IN:
Janet Chrzan and John Brett (eds.)
Food Research: Nutritional Anthropological and Archaeological Methods, Berghahn Books, 2017, pp 183-197.

CHAPTER 11

Nutritional Stress in Past Human Groups

Alan H. Goodman

Introduction and Purpose

Human skeletal remains are the most direct means for assessing mal and particularly undernutrition in past populations. Despite the man scientific challenges to reconstructing past nutrition from bones and tee disciplinary collaborations are making it possible to infer the general nu status of past individuals and groups. This chapter's purpose is to provi oretical and methodological introduction to the study of the nutritior of past populations, a major component of paleonutrition—the study o: (Turner and Livengood, this volume) and nutritional status (this chapte populations. I begin by outlining how the skeleton responds generally iological perturbations or stress. Against this background, I review thr most common and accepted paleonutrition indicators: linear bone growt enamel hypoplasias, and porotic hyperostosis. Specific attention is pai "state of the art": what has been established for each nutritional stress i and what additional knowledge is most essential. Where possible, I focu possible adaptive and functional implications of these nutritional stres tors, inferred from their study in contemporary contexts.

The Systemic (Nutritional) Stress Perspective

A stress model (Figure 11.1) focuses on skeletal manifestations of physi perturbation (or stress) (Goodman and Armelagos 1989; Goodman and 2002). The model is useful for illustrating connections between systemic and skeletal manifestations of undernutrition on the one hand, and the a and functional consequences of these stress indicators on the other hand

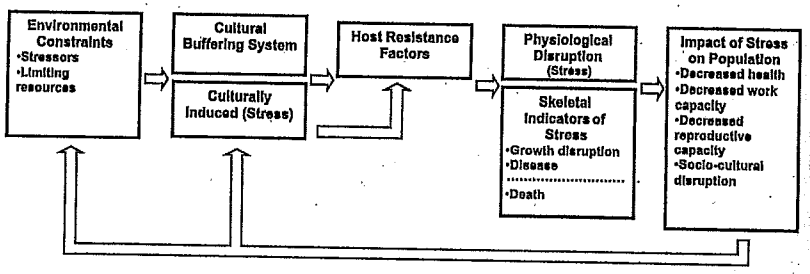

Figure 11.1. Stress model adapted for use in skeletal series. Although stress as a physiological disruption cannot be directly measured, various skeletal changes may be used to infer stress and its impact on individual and population adaptation (from Goodman and Armelagos 1989).

The left side of the model starts with environmental constraints to adaptation: limiting resources and stressors. Stressors may be related to climatic extremes, such as excesses of heat and cold, or low partial pressure of oxygen. The most important limiting resources are likely to be basic ones: water, shelter, and nutrients.

Cultural systems are generally effective in buffering environmental constraints and stressors. Agriculture, for example, is an economic adjustment that brings about a number of changes in a culture's ability to buffer stresses. Energetic efficiency and the amount of food produced per unit area are greater for agriculture than for hunting and gathering. Thus, agriculture would seem to provide a buffer against caloric insufficiency. However, greater population density and socioecological changes are frequent covariates of agricultural intensification that may have unforeseen consequences for health and nutrition (Cohen and Armelagos 1984). Thus, culture acts to both buffer stress and produce new stressors and constraints (Goodman and Armelagos 1989).

Both environmental constraints that are not well buffered by the cultural system and newly produced cultural stressors reach individuals. From then on, the degree of adaptation to these stressors and constraints depends on the individual's level of host resistance, a function of genetic, developmental, and physiological statuses. Unfortunately, chronic stressors and limiting resources often overwhelm adaptive systems. Individuals who have poor nutrition, for example, are less resistant to infectious diseases, and infectious disease further lowers nutritional status (Allen 1984; Martorell 1980; Mata Urrutia, and Lechtig 1971).

The severity and duration of the stress response may be viewed as a function of the degree of cultural and environmental constraints and stressors, balanced against the adequacy of the cultural buffering system and individual resistance resources. Fortunately for the paleo-epidemiologist, the stress response, a stereotypic physiological change resulting from the struggle to adjust, is frequently

manifested in relatively permanent skeletal changes. Because bone and teeth are limited in their response repertoire, less is known of the stressors that were the cause of the struggle, but fortunately one finds clear evidence of a struggle to adapt.

The significance of health and adaptation extends beyond the individual to the population and society. Undernutrition, for example, has a negative effect on work capacity, fertility, morbidity, and mortality, causing secondary disruptions to the social, political, and economic structure of a community (Allen 1984). The following section provides information on three main indicators of nutritional health status.

The Evidence of Nutritional Stress in Skeletal Tissues

A wealth of indicators have been developed to gain insight into stress, disease, and nutrition from the skeleton (Goodman and Martin 2002). In this section I highlight three main indicators of nutritional status.

Linear Bone Growth and Adult Stature

Introduction: Contemporary studies and functional interpretations

Because growth is sensitive to a wide range of environmental conditions, studies of human growth and development have long been key tools for the analysis of stress and adaptation in past and present groups. Growth in parameters such as height, weight, and arm circumference are sensitive indicators of nutritional status (Sutphen 1985; Eveleth and Tanner 1976), defined as "the state resulting from the balance between the supply of nutrients on the one hand and the expenditure of the organism on the other" (McLaren 1976: 3).

The most important distinction to be made in paleonutriton is between studies of subadults and adults (Johnston and Zimmer 1989). The main advantage of studies of subadults is that they provide the most sensitive measures of changes in nutritional status, whereas studies of adults provide an index of the cumulative genetic and environmental conditions during all of development. Adult size and shape is subject to catch-up growth, the increase in growth rate following recovery from growth-dampening conditions. Catch-up growth can obscure the effects of transient but significant growth variations. Furthermore, variation in subadults is likely due to immediate conditions, whereas size variation in adults is more likely to reflect chronic conditions.

Because growth is rather quickly affected by nutrient deficiency, it is a sensitive indicator of nutritional stress. Acheson (1960) suggests that growth's sensitivity to poor nutrition is logical when interpreted in adaptive terms. The growing

organism can usually catch up, once adequate nutrition is restored. However, if deprivation is severe or long-lasting, then a short-term adaptation can become a warning sign of impairment of critical functions such as disease resistance and reproductive capacity. In this regard, growth may also be used to infer deficits in other critical functions (Allen 1984). Chavez and Martinez (1982) have found that mild-to-moderate malnutrition in Mexican peasant children has a profound effect on their activity levels, social behavior, learning patterns, and disease resistance. For example, those children who are most malnourished (and show the greatest deficiencies in growth) tend to be far less exploratory, cry more often, are less interactive with their parents, and are ill for longer periods of time compared to their better nourished peers (ibid.).

Because of its sensitivity and nonspecificity, Tanner (1986) has proposed that growth variation is useful as a measure of equality of conditions within a society. He suggests that when variation in growth between socioeconomic groups disappears, one may be able to say that in that society, all individuals have equal access to resources. In summary, the anthropometric status of living individuals and groups provides insight into their adaptation to environmental influences affecting diet.

Anthropometric studies of skeletal populations

Subadult growth. Despite the potential of studies of subadult growth in prehistoric populations, several practical factors have limited their development (Goodman and Martin 2002; Johnston and Zimmer 1989). First, prehistoric series are frequently plagued by small sample size, particularly after five years of age. This is the primary reason for the paucity of comparative studies of growth of prehistoric subadults. Second, there is a technical problem of measuring long bones with and without epiphyses (the unattached growing ends of long bones), which are frequently destroyed or otherwise lost to archaeological recovery. This irregularity has the potential of adding considerable measurement error. Third, because the archaeological sample is cross-sectional (in fact, a death assemblage), the results can be used to infer periods of peak stress only when demographic parameters are relatively stable. Furthermore, cemetery-based studies represent not the healthy or even the "average" child, but those who died (Wood et al. 1992).

Fourth, dental age is the best known proxy for chronological age. This can be considered a potential source of error in that a bias arises toward a more conservative estimation of growth failure, as dental age (based on calcification and eruption times) is also likely to be somewhat affected by environmental conditions (Garn, Rohmann, and Guzman 1963). Similarly, inability to distinguish sex of subadults disallows comparisons between boys and girls. And fifth and finally, we have only limited ability to compare growth in prehistory directly to that of contemporary groups. The only sample from which longitudinal growth of long bones is well established is that of the childhood growth study conducted in

Denver, Colorado (Maresh 1955). Despite these limitations, a number of useful paleoepidemiological studies have focused on subadult growth (e.g., Armelagos et al. 1972; Cook 1971; Jantz and Owsley 1984; Johnston 1962).

Anthropometry of adults. Studies of adults are less constrained by problems of assignment of age and sex or small sample sizes than are subadult samples. In addition to separate study of male and female growth, adult samples allow for comparison of patterns of growth (sexual dimorphism studies) between males and females. Small sample sizes, which frequently limit the power of analysis of subadult growth, are less of a factor in analysis of adult growth.

The largely technical advantages of studying adults are unfortunately offset by disadvantages. As in studies of adult anthropometry of living populations, the main drawback to studies in prehistory revolves around the loss of sensitivity for clarifying underlying processes affecting growth and ultimate size at adulthood. The loss of the most stressed segment of the population (subadults) due to death before adulthood, coupled with the ability to catch up in growth, renders adult morphology less sensitive to environmental variation when compared to subadult growth and development.

Nonetheless, comparisons of adult stature can be very informative, and a wide variety of studies of adult anthropometry have been performed. In a review of stature (derived from long-bone lengths) in prehistoric populations from Mesoamerica, Genoves (1967: 76; also Bass 1971; Brothwell 1981; Ubelaker 1981) has found that female stature decreased from a mean of around 62 inches in northern Mesoamerica and the American Southwest to about 58 inches in southern Mesoamerica. Male stature also declined from around 66 inches in the north to around 62 inches in the south. Genoves suggests that subsistence differences may be responsible for the gradient in heights from the north to the south.

Summary

Anthropometric studies of living populations have profoundly influenced similar studies of prehistoric populations. Studies of adult stature and subadult long-bone lengths are best regarded as nonspecific indicators of stress. Because the consequence of growth faltering looks the same regardless of specific stressor or limiting resources, contextual information is needed to interpret the cause of the growth faltering. Some sense of the severity and temporal pattern of stress may be inferred from the pattern of disruption. Like studies of mortality, studies of subadult growth are frequently hampered by imprecision in estimation of age and small sample sizes. Despite these limitations, anthropometric studies of past populations continue to yield valuable information, which is perhaps a testament to the vigor of this indicator. Our ability to assess subadult growth and adult morphology from skeletal remains provides a powerful tool for the assessment of nutritional status in prehistoric humans.

Linear Enamel Hypoplasias

Pathophysiology and etiology

Linear enamel hypoplasias (LEH) are a class of developmental enamel defects (DED). Visibly recognizable as transverse or linear deficiencies in enamel thickness, they are the permanent end result of a disruption during the enamel secretion stage of tooth crown development (Suckling 1989; Goodman and Rose 1990). The importance of these defects rests in the fact that they provide an indelible indicator of periods of stress during early life and tooth crown development (prenatally to twelve months for deciduous teeth, and birth to seven years for permanent teeth).

Enamel hypoplasias vary from light multiple and single pits to lines of increased enamel thickness and thick bands of missing enamel. Based on the pattern of defects within and among teeth, hypoplasias can reliably be distinguished as resulting from one of three conditions: (1) a hereditary anomaly, (2) a localized trauma, or (3) a systemic metabolic stress (Shawashy and Yaeger 1986). Although enamel hypoplasias due to systemic stresses are common and easily discerned from defects due to non-systemic factors, it is difficult to attribute them to a more exact cause (Pindborg 1982). Cutress and Suckling (1982) compiled a list of nearly a hundred factors considered to be possible causes of enamel defects. The list of potential causes, which includes many nutritional insufficiencies as well as drug toxicities and almost any disease that severely stresses metabolism, gives credence to the view that enamel defects are highly sensitive to physiological and metabolic changes and are best considered to be indicators of nonspecific stress.

Perhaps the most important characteristic of LEHs is that an estimation is possible of the developmental age of the individual at the time of their formation. This information may be inferred from the location of enamel defects on tooth crowns. Thus, by locating LEHs on tooth crowns, one can begin to develop a chronological record of physiological stress experienced by individuals (Kreshover 1960; Sarnat and Schour 1941). For example, Swardstedt (1966) compared the prevalence of enamel defects by half-year developmental periods in "social groups" at Westerhus in medieval Sweden.

Because a number of issues influence the accurate estimation of age at development of a defect (see Goodman and Rose 1990, 1991 for reviews), it should be kept in mind that developmental age is not exactly equal to chronological age. Nonetheless, this methodology provides a very useful and unique understanding of stress experienced by adolescents and adults during infancy and childhood.

Enamel hypoplasias in contemporary and prehistoric populations

Epidemiological studies of the frequency of enamel hypoplasias in contemporary populations find an association between the prevalence of enamel hypoplasias and general living conditions. Though direct comparison is difficult due to differences in method of diagnosis and sampling of individuals and teeth (Goodman and Armelagos 1985a, 1985b), individuals in developed countries tend to have lower rates of enamel defects than do individuals from underdeveloped areas. The frequency of individuals with one or more hypoplasias on permanent teeth is generally less than 10 percent in most populations from developed, industrialized countries (Cutress and Suckling 1982) and is usually over 50 percent in developing countries (Goodman and Rose, 1990, 1991).

The pioneering work of Sweeney (Sweeney and Guzman 1966; Sweeney et al. 1969; Sweeney, Saffir, and de Leon 1971) firmly established an association between enamel hypoplasias and malnutrition. Sweeney and co-workers (1971) found an increase in enamel hypoplasias of the deciduous upper central incisor (teeth whose crowns develop from about 6 months prenatally to about 3 months postnatally) and with severity of malnutrition in Guatemalan children: 43 percent of the children aged two to seven with second-degree malnutrition (61% to 75% weight-for-age) had hypoplasias, whereas 73 percent of children with the more severe third-degree malnutrition (60% or less weight-for-age) had enamel hypoplasias.

Goodman and colleagues (1987) studied the frequency and chronological distribution of enamel hypoplasias in Mexican children from five rural communities selected because of the presence of endemic mild-to-moderate malnutrition (children at 60%–95% weight for age). They found one or more hypoplasias on 46.7 percent of 300 children examined. As weaning generally takes place in the second year in these Mexican communities, they suggest that the increased frequency of hypoplasias may result from undernutrition and infectious diseases associated with weaning. A prospective study in Tezonteopan, another town in highland Mexico, found that LEHs are about half as frequent in children who were provided nutrient supplements (Goodman, Martinez, and Chavez 1991). The supplement, which contained over twenty nutrients (calories, proteins, and a soup of micronutrients), also reduced the incidence and severity of respiratory and diarrheal diseases in the children that took them, so the reduction in LEH frequency cannot be attributed to a single nutritional factor (ibid.).

LEHs have frequently been used to compare stress levels among different prehistoric populations. Numerous authors, including Cassidy (1984); Goodman and co-workers (1984); Perzigian, Tench, and Braun (1984); and Smith, Bar-Yosef, and Sillen (1984) have noted an increased frequency of defects in agriculturalists versus hunting and gathering groups. Goodman, Armelagos, and Rose (1984) also note that the peak period of stress tends to be earlier in the agriculturalists versus gatherer-hunters.

Hutchinson and Larsen (1988) found a greater frequency of hypoplasias in individuals from the Georgia coast post-European contact as compared with pre-contact individuals. Corruccini, Handler, and Jacobi (1985) evaluated the

chronological distribution of enamel defects in enslaved Africans from Barbados. They found a relatively late peak age at development of defects (around 3.5 to 4.0 years) and attribute this peak to a historically documented late age at weaning and post-weaning stress.

Summary

Linear enamel hypoplasias are one of the most frequently studied skeletal manifestations of stress. In comparison to measures of completed growth, which tend to signify chronic stress, these measures of growth disruption are time-specific and may indicate more acute periods of stress. Like growth status, they are best thought of as nonspecific (general) indicators of stress. However, when combined with measures of achieved growth they can help to provide information on the severity and temporal pattern of stress.

Enamel hypoplasias are a well-studied paleo-epidemiological tool. They have been subject to strict tests of reliability, and their etiology has been studied through ecological, case-control, and prospective designs (Goodman and Rose 1991). These studies have helped sharpen our understanding of the utility of these defects, but they have not answered all questions. Among a few concerns needing further attention are the best means of estimating an individual's age at development of defects, the best set of teeth for evaluation of stress in survey studies, and the way to interpret variations in the size and shape of enamel defects.

Without doubt a great deal of recent work has gone into understanding the cause of LEH and possible functional inferences to be derived from LEH studies. Studies of contemporary populations have shown a consistent increase in the prevalence of enamel hypoplasias for groups that live in poorly nourished and underdeveloped communities. Though the association of enamel defects with weaning suggests a strong role for a nutritional cause, it is not clear how important nutrition is, which nutrients are most critical, and how nutrition interacts with other factors such as infectious and parasitic diseases.

Porotic Hyperostosis and Anemia

Background: Pathophysiology, etiology, and functional inferences

Anemias potentially affect bones involved in the production of red blood cells. While the possible etiologies of anemia include discrete genetic traits (Mensforth et al. 1978), hereditary hemolytic anemia (Angel 1964, 1966, 1967), or some form of nutritional disorder (Nathan and Haas 1966), nutritional anemia has been suggested as the primary factor in the etiology of porotic hyperostosis for the vast majority of the documented cases in prehistory (Mensforth et al. 1978; Mensforth 1991; Stuart-Macadam 1987).

Porotic hyperostosis is a descriptive term for lesions on the cranium, the roof of the eye orbits, and the ends of long bones. These lesions are produced by bone marrow proliferation that is diagnostic of anemia. As the name implies, the lesion has a very porous (coral-like) appearance that 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 layer of bone becomes thinner and may eventually disappear, exposing the inner trabecular bone, which is quite porous.

The lesions of porotic hyperostosis typically involve thinning and destruction of the outer tables of the cranial vault, accompanied by thickening and exposure of the deeper diploic tissue. Porotic hyperostosis is usually symmetrically distributed and presents as a tight cluster of small porous openings that are visible to the naked eye. The lesion is typically exhibited on bones of the cranial (frontal, temporals, parietals, occipital) and the superior border of the eye orbits. Many researchers have given the expression of the disease as it appears in the orbits the label "cribra orbitalia," because for many years it was not clear that the two locations (vault and orbit) had the same etiology. There is now overwhelming evidence that both types of lesions are part of the same disease process (see Stuart-Macadam 1987, 1989) and should be referred to as porotic hyperostosis.

In a review of the literature on clinical evidence of bone changes in anemic individuals, Stuart-Macadam (1987) provided compelling evidence that bony lesions are a product of iron-deficiency anemia. In addition to a thickening of the diploe of cranial and orbital bones in anemia patients, the lesion is usually distributed in a symmetrical pattern on the cranium. In her analysis of clinical data combined with X-ray and skeletal observations of the disease, Stuart-Macadam demonstrated that anemia is responsible for the lesion seen on bones.

Consensus has developed that porotic hyperostosis in the New World is most likely related to iron-deficiency anemia (Mensforth et al. 1978). This view, according to Mensforth and co-workers (1978: 7), developed from three lines of evidence. First, iron-deficiency anemia and porotic hyperostosis are widespread throughout the New and the Old Worlds. Second, the distribution of porotic hyperostosis corresponds to the distribution of dietary staples that are low in utilizable iron. Finally, there is no pre-Columbian evidence to support the occurrence of skeletal changes that are characteristically found in the hemolytic anemias associated with hemoglobin variants. Some sense of the functional meaning of these porotic lesions may be estimated from studies of anemia in contemporary populations. Iron-deficiency anemia is one of the most widespread and common nutritional problems in the contemporary world (Dallman, Yip, and Johnson 1984). It is particularly pronounced in children, adolescents, and women during child-bearing years (Dallman, Simes, and Stekel 1980).

Indeed, the functional consequences of mild anemia are well understood and have been shown to be profound (Scrimshaw 1991). Iron deficiency—even at levels where the most common measures of iron status, hemoglobin and hematocrits, are normal, that is, iron deficiency without anemia—can lead to a suite

of functional costs. Various organs and systems show structural changes with borderline iron deficiency. Vyas and Chandra (1984: 45) note that the multiple consequences of iron deficiency are not surprising because iron is "an essential cofactor of several enzyme systems that play an important role in metabolic processes and cell proliferation." Many of these enzymes are involved in vital functions such as DNA synthesis, mitochondrial electron transport, and catecholamine metabolism.

The organism-level consequences of iron deficiency are typically divided into three areas: (1) disease resistance, (2) activity/work capacity, and (3) cognition and behavior. Mild iron deficiency, without low hemoglobin, is associated with learning deficiencies (Howell, 1971). Of particular note are changes in attention and memory control processes. Howell (1971) showed that 3- to 5-year-old anemic children had decreased attention spans, and Sulzer, Wesley, and Leonig (1973) illustrated that anemic children of the same age had lower IQ measures and impaired associative reactions.

The effects of iron deficiency and anemia on work capacity are particularly profound (Scrimshaw 1991). Anemic subjects cannot maintain the same pace and duration of work as can non-anemic subjects, and they reach a lower mean maximal workload. Anemic Guatemalan laborers performed much worse on the Harvard Step Test (a measure of moderate exertion) than their non-anemic peers, and the work output and pay of Indonesian rubber tappers correlates almost perfectly with their hemoglobin levels (ibid.). Decreased oxygen affinity and increased cardiac output are the "adaptive" responses to anemia (Vyas and Chandra 1984). However, these adaptations can only cover for deficiency when the organism is sedentary or at rest. The example of the Indonesian rubber tappers suggests a troubling synergy: iron deficiency leads to less pay, and less pay further erodes diets.

Iron deficiency has a variety of effects on immunocompetence and infection. Experimentally induced iron deficiency results in a reduction in lymphocyte proliferation, the production of rosette-forming T-cells, and the microbicidal capacity of neutrophils (Dallman 1987; Vyas and Chandra 1984). In humans, iron supplements have led to a decreased prevalence of diarrhea and upper- and lower-respiratory infections. Thus, lesions indicative of porotic hyperostosis and anemia in prehistory have potential to provide insights as to how well children and adult performed in terms of cognitive tasks, work capacity, and infection resistance.

Paleoepidemiology

Several examples serve to highlight the complexity of interpreting rates of porotic hyperostosis in past groups. Lallo, Armelagos, and Mensforth (1977) and Mensforth et al. (1978) recorded the frequency and distribution of lesions by age. Using very small age group categories, Lallo and colleagues (1977) demonstrated

that the clustered lesions in younger children reflected an increased need for iron metabolism during growth and development at Dickson Mounds, Illinois. An analysis of the relationship between porotic hyperostosis and infectious disease strongly suggested that the two occurred together and acted in a synergistic fashion, with porotic hyperostosis increasing the likelihood of infectious disease. Porotic hyperostosis had an earlier age of onset than did infection, and the diseases co-occur with high frequency at subsequent ages. Thus, Lallo and colleagues (1977) were able to document their claim that iron deficiency predisposed children to infectious disease, possibly by lowering their resistance.

On the other hand, Mensforth et al. (1978) showed the reverse process for a prehistoric population from Libben, Ohio: infectious diseases predisposed children to iron-deficiency anemia. By distinguishing between healed and unhealed lesions, the researchers revealed a synergistic relationship whereby infectious disease acted as the initial stress that predisposed Libben individuals to iron deficiency (ibid.). The importance of these two studies is that different ecological and cultural variables worked to cause the same lesion (porotic hyperostosis) in each archaeological population, even though different underlying conditions precipitated the response.

Summary and epidemiological considerations

In order to better understand the functional consequences of porotic hyperostosis, it would be useful to have more precise information on the severity and duration of anemia necessary to cause the osseous changes. My assumption is that porotic hyperostosis is a relatively severe and chronic manifestation of iron deficiency. However, this point has not been well documented by studies that directly compare standard measures of iron status (such as hemoglobin, hematocrit, and plasma transferrin) with skeletal changes. Also, although it is now fairly standard to record the severity and extent of porotic lesions as well as the degree of remodeling, there is no reference standard for evaluation of degree of involvement or degree of remodeling, nor is there a set method for classification of pattern of involvement or choice of bone(s) for evaluation in incomplete skeletons.

The above epidemiological issues notwithstanding, tremendous advancements in the study of porotic hyperostosis have been made thanks to the detailed research of Mensforth (1991; Mensforth et al. 1978), Stuart-Macadam (1987, 1989), and others. Today we have a fairly well developed understanding of how porotic lesions are formed, and we are relatively confident about differential diagnosis. It bears repeating that iron deficiency is not the only cause of anemia and seldom occurs without other nutrient deficiencies. In all, though, understanding the functional significance of iron status in contemporary populations and the severity of porotic hyperostosis in some past populations can lead to a sense of how infirmity may have affected the lives and livelihood of past peoples.

Conclusions

Many of the most exciting developments in anthropology have come in the overlapping fields of paleonutrition, paleopathology, and paleodemography. These fields, which respectively focus on the nutrition, health, and demography of past populations, have all enjoyed promising advancements in methodology. Even more consequentially, improved understanding of the context, causes, and consequences of morbidity and mortality in individuals and groups has led to new inferences and research directions. The human skeleton found in the archaeological context is now widely understood to hold key historical and human ecological information.

This chapter has provided a current assessment of the toolkit for measurement and analysis of nutrition from skeletal remains. For illustrative purposes, I have focused on three commonly used, well accepted indicators of nutritional status. All indicators suffer from incomplete understanding of the biological processes leading to their formation. Still, when studied together and with data archaeological information on diets, these data can provide critical insights as to nutritional status and adaptation in past populations.

Alan Goodman is Professor of Biological Anthropology at Hampshire College in Amherst, Massachusetts. His interests focus on the intersections of biology and culture, and particularly on the health and nutritional consequences of political-economic processes. Goodman is the editor or author of seven books and numerous articles including *Race: Are We So Different?* (co-authored with Moses and Jones) and *Nutritional Anthropology* (co-edited with Dufour and Pelto). He received his PhD in anthropology from the University of Massachusetts and was a postdoctoral fellow in international nutrition at the University of Connecticut. Formerly Vice President for Academic Affairs and Dean of Faculty at Hampshire College, he is also a past President of the American Anthropological Association.

Acknowledgements

This chapter updates and abstracts from a prior chapter (Goodman and Martin 2002). This chapter is dedicated to George Armelagos (1936–2014), who taught me many of the specifics of paleonutrition as well as how to think like an anthropologist and a scientist.

References

Acheson, R. M. 1960. Effect of Nutrition and Disease on Human Growth. In *Human Growth,* ed. J. M. Tanner, 73–92. New York: Pergamon Press.

Allen, Lindsay. H. 1984. Functional Indicators of Nutritional Status of the Whole Individual or the Community. *Clinical Nutrition* 3(5): 169–175.

Angel, J. Lawrence. 1964. Osteoporosis: Thalassemia? *American Journal of Physical Anthropology* 22: 369–374.

———. 1966. Porotic Hyperostosis, Anemias, Malarias, and Marshes in Prehistoric Eastern Mediterranean. *Science* 153: 760–763.

———. 1967. Porotic Hyperostosis or Osteoporosis Symmetrica. In *Diseases in Antiquity,* ed. D. R. Brothwell and A. T. Sandison, 378–389. Springfield, IL: Charles C Thomas.

Armelagos, George J., J. H. Mielke, H. O. Kipling, D. P. Van Gerven, J. R. Dewey, and P. E. Mahler. 1972. Bone Growth and Development in Prehistoric Populations from Sudanese Nubia. *Journal of Human Evolution* 1: 89–119.

Bass, William M. 1971. *Human Osteology: A Laboratory and Field Manual of the Human Skeleton.* Columbia, Missouri: University of Missouri Archaeological Museum Society.

Brothwell, Don R. 1981. *Digging Up Bones.* Ithaca, NY: Cornell University Press.

Cassidy, Claire M. 1984. Skeletal Evidence for Prehistoric Subsistence Adaptation in the Central Ohio River Valley. In *Paleopathology at the Origins of Agriculture,* ed. M. N. Cohen and G. J. Armelagos, 307–346. New York: Academic Press.

Chavez, Adolfo, and C. Martinez. 1982. *Growing Up in A Developing Community.* Mexico City: Instituto Nacional de la Nutricion.

Cohen, Mark N., and G. J. Armelagos, eds. 1984. *Paleopathology at the Origins of Agriculture.* New York: Academic Press.

Cook, Della C. 1971. Patterns of Nutritional Stress in Some Illinois Woodland Populations. Unpublished thesis. Department of Anthropology, University of Illinois.

Corruccini, Robert S., J. S. Handler, and K. P. Jacobi. 1985. Chronological Distribution of Enamel Hypoplasias and Weaning in a Caribbean Slave Population. *Human Biology* 57: 699–711.

Cutress, Terry W., and G. W. Suckling. 1982. The Assessment of Non-carious Defects of Enamel. *International Dental Journal* 32: 117–122.

Dallman, P. 1987. Iron Deficiency and the Immune Response. *American Journal of Clinical Nutrition* 46: 329–334.

Dallman, P., R. Simes; A. Stekel. 1980. Iron Deficiency in Infancy and Childhood. *American Journal Clinical Nutrition* 33: 86–118.

Dallman, P. R., R. Yip, and C. Johnson. 1984. Prevalence and Causes of Anemia in the United States, 1976 to 1980. *American Journal of Clinical Nutrition* 39: 437–445.

Eveleth, Philis B., and J. M. Tanner. 1976. *Worldwide Variation in Human Growth.* International Biological Programme 8. Cambridge University Press.

Garn, Stanley M., C. G. Rohmann, and M. A. Guzman. 1963. Genetic, Nutritional and Maturational Correlates of Dental Development. *Journal of Dental Research* 44: 228–242.

Genoves, Santiago. 1967. Proportionality of the Long Bones and Their Relation to Stature among Mesoamericans. *American Journal of Physical Anthropology* 26: 67–78.

Goodman, Alan H., L. H. Allen, G. P. Hernandez, A. Amador, L. V. Arriola, A. Chavez, and G. H. Pelto. 1987. Prevalence and Age at Development of Enamel Hypoplasias in Mexican Children. *American Journal of Physical Anthropology* 72: 7–19.

Goodman, Alan H., and G. J. Armelagos. 1985a. Factors Affecting the Distribution of Enamel Hypoplasias within the Human Permanent Dentition. *American Journal of Physical Anthropology* 68: 479–493.

———. 1985b. The Chronological Distribution of Enamel Hypoplasia in Human Permanent Incisor and Canine Teeth. *Archives of Oral Biology* 30: 503–507.

———. 1989. Infant and Childhood Morbidity and Mortality Risks in Archaeological Populations. *World Archaeology* 21(2): 225–243.

Goodman, Alan H., G. J. Armelagos, and J. C. Rose. 1984. The Chronological Distribution of Enamel Hypoplasias from Prehistoric Dickson Mounds Populations. *American Journal of Physical Anthropology* 65: 259–266.

Goodman, Alan H., and Martin, D. L. 2002. Reconstructing Health Profiles from Skeletal Remains. In *The Backbone of History,* ed. R. H. Stekel and J. C. Rose, 11–60. Cambridge University Press.

Goodman, Alan H., C. Martinez, and A. Chavez. 1991. Nutritional Supplementation and the Development of Linear Enamel Hypoplasias in Children from Tezonteopan, Mexico. *American Journal Clinical Nutrition* 53: 773–781.

Goodman, Alan H., and J. C. Rose. 1990. Assessment of Systemic Physiological Perturbations from Dental Enamel Hypoplasias and Associated Histological Structures. *Yearbook of Phys. Anthropol.* 33: 59–110.

———. 1991. Dental Enamel Hypoplasias as Indicators of Nutritional Status. In *Advances in Dental Anthropology,* ed. M. Kelley and C. Larsen, 279–293. New York: Wiley-Liss.

Howell, D. 1971. *Significance of Iron Deficiencies: Consequence of Mild Deficiency in Children; Extent and Meaning of Iron Deficiency in the United States.* Washington, DC: National Academy of Sciences.

Hutchinson, Dale L., and C. P. Larsen. 1988. Determination of Stress Episode Duration from Linear Enamel Hypoplasias: A Case Study from St. Catherine's Island, Georgia. *Human Biology* 60: 93–110.

Jantz, Richard L., and D. W. Owsley. 1984. Temporal Changes in Limb Proportionality among Skeletal Samples of Arikara Indians. *Annals of Human Biology* 11(2): 157–163.

Johnston, Frank E. 1962. Growth of the Long Bones of Infants and Young Children at Indian Knoll. *American Journal of Physical Anthropology* 20: 249–254.

Johnston, Frank E., and L. O. Zimmer. 1989. Assessment of Growth and Age in the Immature Skeleton. In *Reconstruction of Life from the Skeleton,* ed. M. Y. Iscan and K. A. Kennedy, 11–22. New York: Alan R Liss.

Kreshover, S. 1960. Metabolic Disturbances in Tooth Formation. *Annals of the New York Academy of Science* 85: 161–167.

Lallo, John, G. J. Armelagos, and R. P. Mensforth. 1977. The Role of Diet, Diseases and Physiology in the Origin of Porotic Hyperostosis. *Human Biology* 49: 471–483.

Maresh, M. M. 1955. Linear Growth of Long Bones of Extremities from Infancy through Adolescence. *American Journal of Diseases of Children* 89: 725–742.

Martorell, Reynaldo 1980. Interrelationships between Diet, Infectious Disease, and Nutritional Status. In *Social and Biological Predictors of Nutritional Status, Physical Growth, and Neurological Development,* ed. L. Greene, 81–106. New York: Academic Press.

Mata, L., J. Urrutia, and A. Lechtig. 1971. Infection and Nutrition of Children of a Low Socioeconomic Rural Community. *American Journal of Clinical Nutrition* 24: 249–259.

McLaren, D. 1976. Concepts and Context of Nutrition. In *Nutrition in the Community,* ed. D. McLaren, 3–12. London: John Wiley and Sons.

Mensforth, Robert P. 1991. Paleoepidemiology of Porotic Hyperostosis in the Libben and BT-5 Skeletal Populations. *Kirtlandia* 46: 1–47.

Mensforth, Robert P., C. O. Lovejoy, J. W. Lallo, and G. J. Armelagos. 1978. The Role of Constitutional Factors, Diet and Infectious Disease on the Etiology of Porotic Hyperostosis and Periosteal Reactions in Prehistoric Infants and Children. *Medical Anthropology* 2(1): 1–59.

Nathan, H., and N. Haas. 1966. "Cribra orbitalia": A Bone Condition of the Orbit of Unknown Nature. *Israel Journal of Medical Sciences* 2: 171–191.

Perzigian, Anthony J., P. A. Tench, and D. J. Braun. 1984. Prehistoric Health in the Ohio River Valley. In *Paleopathology at the Origins of Agriculture,* ed. M. N. Cohen and G. J. Armelagos, 347–366. New York: Academic Press.

Pindborg, J. J. 1982. Aetiology of Developmental Enamel Defects Not Related to Fluorosis. *International Dental Journal* 32: 123–134.

Sarnat, Bernard G., and I. Schour. 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.

Scrimshaw, Nevin 1991. Iron deficiency. *Scientific American* (October): 46–52.

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

Smith, P., O. Bar-Yosef, and A. Sillen. 1984. Archaeological and Skeletal Evidence for Dietary Change during the Late Pleistocene/Early Holocene in the Levant. In *Paleopathology at the Origins of Agriculture,* ed. M. N. Cohen and G. J. Armelagos, 101–127. New York: Academic Press.

Stuart-Macadam, Patricia 1987. Porotic Hyperostosis: New Evidence to Support the Anemia Theory. *American Journal of Physical Anthropology* 74(4): 521–526.

———. 1989. Nutritional Deficiency Disease: A Survey of Scurvy, Rickets and Iron Deficiency Anemia. In *Reconstruction of Life from the Skeleton,* ed. M. Y. Iscan and K. A. R. Kennedy, 201–222. New York: Alan R. Liss.

Suckling, Grace 1989. Developmental Defects of Enamel: Historical and Present-Day Perspectives on Their Pathogenesis. *Advances in Dental Anthropology* 3(2): 87–94.

Sulzer, J. L., H. H. Wesley, and F. Leonig. 1973. Nutrition and Behavior in Head Start Children: Results from the Tulane Study. In *Nutrition, Development and Social Behavior,* ed. D. J. Kallen, 73–242. DHEW publication no. Washington, DC: NIH.

Sutphen, J. L. 1985. Growth as a Measure of Nutritional Stress. *Journal of Pediatric Gastroenterology and Nutrition* 4: 169–181.

Swardstedt, Torsten 1966. *Odontological Aspects of a Medieval Population from the Province of Jamtland/Mid-Sweden.* Stockholm: Tiden Barnangen, AB.

Sweeney, E. A., J. Cabrera, J. Urritia, and L. Mata. 1969. Factors Associated with Linear Hypoplasia of Human Deciduous Incisors. *Journal of Dental Research* 48: 1275–1279.

Sweeney, E. A., and M. Guzman. 1966. Oral Conditions in Children from Three Highland Villages in Guatemala. *Archives of Oral Biology* 11: 687–698.

Sweeney, E. A., J. A. Saffir, and R. de Leon. 1971. Linear Enamel Hypoplasias of Deciduous Incisor Teeth in Malnourished Children. *American Journal of Clinical Nutrition* 24: 29–31.

Tanner, James M. 1986. Growth as a Mirror of the Condition of Society: Secular Trends and Class Distinctions. In *Human Growth: A Multidisciplinary Review,* ed. A. Demirjian, 3–34. London: Taylor and Francis.

Ubelaker, Douglas H. 1981. *Human Skeletal Remains: Excavation, Analysis, Interpretation.* Chicago: Aldine.

Vyas, D., and R. K. Chandra. 1984. Functional Implications of Iron Deficiency. In *Iron Nutrition in Infancy and Childhood,* ed. A. Stekel, 45–59. New York: Raven Press.

Wood, James W., G. R. Milner, H. C. Harpending, and K. M. Weiss. 1992. The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples. *Current Anthropology* 33: 343–358.