# Infant and childhood morbidity and mortality risks in archaeological populations

Alan H. Goodman and George J. Armelagos

## Introduction

Our understanding of the impact of disease on prehistoric groups has increased significantly in the last decade (Larsen 1987). We can evaluate the impact of socio-economic and environmental changes and political organizations on the health of archaeological populations, and we are able to determine the subgroups within a population that are most at risk. In studies of archaeological populations that have experienced shifts in subsistence, infants and children appear to be at greater risk than other segments of society (Goodman et al. 1984a; 1984b), a pattern that is consistent with studies of many contemporary groups (Preston 1980; Wood 1983). Deaths of infants and children (those under five years) in less developed countries currently account for 40 per cent of the world's total burden of mortality (Dyson 1984) and the handicap of morbidity and suboptimal functioning is focused on this group to a similar, disproportionate level (Soedjatmoko 1984). The same burden of mortality and morbidity was likely to have been placed on those under 5 years in prehistory.

In this paper we provide an overview of developments in method and theory relating to the paleoepidemiological study of infants and children, those under five years of age. First, we will provide a model or framework for studying health in archaeological populations, with special reference to infants and children. We then briefly review common indicators of health which relate to infants and children, and provide illustrations of the use of these indicators drawn from ongoing research with human skeletal remains from Dickson Mounds, Illinois, USA and Wadi Halfa, Sudanese Nubia. Finally, we address the possible functional and adaptive consequences to the population and culture of this group's health.

## The model

To examine the broader effects of disease and death to those under five years of age, we have used a model that considers the potential insults that may have an impact on this age group. Goodman and co-workers (Goodman et al. 1984b) have constructed a model that has been successfully applied to a number of archaeological populations.

*World Archaeology Volume 21 No. 2 Archaeology of Public Health*
 0043–8243/89/2102/225 $3.00/1

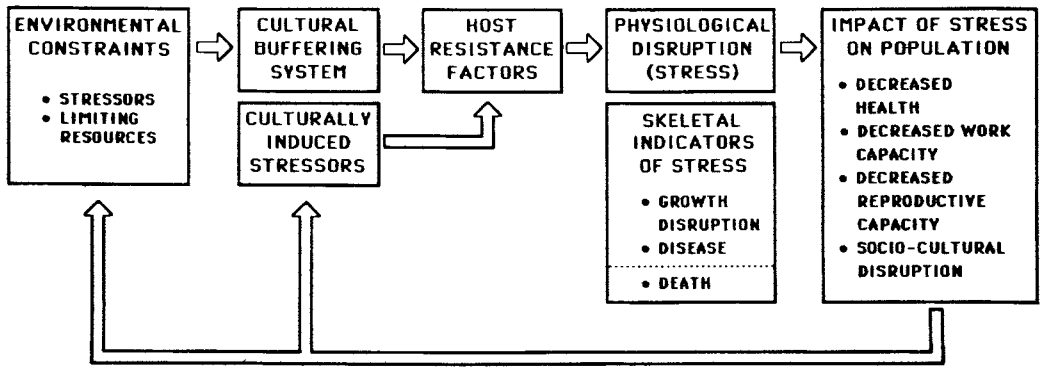


*Figure 1* A general model for the study of stress in skeletal populations (modified from Goodman et al. 1984b).

This model, which has been modified in this paper (Fig. 1), provides a systematic framework for analysis of how insults, which can cause physiological disruption, potentially affect the individual and the population.

In this model, the environment is the source of both the resource necessary for survival and the stressors that are adversely affecting adaptation. Culture mediates the process of extracting the necessary materials from the environment through its technological, social and ideological systems. The cultural system can act to buffer the individual and population from stressors. There are numerous examples of cultural practices which act to protect individuals from environmental stressors. Clothing and shelter are obvious responses to cold ambient temperatures. However, all stressors are not buffered and some of these may pose significant harm to the adaptation of individuals and the group.

Cultural systems can also generate significant stressors. Thus, if stressors from the environment are not buffered, or if stressors are culturally induced, then the individual and the population will need to respond. If the individual has the ability to resist the stressor, then it is not likely to have a biological impact. But if the individual lacks the resources and reserves to combat these stressors, then these stressors are likely to cause physiological disruption (biological stress response).

Because of both biological and cultural differences in the availability of and access to resources and reserves, not all individuals are equally at risk. Some individuals, such as female infants and children, may have less access to food and other culturally distributed resources than their male age-peers (Chen et al. 1981). Although young girls may generally be more biologically resistant to stresses during development than young boys (Stinson 1985), decreased allocation of food may tip the balance in favor of boys (Chen et al. 1981).

Certain segments of the populations may also be at greater risks because their biological requirements are not matched by biological resources. Newborns, for example, are born with very immature immune systems (Chandra 1975; Cooper and Lawton 1974). They must rely on immunity conferred during their time in utero and transferred via breast feeding. Because of their state of biological immaturity, infants are frequently unable to rally from stressors that have only mild effects on a more

mature individual. For these and other reasons, mortality is particularly high during the first year in many communities (Mosley 1984; Preston 1980).

Once weaning begins, a second peak in both morbidity and mortality is frequently seen (Gordon et al. 1963; 1967). Infants and young children become dependent on their own natural defenses at a time when these defenses are just beginning to develop. If nutrition is inadequate, as it frequently is at this age, then these defenses will be further hindered. Thus, it is not unusual to see weaning age infants and children undergoing repeated bouts of chronic diarrhea, upper respiratory disease, and malnutrition (Mata et al. 1971; McNeish 1986).

Sometimes these infants and children 'rally' from these bouts of ill health and make it through this dangerous period. Despite the recovery, however, the repeated insults may have a lasting adaptive cost in terms of such functional abilities as growth, reproduction, activity patterns, cognition, behavior, and social performance (Allen 1983; Haas and Harrison 1977). Finally, all too frequently these infants and children do succumb to these repeat insults. Despite all our advances in knowledge and material wealth, it is still all too common to find that the chances of survival are less than 50 per cent in some developing areas of the world (Dyson 1984). There is little reason to suspect that survival was greater in prehistoric populations.

Although the record is far from complete, many of these bouts of stress leave markers on bone and teeth. These markers, which are briefly reviewed in the following section, can be used to reconstruct the history of morbidity and mortality experiences during infancy and childhood. From this record of the type, severity, frequency, and distribution of ill health we can begin to draw inferences about the functional and adaptive effects of illness on the individual and the society.

As anthropologists interested in the lives of individuals and groups, it is important to realize that disease and death may have important functional and adaptive consequences for both individuals and the groups to which they belong. For example, it has been demonstrated that subadult (under 15 years) mortality would not be as disruptive to the productive and reproductive potential of the population as would a similar level of mortality to the adult segment of the population (Armelagos and Goodman 1988). Subadults have received the least cumulative investment and are most quickly replaced in a demographic sense. None the less, even if survived, stresses during infancy and childhood may have lasting effects on functional capacities and resistance to disease (Baker and Osmond 1986a; 1986b; Chandra 1975) and may increase future mortality (Chen et al. 1980). The net effects on a culture of increased morbidity and mortality to subadults can severely hinder ability to maintain resources and flexibility to meet future challenges (Allen 1983).

## The evidence of stress from skeletal remains

Bones provide support for our muscle structure, protect the vital organs such as the brain and the orbits, produce the red blood cells and maintain the chemical balance in the body. When these functions are interfered with, it may be evidence of physiological disruption or disease (Huss-Ashmore et al. 1982)

The response of the human skeleton to stressors is deceptively simple because bone is limited in its potential responses. Osteons, the building unit of bone, can be formed or they can be resorbed, or they can incorporate both processes. The interpretation of these responses, while difficult, can reveal a great deal about the stressors to which an individual was exposed.

Many diseases leave specific marks or 'signatures' on bone. Tuberculosis, syphilis, and leprosy result in such characteristic skeletal changes that these alterations can be diagnostic. For example, in severe cases of tuberculosis there is often a collapse of the vertebral body and resorptive lesions in other parts of the skeleton. Such changes may lead a pathologist to a diagnosis of tuberculosis (Ortner and Putschar 1984; Steinbock 1976).

There are many pathogens, however, that leave only generalized changes in the skeleton. For example, we often find a hyper-reaction of the outer, periosteal bone that reflects a pathogenic change due to inflammation. These 'non-specific' periosteal lesions involve a roughened appearance of the outer periosteal bone. A variety of micro-organisms, including staphylococcus and steptococcus, can cause these changes.

Unfortunately, there are many pathogens, viruses in particular, that leave no evidence of their virulence on bone. These pathogens cause harm to soft tissue only, or work so quickly that the hard tissues (bones and teeth) do not have enough time to respond to the stressful presence of the microbe. Because of the possibility of this type of dynamic, Ortner (1989) has suggested that a periosteal reaction may actually indicate a positive outcome, namely that the individual has had time to set forth an appropriate biological response to infection. While this point is well taken, and the issue is one that requires further study and debate, we contend that such severe responses indicate problems that are more likely to have a negative effect on adaptation (Goodman et al. 1984a; Goodman and Armelagos 1988).

Nutritional deficiencies can also leave specific lesions which are easily diagnosed from bone. Deficiencies of vitamin D during bone growth (rickets) result in a constellation of characteristic changes. Similarly, vitamin C deficiency (scurvy) leaves unique changes, though these are more difficult to diagnose in prehistoric remains (Huss-Ashmore et al. 1982; Larson 1987; Martin et al. 1985; Wells 1975).

A major breakthrough in analyzing nutritional disease resulted from a movement away from using single indicators of stress to an approach that considers multiple indicators which are systematically analyzed to provide an understanding of nutritional stress (Buikstra and Cook 1980; Huss-Ashmore et al. 1982). This movement came with the realization that undernutrition almost always involves multiple nutrients, and its effects are widespread and general (Huss-Ashmore et al. 1982) When examined together, a number of lesions and growth indicators can provide clues to the pattern of nutritional deficiency.

## Measures of morbidity and mortality in infants and children

### *Growth and development*

Long bone growth, may provide information about an individual's past physiological state. Since we are by necessity using cross-sectional data, comparison with longitudinal growth studies are very difficult. Furthermore, there are few growth standards from peasant agriculturalists so it is often necessary to use growth standards developed from children in industrialized nations. Even with these difficulties, however, we can often see indications of growth retardation. Comparing the growth of infants and children from subgroups within a population or time successive groups may be particularly useful for understanding variations in health and adaptation (Goodman et al. 1984a)

The use of histological techniques has recently provided an additional tool for analyzing the impact of nutritional deficiencies on bone growth and maintenance. Microscopic analysis of cross-sections of femora reveal that some children have very thin cortical bone (Huss-Ashmore 1981). By examining the percentage of cortical bone for each individual, and comparing it with an age matched sample, we can determine those individuals experience nutritional difficulties. It is even possible to ascertain if the bone loss is the result of a lack of osteonal deposition or an increase in bone resorption (Martin and Armelagos 1979).

Finally, there are two additional methods that can be used to identify disruptions to normal growth and development. Harris lines (lines of increased radiopaque density) found by radiographic analysis have been used as an indicator of growth arresting and recovery (Park 1964) (Fig. 2). Harris lines can also be used to assess the age at which an individual experienced growth disruption and subsequent recovery. Since growth occurs at both ends of a long bone, Harris lines will maintain their relative position to the midshaft. If a researcher knows the relative growth rates of the proximal and distal portion of the long bone, then the age at which the line developed can be determined (Goodman et al. 1984a).

The analysis of defects in dental enamel provides another measure of growth disruption. Dental enamel hypoplasia (Fig. 3) is a quantitative deficiency in enamel thickness resulting from a disruption in enamel matrix formation (Sarnat and Schour 1941). Enamel defects can result from systemic disruption, hereditary conditions, or localized trauma (Yaeger and Sharawy 1986). Since systemic disruptions are likely to affect more than one tooth, we use the occurrence on multiple teeth as the criteria for assessing a systemic cause (Goodman et al. 1980). Unlike bone, once enamel matures it can not be remodeled. Enamel is secreted in a regular ring-like pattern and the crown development provides a permanent chronological record of any physiological disruption

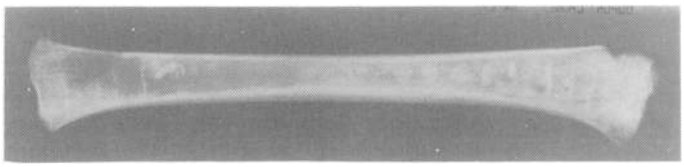

*Figure 2* Radiograph of a Harris lines in the distal femur of a Native American.

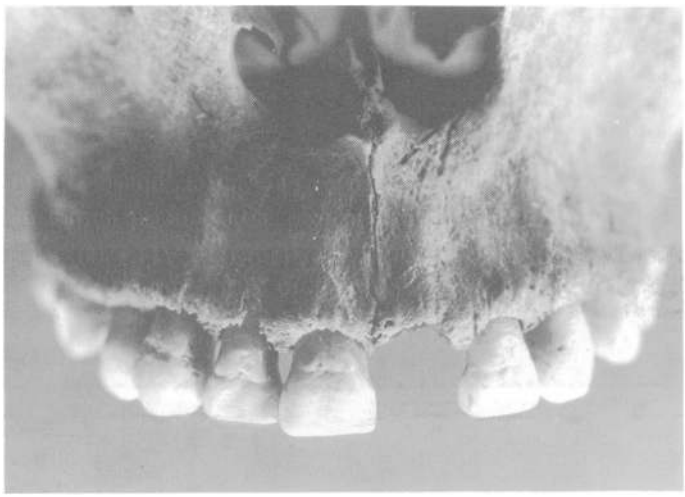

*Figure 3* Enamel Hypoplasia on the upper, anterior teeth of a Native American.

(Sarnat and Schour 1941; Massler et al. 1941). An understanding of the time and rate of enamel formation allows one to define the developmental age at which the metabolic disruption occurred.

One can examine the chronological pattern of hypoplasia. In adults, this can provide a record of the age between birth to six years (roughly the time of development of permanent tooth crowns) during which they experienced physiological stress of a degree severe enough to disrupt amelogenesis. In subadults with deciduous dentition, one can ascertain the chronology of hypoplasias (stress) during the last two trimesters of pregnancy and through the first year of life (the time during which deciduous tooth crowns develop). Blakey and Armelagos (1985) have found a peak in hypoplastic defects near the end of gestation/time of birth for Dickson Mounds subadults. Storey (1988) finds a similar peak for subadults from Teotihuacan, Mexico, but a later peak for subadults from Copan, Honduras.

One can also evaluate the impact of this disruption on other aspects of the individual's health. As enamel hypoplasias are relatively permanent, one can, for example, evaluate the relationship between stresses during early life (indicated by the presence of an enamel hypoplasia) and health and mortality in adulthood. Do adults who were stressed as children suffer from other insults and do they live as long as those who were not stressed?

### *Porotic hyperostosis*

Porotic hyperostosis, which most frequently affects the cranium and the roof of the orbits, can be used to diagnose iron deficiency anemia (Fig. 4). As the name implies, these lesions have a very porous, coral-like appearance, which develops when the diploe (the trabecular portion of the cranial bone that separates the inner and outer surfaces) expands. With the expansion of the diploe, the outer, periosteal layer of bone becomes thinner. It may eventually disappear exposing the porous trabecular bone (diploe).

*Figure 4* Porotic hyperostosis (cribra orbitalia) of the orbital surfaces.

The expansion of the diploe can be caused by any anemia that stimulates red blood cell production. While there are a number of anemias (sickle cell anemia, thalassemia, iron deficiency) that can cause these changes, the relatively minor manifestation of the lesion (on the cranial surface and roof of the orbits) and its high frequency in children and young adult females suggests iron deficiency as the most likely cause (Mensforth et al. 1978).

*Mortality (life expectancy)*

There has been a long standing debate over the accuracy of life tables, derived from skeletal assemblages, for the reconstruction of the mortality pattern of archaeological groups (Buikstra and Mielke 1985). This debate, however, should not obscure the general agreement that death is the ultimate indicator of failure to adapt. Death may be fruitfully viewed as the end result of an accumulated set of biological, behavioral and cultural challenges to the individual.

While there is a great deal of controversy over the best methods for analysis of mortality, many still favor the use of life tables. This is especially true where there is strong evidence to suggest that the death assemblage is an accurate cross-section of the former population (Buikstra and Mielke 1985).

**Dickson Mounds, USA**

The changes in subsistence from AD 950 to AD 1300 in the Dickson Mounds (Lewiston, Illinois) area were profound. In the period from AD 950 to AD 1200, there was a shift from a Late Woodland adaptation, characterized as a general gathering-hunting strategy, to one which emphasized agriculture. The latter phase of this

*Table 1* Frequency of infectious lesions (periostitis and osteomyelitis) and porotic hyperostosis[a]

| | | *Postcranial infectious lesions* | | *Porotic hyperostosis* | | *Porotic hyperostosis and infectious lesions* | |
|---|---|---|---|---|---|---|---|
| *Dickson population* | *N* | *N* | % | *N* | % | *N* | % |
| Late Woodland | 44 | 9 | 20.5 | 6 | 13.6 | 3 | 6.5 |
| Mississippian-acculturated Late Woodland | 93 | 45 | 48.4 | 29 | 31.2 | 20 | 21.5 |
| Middle Mississippian | 101 | 74 | 73.3 | 52 | 51.5 | 41 | 40.6 |
| Total | 238 | 128 | 53.8 | 87 | 36.5 | 64 | 26.9 |

[a]Modified from Lallo *et al.* (1977:479).

development (Mississippian Acculturated Late Woodland) was a period in which maize agriculture becomes established at Dickson Mounds. From AD 1200 to 1300 there may have been a further intensification of agriculture in what has been called the Middle Mississippian period. How did this change affect subadult health?

When these earlier groups from Dickson Mounds are compared to the Middle Mississippian people there is evidence of a remarkable deterioration in health. In this short period there was a four-fold increase in iron deficiency anemia (porotic hyperostosis) and a three-fold increase in infectious disease (periosteal reaction) (see Table 1). The frequency of individuals with both iron deficiency and infectious lesions increases from 6 per cent in the Late Woodland period to 40 per cent in the Middle Mississippian period. Furthermore, individuals with both conditions display a synergistic disease interaction (Lallo et al. 1977; Lallo et al. 1978). That is, individuals with both lesions show a more severe manifestation of each condition.

The relationship of infection and anemia in children can be ascertained by examining the age of onset and distribution of the lesions by age. The infectious lesions in children under ten years of age peaks during the first year, while the porotic lesions are most prevalent during the second and third year (Table 2). This pattern suggests that

*Table 2* Age specific frequency of porotic hyperostosis for the combined samples from Dickson Mounds[a]

| | | *Porotic hyperostosis* | |
|---|---|---|---|
| Age | *N* | *N* | % |
| 0–0.9 | 79 | 16 | 20.3 |
| 1–1.9 | 45 | 13 | 28.9 |
| 2–4.9 | 43 | 20 | 46.5 |
| 5–9.9 | 45 | 28 | 62.2 |
| 10–14.9 | 26 | 10 | 38.5 |
| Total | 238 | 87 | 36.6 |

[a]Modified from Lallo et al. (1977:478).

individuals who do survive the infectious pathogen may have problems in maintaining adequate iron reserves. An increase in iron requirements, secondary to infection, coupled with a low dietary intake and bioavailability, is the most likely explanation for this pattern.

Experimental studies have shown that the bioavailability of the non-heme iron found in corn is very low (Hallberg 1981; Monsen et al. 1978). Known immune system responses to iron deficiency include abnormalities in cell-mediated immunity and ability of neutrophils to kill a variety of type of bacteria (Dallmann 1987). Pollitt (1987) suggests a variety of effects of iron deficiency on mental development. While such effects are difficult to track in prehistory, their presence needs to be considered.

One way the biological cost of infection and anemia can be determined is from an analysis of a life table constructed for individuals who died before their tenth year (Fig. 5). It is apparent that individuals with infections show a dramatic decrease in life expectancy. Those born with periosteal lesions can be expected to live less than a year. Even those suffering from porotic hyperostosis display a decrease in life expectancy of two to six months at each age group.

The impact of cultural change at Dickson can be seen in other aspects of growth and development. There is evidence of delayed growth in the long bone length and circumference of Mississippian children between their fifth through fifteenth year (Lallo, 1973; Goodman et al. 1984b). This delay may be a result of chronic stresses around 2 to 5 years of age.

The frequency and chronology of hypoplastic defects in the dental enamel of the Dickson Mounds population supports the argument that the shift to agriculture had deleterious effects on the health of the group. There is an increase in hypoplasia from 0.90 defects per individual (Late Woodland) to 1.61 per individual in the Middle Mississippian period. Individuals with one or more hypoplasias increases from 45 per cent to 80 per cent during the same period (Goodman et al. 1980; 1984b). Since the

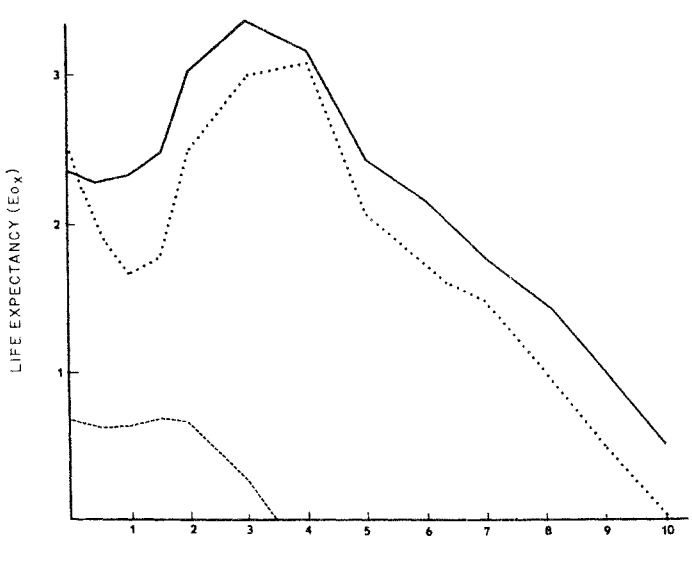


*Figure 5* Life expectancy for the Dickson Mounds population for those dying within the first ten years. Individuals with infection (- - -), or porotic hyperostosis (....), showed reduced life expectancy as compared to the total population (____).

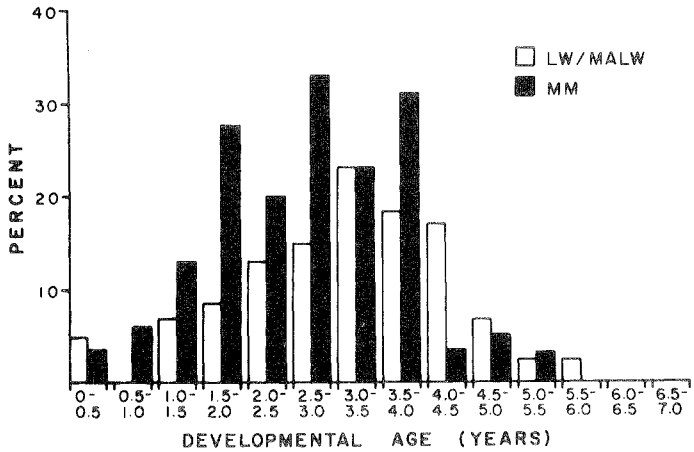


*Figure 6* Frequency distribution of enamel hypoplasias by half year periods in two Dickson Mounds populations. MM, Middle Mississippian; LW/MALW, Late Woodland/Mississippian Acculturated Late Woodland.

chronological development of the enamel is well understood, it is possible to determine the age at which the hypoplasias occurred during the life of the individual. The hypoplastic lines in adults provide a 'metabolic memory' of events which occurred during their childhood.

The chronology of enamel hypoplasia shows that the Dickson Mounds population experienced peak stress between the ages of two and four which corresponds to the period of weaning (Fig. 6). The comparison of the chronology between the earlier groups and the intensive agriculturalists at Dickson Mounds shows an earlier age of onset of hypoplasia in the later group, suggesting an earlier age of weaning.

Enamel hypoplasia is considered a relatively benign pathology. However, Goodman and Armelagos (1988) have calculated the mean age at death for those with and without hypoplasias and find startling differences. Individuals with no lesions have a mean age at death 4.4 years greater than individuals with one hypoplasia and 10.2 years greater than individuals with two or more hypoplastic episodes.

There are two hypotheses that have been proposed to explain the difference in mean age at death. The first hypothesis suggests that those with hypoplasia represent a group of individuals who were challenged by the insult early in their lifetime and continued to be subjected to insults during the rest of their lives. The increased 'wear and tear' throughout their lives leads to an earlier death. Another hypothesis suggests that major insults occur at a critical period of immunological development. This hypothesis suggests that individuals experiencing severe stress during the development of their immunological system may irreparably damage their ability to fight infection throughout their life time. The 'damaged goods' hypothesis suggests that significant thymolymphatic growth which is essential for developing effective immunological competence occurs prenatally, in infancy and in early childhood.

Clark and co-workers (1986) use the growth of the vertebral column to offer support to the 'damaged goods' hypothesis. The vertebral neural canal (VNC) rapidly develops during the same time that neurological and thymolymphatic tissues are also rapidly developing. A series of statistical analyses suggest that decreased mean VNC size is

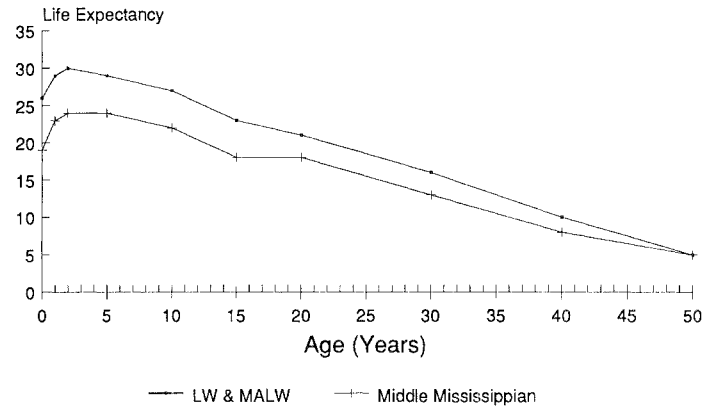


*Figure 7* Age specific life expectancies for the Dickson populations.

associated with greater vertebral wedging (a measure of morbidity) and decreased mean age at death (Clark et al. 1986).

The impact of multiple stressors affected the mortality pattern of the Dickson Mounds population. Change in the mortality profile is the final measure of the biological cost of the shift to agriculture at Dickson Mounds. A comparison of age specific life expectancies (Fig. 7) shows a decrease in life expectancy at all ages for the intensive agriculturalist. This difference, however, is greatest in the infant-childhood segment of the population.

In summary, the population at Dickson Mounds suffered biologically from the shift to intensive agriculture. The effects of this shift seem to have particularly affected infants and children (see Fig. 8; Table 3). The 'success' of this economic change appears to have taken place at a biological cost to infants and children.

The ability to reduce birth spacing, which had to have occurred, allowed the population not only to meet the increase in mortality but also to meet the increased labor needs for intensifying agriculture. But there was an increase in nutritional and infectious disease load which affected all segments of the population and especially


*Figure 8* Age-specific frequencies of Harris lines (....= distal and --- = proximal tibia) compared to enamel hypoplasias (___) at Dickson Mounds.

*Table 3* Summary of health changes at Dickson Mounds.

| Indicator | Patterns of stress[a] | Subgroup affected (age in years) | Differences observed | General comment |
|---|---|---|---|---|
| Long bone growth | + | 1–7 | Attained growth is less for tibia and femur in the 5–10-yr group[b] | Decreased growth velocity at 2–5 yr: chronic (nutrition-related). Some catch-up growth |
| Sexual dimorphism | 0 | 0–15 | None (pelvic and femoral measures) | Weak indicator-test of stress for methodological and theoretical reason |
| Harris lines | 0 | 1–3 | None (distal and proximal tibia) | Weak indicator based on experimental evidence |
| Enamel hypoplasias | + | 2–4 | Frequency in MM is twice that of LW. MALW is intermediate[b] | Good seasonal (nutrition-related) stress indicator |
| Wilson Bands | + | 2–4 | Approximately 70% increase from MALW to MM[b] | Nutrition related. Strong predictor of age at death |
| Porotic hyperostosis | ++ | 0–15 | Fourfold increase from LW to MM with increase in severity and involvement[b] | Nutritional stress (iron), with possible infectious disease synergy |
| Infectious Lesions | ++ | All ages | Threefold increase from LW-MALW to MM. Also, increase in severity[b] | Chronic infection. Endemic in MM. Synergistic with porotic hyperostosis |
| Trauma | + | 15–65 | Twofold increase from LW-MALW to MM for appendicular fractures[b] | Strongest difference noted in males. Suggests inter-personal strife |
| Degerative | + | 15–65 | 65% increase from LW to MM. Increase in severity (centrum and osteophytosis)[b] | Chronic wear and tear (physical stress) is greatest in males |
| Mortality | ++ | 0–10 | $d_x + q_x$ increase while $l_x$ and $e^o_x$ decrease. Cumulative differences are significant [b] | Best indicator of stress and inability to adapt. Most severe in subadults |

[a] −, decrease through time; 0, no change through time; +, increase through time; ++, strong increase through time
[b] Observed differences were statistically significant.

infants and children. This burden ultimately may have caused decreases in function which may have contributed to the collapse of the population.

**Wadi Halfa, Sudanese Nubia**

Similar changes in health to that which is seen at Dickson Mounds have been observed in other populations undergoing similar economic shifts (Cohen and Armelagos (eds) 1984). An example from the Old World is the Wadi Halfa area of Sudanese Nubia, where the archaeological record is remarkably complete. At Wadi Halfa there are biological remains which date back to the Mesolithic period. Although the critical material from the earliest agriculturalist is not available, there are two series of populations which reflect a less intensive (A-Group and G-Group) and a more intensive adaptation to agriculture (Meroitic, X-Group, and Christians). These samples provide a wealth of data for understanding the impact of change in subsistence (Martin et al. 1984).

Populations from the Meroitic (350 AD), X-Group (AD 350–550) and Christian (AD 550–1300) periods reveal similar patterns of pathology to those populations found at Dickson Mounds. There is, for example, similarity in the pattern of porotic hyperostosis (iron deficiency anemia), in which the children between 2 and 6 years of age and young adult females are most affected. However, a significantly lower prevalence of periosteal reaction was found in the Nubian populations (Martin et al. 1984). This surprising finding can be explained by the discovery that the Nubians were ingesting tetracycline, a broad spectrum antibiotic (Bassett et al. 1981) produced by mold-like bacteria (*Steptomycetes*) which contaminated their grain.

Growth retardation in the Nubians is extremely difficult to demonstrate from the cross-sectional data (Armelagos et al. 1972). Long bone length and widths fail to show any definitive evidence of growth retardation. The comparison with standards developed from United States children (Maresh 1955) shows that the Nubians are smaller, but are experiencing similar patterns of growth. However, when the long bone lengths and widths are compared with the thickness of cortical bone, problems in growth are evident (Fig. 9). Nubian cortices are very thin and are equivalent to the bone thickness found in 2-year-old children from the United States (Huss-Ashmore 1981). A histological analysis performed on some of these subadults confirmed a pattern of rapid resorption of bone at the inner surfaces (Huss-Ashmore 1981).

Martin and Armelagos (1979) further showed that young adult women (aged 19–25) from this same population were experiencing problems maintaining cortical bone (Fig. 10). There is a significant increase in rates of endosteal (inner surface) resorption (when compared to males of the same age). While these women were able to form osteons on their periosteal (outer) surfaces, there was no indication that these osteons were being mineralized. In other words, there was rapid resorption of bone from the inner surface, most likely being used as a source for calcium for the pregnant and lactating women (Martin et al. 1988)

The data from Nubia are particularly insightful in showing how early deprivation is linked to the entire population. The evidence from bone growth and remodeling shows a clear linkage between maternal and infant health. The compromised health and

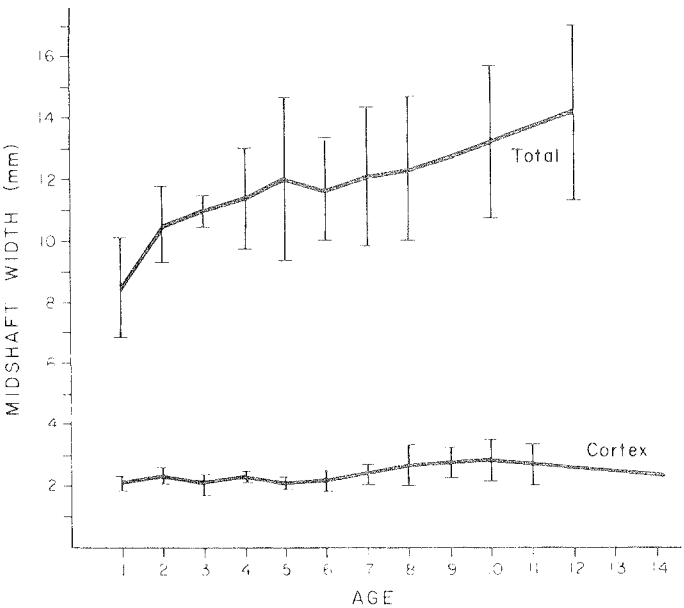


*Figure 9* Growth in femoral midshaft width and cortical thickness of Sudanese Nubian juveniles.

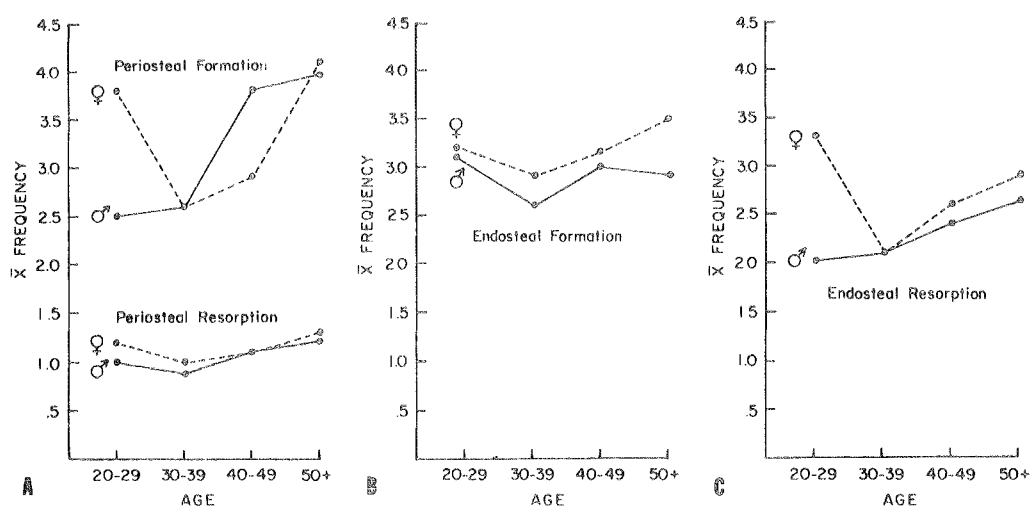


*Figure 10* (A) Average frequencies of forming osteons and resorption spaces at the periosteum. Though periosteal resorption remains relatively constant, formation shows variability. (B) and (C) Average frequencies of forming osteons and resorption spaces at the endosteum. Endosteal formation shows slight variation. Endosteal resorption increases dramatically for Sudanese Nubian females, aged 20-29 years (from Martin and Armelagos 1979:575).

nutritional status of mothers, especially during pregnancy and lactation, affects similar states in the infant and child. As well, repeated pregnancies, which can result from increased infant mortality, drains resources from mothers and results in an increase in maternal morbidity and mortality (Martin et al. 1988).

A final way that this linkage has been demonstrated is by the skeletal evidence for care of a hydrocephalic child. This child X-group lived until the age of around 10,

although a quadrapelegic. It is clear that s/he was cared for in a meticulous way and that this care had an effect on the distribution of resources and labor in the group. What this example so clearly reminds us of is that disease is not an isolated event, but is one that affects families and larger social groupings.

## Conclusions

Monitoring the health of infants and children can provide the prehistorian with a rich variety of information about the health of a community. As this segment of the population is very sensitive to environmentally and culturally produced insults, changes in morbidity in this segment of the population should provide one of the first signs of changes in environment and culture.

While this is a sensitive segment of the population, this should not imply that its health is any less important than the health of other age groups. The prehistorian and archaeologist may be in a unique position to follow the dynamic of how stresses occurring early in life affect the future functioning of the individual and may have significant consequences for the group. Data from Dickson Mounds suggest that individuals who are stressed early in life, as evidenced by porotic hyperostosis and enamel hypoplasias, have a decreased life expectancy. Despite a compensatory decrease in birth spacing, the cumulative effects of these stressors may have led to the collapse of this society. Data from Nubia suggest that the health and nutritional status of infants and children is delicately linked to the function of mothers, families and larger social units.

12.ii.89

*A. H. Goodman*
*School of Natural Science*
*Hampshire College, Massachusetts*
*G. J. Armelagos*
*Department of Anthropology*
*University of Massachusetts*

## References

Allen, L. H. 1983. Functional indicators of nutritional status of the whole individual or the community. *Clinical Nutrition*, 3(5): 169–75.

Armelagos, G. J. and Goodman, A. H. 1988. The origins of agriculture: population growth during a period of declining health. Paper presented at the annual meetings of the Society for Economic History (Detroit).

Armelagos, G. J., Mielke, J., Owen, K., Van Gerven, D., Dewey, J. R. and Mahler, P. 1972. Bone growth and development in prehistoric populations from Sudanese Nubia. *Journal of Human Evolution*, 1: 89–119.

Baker, D. J. P. and Osmond, C. 1986a. Childhood respiratory infection and adult chronic bronchitis in England and Wales. *British Medical Journal*, 293: 1271–5.

Baker, D. J. P. and Osmond, C. 1986b. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* I: 1077–81.

Barac-Nieto, M. 1987. Physical work determinants and undernutrition. *World Review of Nutrition and Dietetics*, 49: 22–65.

Bassett, E., Keith, M., Armelagos, G. J. and Martin, D. L. 1981. Tetracycline-labeled bone from prehistoric Sudanese Nubia (AD 350). *Science*, 209: 1532–4.

Blakey, M. L. and Armelagos, George J. 1985. Deciduous dental defects in prehistoric Americans from Dickson Mounds: prenatal and post natal stress. *American Journal of Physical Anthropology*, 66: 371–80.

Buikstra, J. E. and Cook, D. 1980. Paleopathology: an American account. *Annual Review of Anthropology*, 9: 433–70.

Buikstra, J. E. and Meilke, J. 1985. Demography, diet, and health. In *The analysis of prehistoric diets* (eds R. I. Gilbert and J. Mielke). Orlando: Academic Press, pp. 359–422.

Chandra, R. K. 1975. Fetal malnutrition and postnatal immunocompetence. *American Journal of Diseases of Childhood*, 129: 450–4.

Chen, L., Chowdhury, A. K. A. and Huffman, L. 1980. Anthropometric assessment of energy-protein nutrition and subsequent risk of mortality among preschool aged children. *American Journal of Clinical Nutrition*, 33: 1836–45.

Chen, L., Hug, E. and D'Souza, S. 1981. Sex bias in the family allocation of food and health care in rural Bangladesh. *Population and Development Review*, 7(1): 55–70.

Clark, G. J., Hall, N. R., Armelagos, G. J., Borkan, G. A. and Panjabi, M. M. 1986. Poor growth prior to early childhood: decreased health and life span in adults. *American Journal of Physical Anthropology*, 70: 145–60.

Cohen, M. N. and Armelgos, G. J. (eds) 1984. *Paleopathology at the Origins of Agriculture*. New York: Academic Press.

Cooper, M. D. and Lawton, A. R. 1974. The development of the immune system. *Scientific American*, 231(5): 58–72.

Dallman, P. R. 1987. Iron deficiency and the immune reponse. *American Journal of Clinical Nutrition*, 46: 329–34.

Dyson, T. 1984. Infant and child mortality in developing countries. *Critical Reviews in Tropic Medicine*, 2: 39–76.

Finch, C. A. and Cook, J. D. 1984. Iron deficiency. *American Journal of Clinical Nutrition*, 39: 471–7.

Goodman, A. H. and Armelagos, G. J. 1988. Childhood stress and decreased longevity in a prehistoric population. *American Anthropologist*, 90(4): 936–44.

Goodman, A. H., Armelagos, G. J. and Rose, J. C. 1980. Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Human Biology*, 52: 515–28.

Goodman, A. H., Lallo, J., Armelagos, G. J. and Rose, J. C. 1984a. Health changes at Dickson Mounds, Illinois (AD 950–1300). In *Paleopathology at the Origins of Agriculture* (eds M. N. Cohen and G. J. Armelagos). New York: Academic Press, pp. 271–306.

Goodman, A. H., Martin, D. L., Armelagos, G. J. and Clarke, G. A. 1984b. Indications of stress from bones and teeth. In *Paleopathology at the origins of agriculture* (eds M. Cohen and G. J. Armelagos). New York: Academic Press, pp. 13–49.

Gordon, J. E., Chitkara, I. and Wyon, J. 1963. Weanling diarrhea. *American Journal of Medical Sciences*, March, 245: 345–77.

Gordon, J. E., Wyon, J. B. and Ascoli, W. 1967. The second year death rate in less developed countries. *American Journal of Medical Sciences*, September: 121–44.

Haas, J. D. and Harrison, G. G. 1977. Nutritional anthropology and biological adaptation. *Annual Review of Anthropology*, 6: 69–101.

Hallberg, L. 1981. Bioavailability of dietary iron in man. *Annual Review of Nutrition*, 1: 123–47.

Hanson, L. A., Carlsson, B., Jalil, F., Hahn-Zoric, M., Hermodson, S., Karlberg, J., Mellander, L., Khan, S. R., Lindblad, B., Thiringer, K. and Zaman, S. 1988. Antiviral and antibacterial factors in human milk. In *Biology of Human Milk* (ed. L. A. Hanson). New York: Raven Press, pp. 141–58.

Hernell, O. and Blackberg, L. 1988. Antiparasitic factors in human milk. In *Biology of Human Milk* (ed. A. Hansen). New York: Raven Press, pp. 159–70.

Huss-Ashmore, R. 1981. Bone growth and remodeling as a measure of nutritional stress. In *Biocultural adaptation comprehensive approaches to skeletal remains* (eds D. L. Martin and P. Bumsted). Research Report Series 20. Amherst, Massachusetts: Department of Anthropology, University of Massachusetts.

Huss-Ashmore, R., Goodman, A. H. and Armelagos, G. J. 1982. Nutritional inference from paleopathology. In *Advances in Archaeological Method and Theory* (ed. M. Schiffer). 5: 395–474. New York: Academic Press.

Lallo, J. 1973. The skeletal biology of three prehistoric American Indian populations from Dickson Mound. Unpublished doctoral dissertation, University of Massachusetts, Amherst.

Lallo, J., Armelagos, G. J. and Mensforth, R. P. 1977. The role of diet, disease and physiology in the origin of poprotic hyperostosis. *Human Biology*, 44: 471–83.

Lallo, J., Armelagos, G. J. and Rose, J. C. 1978. Paleoepidemiology of infectious disease in the Dickson Mounds population. *Medical College of Virginia Quarterly*, 14(1): 17–23.

Larsen, C. S. 1987. Bioarchaeological interpretations of subsistence economy and behaviour from human skeletal remains. *Advance in Archaeological Method and Theory*, 10: 339–445.

Maresh, M. M. 1955. Linear growth of long bones of extremities from infancy through adolescence. *American Journal of Diseases of Children*, 89: 725–42.

Martin, D. L. and Armelagos, G. J. 1979. Morphometrics of compact bone: an example from Sudanese Nubia. *American Journal of Physical Anthropology*, 51: 571–8.

Martin, D. L., Armelagos, G. J., Goodman, A. H. and Van Gerven, D. P. 1984. The effects of socioeconomic change in prehistoric Africa: Sudanese Nubia as a case study. In *Paleopathology at the origins of agriculture* (eds M. N. Cohen and G. J. Armelagos). New York: Academic Press, pp. 193–214.

Martin, D. L., Armelagos, G. J. and Henderson, K. A. 1988. The persistence of nutritional stress in Northeastern Africa (Sudanese Nubian) populations. In *Famine in Africa*, vol. 1, (eds R. A. Huss-Ashmore and S. Katz). London: Gordon and Breach, pp. 185–209.

Martin, D. L., Goodman, A. H. and Armelagos, G. J. 1985. Skeletal pathologies as indicators of quality and quantity of diet. In *The analysis of prehistoric diets* (eds R. I. Gilbert and J. Mielke). Orlando: Academic Press, pp. 227–79.

Massler, M., Schour, I. and Sarnat, B. 1941. Development of the child as reflected in calcification patterns of the teeth. *American Journal of Diseases of Childhood*, 62: 33–67.

Mata, L. J., Urrutia, J. J. and Lechtig, A. 1971. Infection and nutrition of children of a low socioeconomic rural community. *American Journal of Clinical Nutrition*, 24: 249–59.

McNeish, A. S. 1986. The interrelationship between chronic diarrhoea and malnutrition. In *Diarrhoea and Malnutrition in Childhood* (eds A. S. McNeish and J. A. Walker-Smith). London: Butterworth, pp. 1–6.

Mensforth, R., Lovejoy, C. O., Lallo, J. and Armelagos, G. J. 1978. The role of constitutional

factors, diet, and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. *Medical Anthropology*, 2: 1–59.

Monsen, E. R., Hallberg, L., Layrisse, M., Hegsted, D. M., Cook, J. D., Mertz, W. and Finch, C. A. 1978. Estimation of availability of dietary iron. *American Journal of Clinical Nutrition*, 31: 134–41.

Mosley, H. W. 1984. Child survival: research and policy. *Population and Development Review*, 10 (supplement): 3–23.

Ortner, D. J. 1989. Theoretical and methodological issues in paleopathology. In *Human paleopathology: current syntheses and future options* (eds D. J. Ortner and A. C. Aufderheide). Washington: Smithsonian Press (in press).

Ortner, D. and Putschar,W. G. J. 1984. *Identification of pathological conditions in human skeletal remains*. Washington: Smithsonian Institution Press.

Park, E. A. 1964. The imprinting of nutritional disturbances on the growing bone. *Pediatorics*, 10 (supplement): 815–62.

Preston, S. H. 1980. *Biological and social aspects of mortality and length of life*. Liege: Ordina.

Sarnat, B. G. and Schour, I. 1941. Enamel hypoplasias (chronic enamel aplasia) in relationship to systemic diseases: a chronological, morphological and etiological classification. *Journal of the American Dental Association*, 28: 1989–2000.

Soedjatmoko, K. 1984. Plenary address: The challenge of world hunger. In *Nutrition in health and disease in international development* (eds A. E. Davis and G. K. Harper). New York: Plenum, pp. 1–16.

Steinbock, R. T. 1976. *Paleopathological diagnosis and interpretation: bone disease in ancient human populations*. Springfield, Illinois: Charles C. Thomas.

Stinson, S. 1985. Sex differences in environmental sensitivity during growth and development. *Yearbook of Physical Anthropology*, 28: 123–47.

Storey, R. 1988. Prenatal enamel defects in Teotihuacan and Copan. *American Journal of Physical Anthropology*, 75: 275–6, abstract.

Wells, C. 1975. Prehistoric and historic changes in nutritional diseases and associated conditions. *Progress in Food and Nutritional Sciences*, 1(2): 729–79.

Wood, C. S. 1983. Early childhood, the critical stage in human interactions with disease and culture. *Social Science and Medicine*, 17(2): 79–85.

Yaeger, J. and Sharawy, M. 1986. Enamel. In *Orban's Oral Histology and Embryology* (ed. S. N. Behaskar). St Louis: CV Mosby, pp. 45–100.

## Abstract

*Goodman, Alan H. and Armelagos, George J.*

**Infant and childhood morbidity and mortality risks in archaeological populations**

Infants and children are nearly universally found to be among the most vulnerable subgroups of a population. Their health can be a sensitive indicator of the health of the population as a whole. Furthermore, repeated bouts of illness during infancy and childhood, periods of rapid development, can have lasting functional effects on the individual and the group. In this paper we provide a framework for studying infant and childhood health in archaeological populations, briefly review methods for studying infant-childhood health in skeletal remains, and provide

examples of the sensitivity and adaptive significance of this segment of the population by examining infant and childhood health at Dickson Mounds, Illinois and Wadi Halfa, Sudanese Nubia. A variety of methods are available for studying infant and childhood health in archaeological groups. Taken together, these methods can provide insights into the patterns and consequences of health in prehistory.