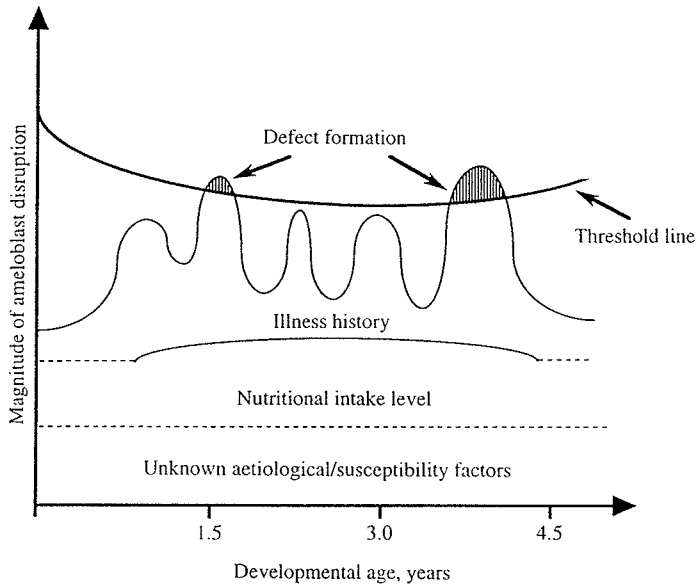

Figure 11.2. Threshold model for the formation of a linear enamel hypoplasia (LEH). Enamel apposition and LEH formation occurs when ameloblasts temporarily cease secretion of enamel matrix. This growth stoppage has been associated with a wide variety of stressors. It is suggested that there is a threshold at which aneloblasts will cease function. This threshold may be reached by a combination of unknown etiological factors, chronic or acute undernutrition and acute morbidity.

controls. This association points to a common physiological perturbation during early gestation that affected both epithelial tissues.

Prior studies of LEH and adult mortality

A few sporadic studies have examined the association between LEH and future mortality. The earliest of these was a study of living Chinese (Anderson & Stevenson, 1930). These researchers found that enamel defects were far less common in older cohorts. They attributed this finding to a selecting out of individuals who were stressed early in life. Unfortunately, it is not clear what specific type of defect they were observing (it is called 'mottled enamel').

The first palaeontological/archaeological data suggesting decreased longevity with childhood LEH comes from the late 1970s. White (1978) assessed hypoplasias on permanent maxillary first molars from South African Plio-Pleistocene Australopithecines (ca. 1.5–3.0 million years before

present). He noted that individuals with maxillary first molar hypoplasias from the Swartkrans site ($n=6$) had 'lower-than-expected' ages at death. These individuals died between four and thirteen years of age, while individuals with non-hypoplastic first molars ($n=110$) died between eight and thirty-one years of age. Using White's estimated ages-at-death, the mean age at death of individuals in these groups has been calculated as 7.8 and 19.6 years, respectively. Although this study suffers from a small sample size and lack of precision in assigning ages of death to fragmentary palaeontological materials, the data none the less demonstrate a dramatic, nearly 12-year decrease in life expectancy associated with LEHs.

Cook and Buikstra (1979) similarly compared 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 postnatally developing dental defects are associated with decreased longevity during both the Middle and Late Woodland periods.

Rose, Armelagos, and Lallo (1978) histologically studied areas of disturbed enamel formation (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, the average age at death of the 21 individuals with Wilson bands is 26.7 versus 42.1 years in the 66 individuals without Wilson bands. In other words, individuals with a record of early life stress (Wilson band) lived 15.4 years less than those without such a record.

Most recently, Duray (1994) has studied the association between age at death and developmental enamel defects at Libben, a large archaic cemetery in Ohio. Duray finds that individuals with developmental defects also die earlier, by about 5 to 6 years, depending on the type of defect, defect severity, and defect location.

These results vary by population studied and means of assessment of enamel developmental defects. However, of greatest note is that all studies, despite methodological and populational differences, suggest a measurable decrease in longevity associated with early life stress. This association, and the potential biocultural processes that might underlie it, are explored in greater detail in the following two case studies.

Dickson mounds: LEH, cultural change and life expectancy

Dickson Mounds is a multicomponent habitation-burial complex located near Lewiston, Illinois. The mounds are associated with three cultural horizons: Late Woodland (LW), Mississippian Acculturated Late Woodland (MALW), and Middle Mississippian (MM) (Harn, 1971, 1978, 1980). During the Late Woodland period (ca. AD 900–1050) the area was occupied by a relatively small (75–125) and semisedentary 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. AD 1050–1175) is a transitional period, possible 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. AD 1175–1300) the Mississippianization of local populations becomes complete with the culmination of trends toward extended and intensified trade networks, increased population density, size, and sedentism, and 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 *et al.*, 1984*b*). Porotic hyperostosis, an indication of iron deficiency anaemia, is four times as prevalent among MM subadults as compared to LW subadults (64% to 16%) (Lallo, Armelagos & Mensforth, 1977). Periosteal infections in subadults increase from 27% in the LW to 81% in the MM (Lallo, Armelagos & Rose, 1978), and the frequency of enamel hypoplasias doubles in adults and adolescents (Goodman, Armelagos & Rose, 1980). Life expectancy is lower at all age intervals in the MM when compared to the combined LW and MALW samples (Moore, Swedlund & Armelagos, 1975). Long bone growth in length and circumference appears to be slower around 2–5 years of age and LEH and enamel histological defects are nearly twice as common in the MM as compared to the LW (Goodman *et al.*, 1980, 1984*b*).

Enamel hypoplasias were recorded on all permanent teeth except third molars in 111 adults and adolescents (50 males, 50 females, 11 adolescents of unknown sex). Hypoplasias are easily identified and were defined operationally as circumferential lines, bands, or pitting of decreased enamel thickness (Goodman *et al.*, 1980; 1984*a*) (Figure 11.1). 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 and co-workers (1941).

Each half-year period between birth and 7.0 years was rated as either stress-positive or stress-negative depending on the identification of LEHs on areas of teeth developing during these time periods. 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 *et al.*, 1980, 1984*a*).

Table 11.1. *Comparison of mean ages at death for individuals by cultural horizon and number of hypoplasias-stress periods between 3.5 and 7.0 years developmental age*

	Sample size	Mean	S.D.	1-way ANOVA (F-ratio)	*A priori* contrasts (A vs. B)	*A priori* 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 $p \leqslant .10$.
[b] 2-tailed $p \leqslant .05$.
[c] 2-tailed $p \leqslant .01$.
[d] 2-tailed $p \leqslant .001$.

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 *et al.*, 1984*a*). However, all individuals yielded a complete record of stress-hypoplasias for the seven half-year periods from 3.5 to 7.0 years.

All individuals in the LW sample have either one or no hypoplasias-stress periods between 3.5 and 7.0 years (Table 11.1). 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 11.1; F-ratio = .35; Goodman & Armelagos, 1988).

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 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 stress periods (Table 11.1, Figure 11.3).

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 11.3). 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 11.1; $p < .01$).

The specificity of differences between hypoplasia groups was tested with a series of *a priori* contrasts (Table 11.1). 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 periods in the total sample (Table 11.1; Figure 11.3). The overall mean age at death of individuals without hypoplasias-stress periods is 35.8 years; 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).

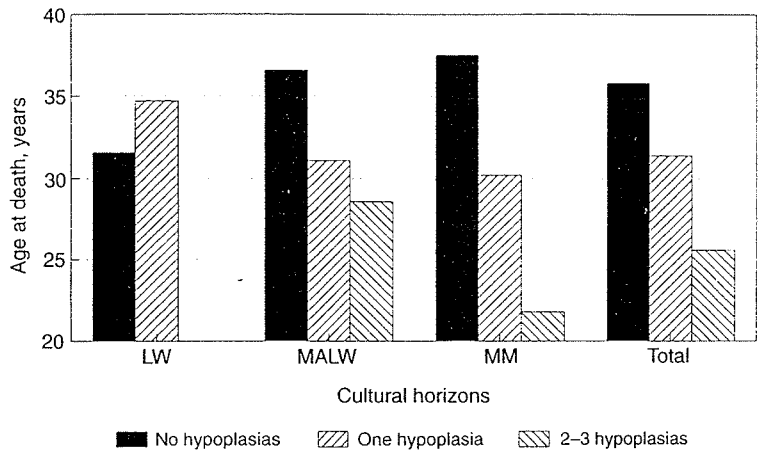

Figure 11.3. Mean ages at death of Dickson Mounds adolescents/adults by number of hypoplasias-stress periods between 3.5 and 7.0 years developmental age. LW = Late Woodland, MALW = Mississippian Acculturated Late Woodland, MM = Middle Mississippian.

There are at least three processes which 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 be causative of 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 frailty or a 'weaker constitution', and the sum effect is earlier death.

Secondly, 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 disfunction (Chandra, 1975; Miler, 1982).

Thirdly, these data may result from differential lifelong patterns of behavioural and culturally based exposure to stressors. An increased lifelong potential for exposure to stressors may be causative of both an increased frequency of childhood stress and earlier ages at death.

It is not possible to rule out any of these processes. All may contribute to the associations which 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 *et al.*, 1984*a*). Furthermore, the greatest difference between stressed and non-stressed 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 the importance of lifelong differences in social status and concomitant differential cultural buffering from stress. Unfortunately, it is difficult to assess cultural buffering in archaeological 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 & Armelagos, 1983). Whereas individuals with no grave goods are more likely to have multiple hypoplasias (17.6%) as compared to individuals with no non-utilitarian offerings (8.7%), their relationship does not explain the association between hypoplasias and age-at-death (Goodman *et al.*, 1983). However, the inability of 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 aetiology of childhood stress and adult mortality.

Hammon–Todd: LEH, cause of death and longevity

The relationship between LEH and adult morbidity has also been studied in the Hammon–Todd Osteological Collection. The advantages of this historical collection as compared to archaeologically derived collections include the availability of data on country of origin, documented age at death, and documented cause of death (from death certificates).

The Hammon–Todd collection, housed at the Cleveland Museum of Natural History, is comprised of individuals who died during the earlier part of this century. They are from the lower socioeconomic segment of the greater Cleveland population. The sample selected is limited to individuals over 18 years of age with birth certificates or other documents which reliably establish birth dates and, therefore, age at death. Additional selection was made for individuals with at least four different anterior teeth with minimal attrition. The study sample consists of 185 individuals who were born between 1852 and 1912 and died between 1915 and 1931. The mean age at death is 37.7 years (range 18 to 67 years).

Defects were recorded on the anterior teeth, either on the left or right side, depending on which was best preserved. As in the Dickson Mounds study, presence or absence of the tooth was scored, estimation was made of enamel lost to study through attrition or other destructive process, and the

Table 11.2. *Comparison of mean ages at death of individuals with and without linear enamel hypoplasias on mandibular anterior teeth*

Tooth	No LEH Mean (S.D.)	LEH Mean (S.D.)	F Ratio	P 2-tail
I_1 (1–4 yrs)*	38.1 (11.4) $n=84$	33.4 (11.3) $n=43$	4.42	.037
I_2 (1–4 yrs)*	38.4 (11.7) $n=107$	34.3 (11.8) $n=39$	3.35	.069
C (2–6.5 yrs)*	37.8 (11.1) $n=74$	35.0 (11.9) $n=81$	2.74	.098

* Years in parenthesis refer to the approximate ages covered by the enamel portion studied.

location of defect was used to estimate the individuals' age at formation of the defect (Goodman & Armelagos, 1988).

Individuals with LEH have a significant decrease in life expectancy (Table 11.2). For example, for the mandibular anterior teeth, there is between a 2.8 (canine) and a 4.7 (central incisor) year decrease in life expectancy with one or more LEHs (Note that the estimated beginning period of development of each tooth has been excluded due to attrition and that the teeth span different developmental periods.)

Table 11.3 provides an analysis of the relationship between mean ages at death of individuals with and without LEH by three annual periods on the mandibular central incisor. Individuals with LEH between 1 and 2 years live slightly, though insignificantly, longer. However, in comparing individuals with and without LEHs between 2 and 3 years, and 3 and 4 years, one finds that individuals with LEH die at a mean earlier age. Individuals with LEH between 2 and 3 die at a mean age of 33.4 years, while those without LEH between 2 and 3 die at a mean age of 39.1 years. This 5.7 year difference is significant at the 5% probability level. Similarly, individuals with LEH between 3 and 4 die at a mean age of 29.6 years, while individuals without LEH between 3 and 4 die at a mean age of 38.3 years (Table 11.3).

The relationship between age at death and age at formation of a LEH is further shown in this comparison of the frequency of LEH by time of occurrence for individuals who died before 30 years of age and those who died after age 30. Differences in the frequency of LEH between these two groups are insignificant up to two years of age. However, between 2.0 and 2.5 years, nearly three times as many of the under-30 group have LEHs as compared to the over-30 group. This increased incidence of defects continues to at least four years of age or the end of the tooth crown's

Table 11.3. *Comparison of mean ages at death of individuals with and without linear enamel hypoplasias by yearly developmental zones on the mandibular central incisor*

I_1	No LEH Mean (S.D.)	LEH Mean (S.D.)	F Ratio	P 2-tail
1–2 years	36.2 (11.4) $n=114$	38.5 (14.6) $n=13$	0.47	ns
2–3 years	39.1 (12.0) $n=113$	33.4 (11.4) $n=33$	5.90	.016
3–4 years	38.3 (11.8) $n=142$	29.6 (12.4) $n=8$	4.01	.047

development. Even though the first year is a period of rapid brain size increase, these data suggest that time periods after the first year can have consequence for longevity (Figure 11.4).

Lastly, mean ages at death are compared for individuals with and without LEH and divided into four common causes of death: infectious disease (usually tuberculosis), heart disease, cancer, and alcoholism (Table 11.4). Sample sizes are quite small. None the less, it is interesting that the greatest effects are seen in the infectious disease and alcoholism groups. Individuals who died of an infectious disease and had a hypoplastic defect died at a mean of 31.2 years, compared to 35.1 years for those who died of an infectious disease without a LEH (differences = 3.9 years). Individuals who died of alcoholism and with a LEH died at a mean age of 31.5 years, or 7.9 years before those who died of alcoholism but without a LEH (Table 11.4). These data suggest that decreased survival with adult infections is perhaps related to weakened immunity or bouts of infectious disease in early life, as evidenced by LEH.

Implications and conclusions

The data presented suggest that metabolic challenges (stresses, perturbations) around ages two to seven that are severe enough to cause a disruption to amelogenesis (and a LEH) are consistently associated with earlier ages at death. This 'postweaning' period is a relatively unexplored time period, and one in which consistent health records are hard to locate consistently.

The main question deriving from the archaeological studies concerns the biocultural processes by which these diverse phenomena are associated. The Dickson data most strongly supports the cultural buffering hypothesis. However, this mechanism is not exclusive, and the data on the association between LEH and idiopathic brain damage suggest that some form of wear

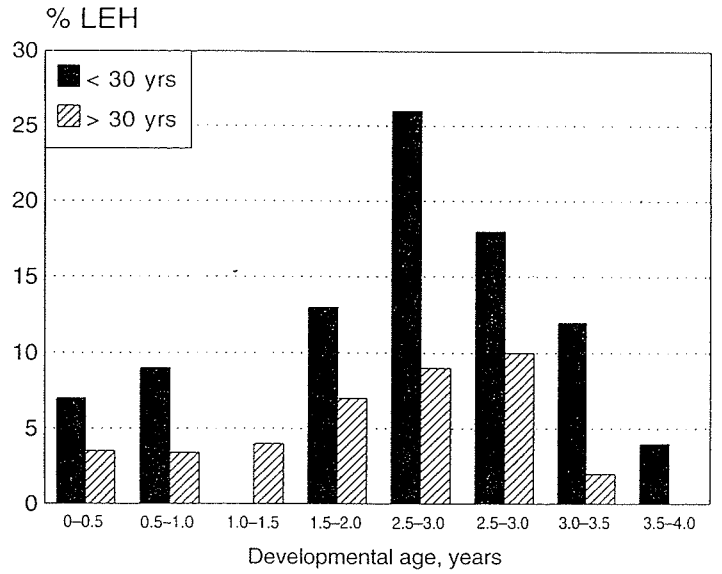

Figure 11.4. Comparison of the percentage of linear enamel hypoplasias (LEH) by half-year developmental periods for individuals who died before and after their thirtieth birthday.

and tear or biological damage mechanism might also be implicated. This is also consistent with the infectious disease results from the Hammon–Todd study.

LEH appears to be an effective tool for studying the longitudinal pattern of stress, morbidity, and mortality in past and living populations. It seems to be sensitive to developmental disruptions, is associated with a number of types of stress, is indelible and its age at formation can be reliably estimated.

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. Whereas this paper has focused mainly on methodological questions, the data beg further elaboration of theory and models that might help to better contextualize data linking early developmental events and adult health.

Table 11.4. *Comparison of mean ages at death of individuals with and without linear enamel hypoplasias on the mandibular central incisor divided by cause of death (COD)*

I_1	No LEH Mean (S.D.)	LEH Mean (S.D.)	F Ratio	P 2-tail
Infectious	35.1 (10.7) $n=48$	31.2 (9.5) $n=30$	2.65	ns
Coronary	43.9 (14.6) $n=16$	41.3 (18.3) $n=6$	0.12	ns
Accident	39.7 (10.4) $n=13$	37.8 (10.2) $n=5$	0.13	ns
Alcoholism	40.4 (7.4) $n=7$	31.5 (4.9) $n=2$	2.45	ns

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12 *The childhood environment and the development of sexuality*

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Introduction

There is an old tradition in biosocial research on sexual behaviour of exploring correlations between aspects of physical development (most often puberty) and aspects of social and sexual development. When relationships are found, they are usually presented as evidence for a common-sense view that the timing of puberty determines the beginning of sexual interest and so the start of sexual behaviour (e.g. Hurlock, 1995). These, in turn, it is suggested, lead to the establishment of long-term relationships and marriage. However, many social scientists reject such biological determinist views of social and sexual development and relationship formation. Their approach to love, sex and marriage has usually been very much more social problem orientated, at least in the quantitative survey traditions, and has been dominated by studies of teenage pregnancy and contraception (e.g. Miller & Moore, 1990). The chief concern has been to see who begins to do what, when, and with what consequences (principally conception) (see Griffin, 1993). Between these two approaches, there is very little of what might be termed a developmental understanding of how young people enter the world of adult sexual activities and relationships. One might expect this gap to be filled by developmental psychology. But examination of textbooks of this field reveal a curious silence. There are almost no accounts of the development of sexuality. Instead, we find discussions of gender identity and often an account of the physical and physiological changes that occur at puberty. The implicit assumption of this approach seems to be that having established a male or female gender identity, sexual behaviour will emerge as an inevitable result of the biological changes seen at puberty. So here, once again, we have another version of biological determinist model which has been the biosocial tradition.

The absence of any elaborated theory of sexual development encourages