

# 11. Cartesian Reductionism and Vulgar Adaptationism: Issues in the Interpretation of Nutritional Status in Prehistory

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*Abstract:* Paleonutrition studies can provide insights into the dynamics of life in past societies. Those insights, however, are at risk because of an increasing focus on short-term adaptations. In this paper I review three short-term focused interpretations of indicators of nutritional status. I argue that those interpretations share a reductionistic and decontextualized approach to human biology, while failing to consider the long-range and systemic consequences of compromised nutritional status.

## Introduction

The ultimate value of homeostatic reactions cannot be judged, therefore, until all of the consequences of such changes have been recognized.

—Dubos 1978:79

The only biological adjustment we can make to deleterious environmental factors is not true adaptation but a form of tolerance achieved at the cost of impaired functioning.

—Dubos 1978:78

In order to provide a basis for discussion of the effects of undernutrition on prehistoric groups, this paper was originally meant to review the functional consequences of undernutrition in contemporary peasants. Although this “ethnobiaeological” approach to undernutrition is still an implied focus, the overriding theme has changed.

At the 1991 American Anthropological Association meeting I presented a paper in a symposium on the possible intersections between medical ecology and political economy. Hans Baer, who had coorganized the symposium and who spoke before me, made the point that medical ecologists had focused on

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finding adaptations, and perhaps in the process had missed quite a bit, including great human cost and suffering and the systemic links between affliction and political-economic processes.<sup>1</sup> In response, I challenged that that characterization was not true for prehistoric medical ecology (including paleonutrition and paleoepidemiology). Recent publications suggest that I may have spoken too soon.

The purpose of this paper is to assess a recent trend in international nutrition and paleonutrition in which signs of compromised nutritional status are viewed as evidence for adaptation. I suggest that those reinterpretations, rather than providing new insights, reify an old Cartesian and mechanistic view of human physiology. Relying on the hope that the human body can homeostatically adapt to insults and deprivations, the theoretical model suggests a disease process in which an insult invades the body and the body homeostatically adjusts. All other systems are disconnected in Cartesian terms from the readjusting subsystem. In the most extreme form, that thinking suggests a vulgarization of the concept of adaptation: signs of stress are seen as adaptations for no other reason than that they exist in stressed but surviving organisms.

The practical implication of that view is serious. In pursuit of adaptations, the functional costs to the individual and to larger social units are ignored. There is no attention to the consequences of not being able to adapt. Furthermore, whereas the interaction between insult and host is usually assumed to lead to adaptive readjustment, there is little need to comprehend, much less change, underlying conditions.

In this paper I summarize new interpretations of the meaning of three nutritional status indicators. First, I review Neiburger's (1990) assessment that linear enamel hypoplasias indicate poor dietary status. Neiburger's comments are used only as a brief example of an extremely mechanistic view of the relationship between dietary intake and physiological perturbation. Stuart-Macadam's (1992) self-titled "new perspective" on porotic hyperostosis and iron deficiency is then discussed. Her view of physiology is seemingly that it is accommodating without costs.<sup>2</sup> Lastly, I review Seckler's (1982) thesis that the mild-to-moderately malnourished, as evidenced by short stature, are actually "small but healthy." Rather than as a sign of stress, Seckler reinterprets small body size to be a sign of healthy, homeostatic adjustment. This older example, played out in the very real world of international nutrition, comes complete with well-honed politics of support for the status quo and provides a third example of excess adaptationism.

## Signs of Stress or of Adaptation: Contested Interpretations

### *Linear Enamel Hypoplasia and Mechanistic Thinking*

Extrapolations as to nutritional or social stress based on the incidence of enamel hypoplasias occurring in extinct populations are assumptions

based on little scientific fact and should . . . be discouraged.

—Neiburger 1988:253

In a comment on a study of linear enamel hypoplasias (LEH) in Neanderthals, Neiburger states general objections to paleonutritional inference from LEH frequencies. He believes that Ogilvie and her coworkers' (1989) study of LEH in Neanderthals is "an error prone interpretation" and more generally that enamel hypoplasia is "a poor indicator of dietary stress" (1990:231). Neiburger proposes that there is a sort of mass amnesia amongst anthropologists regarding the etiology of enamel hypoplasias. Apparently because of their lack of training in the rigors of hard sciences such as dentistry (Neiburger's field), anthropologists are especially subject to uncritically following the latest trends, which in this case relates LEH to nutritional status. Paleopathologists, in his view, uncritically consider enamel hypoplasia to be a sort of perfect assay of dietary adequacy. In particular, Ogilvie and colleagues (1989) are wrong to infer nutritional problems in Neanderthals from high LEH prevalences. Inferences, Neiburger exhorts, cannot be based on an imperfect indicator.

The specifics of Neiburger's comments are easily refuted. LEHs are usually considered to be indicators of nutritional status during development, not dietary status (Goodman 1991; Goodman and Rose 1991). The difference between the two concepts is critical. By diet, Neiburger seems to mean what is eaten. However, nutritional status is 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). It is the end result of numerous factors affecting access to and utilization of nutrients. Nutritional status is a more complex concept than dietary intake. LEH does not directly reflect diet; however, hypoplasias do provide a window into the past physiological activity of ameloblasts, the enamel-forming cells, which are affected by diet and other conditions that contribute to the nutritional status of the organism.

What is more serious and disturbing about Neiburger's evaluation is the suggestion that a one-to-one relationship between insult and physiological response is likely. He is seemingly looking for an easily interpreted, invariable, and unchanging relationship between cause and effect, between pathogenic organism and disease. However, disease processes are complex; one rarely finds a one-to-one relationship between exposure to an insult and disease. Host and pathogen dialectically interact (Levins and Lewontin 1985). The disease process is not linear and not invariant. Neiburger eventually implies that further studies using LEH should cease, given the imperfect relationship. Of course, that logic would stop nearly all medical research.

In summary, Neiburger's critique of inferences made on the basis of LEH correctly points to the need for paleopathologists to pay attention to studies of LEH among living individuals and not to be overly specific about etiology. However, stripped of his misinterpretation of the concept of nutritional status, Neiburger's view of disease etiology remains simplistically mechanistic. He implies that human physiologies should have invariant responses. In the following sections I will try to show that his mechanistic view sets the table for considering stress indicators as adaptations.

*Anemia, Porotic Hyperostosis, and Vulgar Adaptationism*

The acceptance of the two concepts, that diet is of little importance to the development of iron deficiency anemia, and that iron deficiency is an adaptive response to stress, has a profound effect on the interpretation of porotic hyperostosis.

—Stuart-Macadam 1992:44

Recent reinterpretations of the cause and adaptive significance of porotic hyperostosis in prehistoric skeletons, as embraced in the preceding quote, extend a mechanistic view of physiology to the problematic of considering pathology as a sign of adaptation (see also Kent et al. 1991). In a paper titled “Porotic Hyperostosis: A New Perspective,” Stuart-Macadam (1992) proposes that paleopathologists have improperly interpreted porotic hyperostosis to be secondary to a dietary deficiency, most likely iron. In her view, there has been little concern for nondietary factors. Stuart-Macadam proposes that “within the context of this new perspective porotic hyperostosis is seen not as a nutritional stress indicator, but as an indication that a population is attempting to adapt to the pathogen load in its environment” (1992:39). What is actually new about this new perspective, and more importantly, what is correct and useful?

What is not new is the interpretation that diet is not always the key to the etiology of anemia. Most researchers would agree that diet and nondietary factors are of potential importance. In some cases parasitism (and parasite load) is certainly the main determinant of iron loss and anemia. That case is likely when parasitism leads to gastrointestinal bleeding (Solomons and Keusch 1981). However, dietary iron intake is frequently the key to iron status. For examples, Dallman and colleagues (1984) have shown that low iron intake is the main cause of anemia in infants, children, and women in the United States, and Calvo and Gnazzo (1990) similarly show that diet is the main determinant of anemia in urban Argentinean children. Just as focusing exclusively on diet is simplistic, focusing exclusively on parasitism is equally simplistic. That point of view has been well taken in the paleopathology literature, at least since the review by Mensforth and coworkers (1978; see also Mensforth 1991).

Up to this point the Stuart-Macadam critique of the literature on porotic hyperostosis parallels Neiburger’s critique of the LEH literature. Both suggest that diet is overemphasized, and both misinterpret the concept of nutritional status. Both are excessively Cartesian in their conception of physiological processes. Stuart-Macadam’s work differs in illustrating an excessive adaptationism.

Following the lead of Weinberg (1984) on iron withholding, Stuart-Macadam (1992) interprets iron deficiency as “*an adaptive response to stress*” (emphasis added). What is problematic about her interpretation is that it is a narrow view of adaptation. Weinberg (1984) makes the point that iron supplementation can lead to pathogen growth. He logically proposes that humans have evolved a physiological defense to some pathogens that includes iron-

withholding mechanisms. However, in Dubos's terms (1978), iron withholding should be seen as an adjustment rather than an adaptation. There is no clear evidence that hypoferremia increases the long-term adaptation of the organism, and there is not a clear relationship between iron withholding, chronic iron status, and porotic hyperostosis. Cartesian reductionism creeps in. It is as if iron can be sequestered from microorganisms without affecting the need for iron in the host.

Iron deficiency—sometimes even at levels where hemoglobin and hematocrits are normal, that is iron deficiency without anemia—leads to a suite of functional costs. A variety of organs and systems show structural changes with borderline iron deficiency. Vyas and Chandra advise that the wide spread costs 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" (1984:45). Those enzymes are aconitase, catalase, cytochrome C, cytochrome C reductase, cytochrome oxidase, formiminotransferase, monoamine oxidase, myeloperoxidase, peroxidase, ribonucleotidyl reductase, succinic dehydrogenase, tyrosine hydroxylase, tryptophan pyrrolase, and xanine oxidase (Vyas and Chandra 1984). Many of them are involved in vital functions such as DNA synthesis, mitochondrial electron transport, catecholamine metabolism, and neurotransmitter detoxification.

The consequences of iron deficiency on an organismal level can be divided into three areas: (1) resistance to disease, (2) work capacity/activity, and (3) cognition and behavior. Iron deficiency has a variety of effects on immunocompetence and infection. Especially noteworthy is its effect on cell-mediated immunity (Dallman 1987). 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; Sherman 1992; Vyas and Chandra 1984). In humans, iron supplements have led to a decreased prevalence of diarrhea and upper- and lower-respiratory infections. After reviewing and finding flawed studies of the supposedly protective effects of iron deficiency against infection, Vyas and Chandra conclude that "it can be stated with confidence that iron supplementation within physiological needs to prevent anemia or in doses necessary to correct anemia may reduce, but certainly not increase, the risk of infection" (1984:49).

Although the relationship between iron deficiency and cognition is harder to specify (partly because iron deficiency is often confounded with other nutrient deficiencies), Pollitt has shown that mild iron deficiency, without low hemoglobin, is associated with learning deficiencies (1987). Of particular note are changes in attention and memory control processes. Those results suggest that iron is a critical element for the normal functioning of the nervous system and that cognitive functions can be disrupted by relatively mild iron deficiency.

The effects of iron deficiency and anemia on work capacity are profound (Scrimshaw 1991). Work capacity has frequently been shown to be proportional to hemoglobin concentration. Anemic subjects cannot maintain the work times of normal subjects and reach a lower mean maximal workload. Anemic Guatemalan laborers performed much more poorly on the Harvard

Step Test than did their nonanemic peers, and the work output and pay of Indonesian rubber tappers correlate almost perfectly with their hemoglobin levels (Scrimshaw 1991). If decreased oxygen affinity and increased cardiac output are the “adaptive” responses to anemia, then those adaptations can cover for deficiency only when the organism is sedentary or at rest. That is, it is an adaptation that does not work well in the real world, perhaps unless one is very wealthy.

In a review of nutrition and host defense, Keusch proposes in response to Weinberg (1977) that the “longterm benefits of hypoferremia are entirely unknown. What is known is the cost—anemia” (1979:287). In light of that evaluation, it would be extremely problematic science, not to mention onerous public policy, to suggest that contemporary individuals with anemia are either adapting or have adapted. Is porotic hyperostosis a sign of adaptation or a sign of nutritional deficiency? I suggest that it is quite clearly a sign of nutritional deficiency from a variety of systemic consequences can be inferred.

### *Small but Healthy?*

The homeostatic theory postulates a *threshold* relationship. Smallness may *not* be associated with functional impairments. . . . the mild to moderately malnourished people in the deprivation theory are simply “small but healthy” people in the homeostatic theory (emphasis in original).

—Seckler 1982:129

Regulation of the rate of growth is likely an important defense mechanism against functional impairments due to malnutrition which would otherwise accompany poverty.

—Seckler 1982:133

Although the previous sections of this paper have focused on nutrition in the past, the debate over the interpretation of small body size has been waged in the arena of contemporary politics and sciences. That debate is reviewed here because it suggests a number of lessons for the study of nutritional status in the past, and it has the potential to spill over into the study of prehistoric nutrition.

Except for a limited number of ethnic groups, there is surprisingly little variation in group mean height attributable to *group-level* genetic differences (Dietz 1983; Graitcer and Gentry 1981; Habicht et al. 1974). Therefore, because of the universality of the growth pattern at the group level, it is possible to estimate prevalences of stunting in groups and to target groups at risk. With anthropometric data, nutritionists and international health researchers have a powerful tool for targeting groups at risk.

Malnutrition has traditionally been defined by degree of severity. Severe malnutrition is obvious from clinical signs. However, at the mild and moderate levels, growth (size) may be the only measurable sign of malnutrition. Heights and weights are frequently collected in international health programs, not only because they are easy measures to take but because they are sensitive

ones. They have the potential to identify malnutrition before it becomes severe and life threatening.

In 1980 the economist David Seckler challenged fundamental assumptions about the meaning and consequences of short stature. As quoted above, Seckler proposed that short individuals in developing countries are short because they have adapted to chronically marginal food availability; the body adjusts to low nutrient availability by reducing growth (1980, 1982). He further suggests that that homeostatic adjustment occurs without adaptive cost. The individuals are essentially "small but healthy" (1980:225). In fact, Seckler asserts that small individuals may be better adapted than larger individuals to an environment in which food scarcity is a chronic problem. His argument should sound vaguely reminiscent.

The implications of considering the formerly marginally malnourished to be small but healthy are profound. In India alone, where Seckler focused his analysis, his rethinking could "clear the books" of hundreds of millions of malnourished peasants. With a simple rethinking, the problem has seemingly gone away. Of course the problem of marginal malnutrition only appears to have been solved. The contested area directly concerns whether the small individuals are, indeed, healthy. If Seckler is correct, then it follows that there are few functional inferences that can be made on the basis of anthropometric data. The small are not marginally malnourished. They are healthy, homeostatically adjusted and adapted.

In a review of the literature on the interrelationship of diet, growth, and infectious disease, Martorell (1980) found that poor growth status is almost invariably associated with both marginal diets and increased disease. The synergetic relationship between diet and disease is often so strong that it is difficult to ascertain the precise cause of poor growth status (Martorell 1989; also see Tompkins 1988). Growth status seems to be a sign of the cumulative stress experienced by an individual, of which poor diet and disease are likely to be of main importance, and, parenthetically, is why growth is a measure of nutritional, not dietary, status. The primary proximate causes of short stature are impoverished diets and disease. To then affirm that short stature is adaptive implies a sanctioning of the presence of the conditions leading to short stature (Martorell 1989), which might ultimately be traced back to poverty and inequality in access to resources (McKeown 1988).

In addition to increased morbidity, poor growth status is associated with a number of other functional consequences. Chavez and Martinez (1982) have systematically addressed the functional cost of marginal diets and small body size in their nutrition-supplementation study. In the late 1960s pregnant mothers and their infants from Tezonteopan, Mexico, were divided into two groups: one received daily nutritional supplementation, and the other group did not. Chavez and Martinez found that the better-nourished/taller children had fewer disease episodes and especially had shorter durations of disease. As well, the better-nourished children displayed more exploratory behaviors,

cried less, and talked at an earlier age. Functional cost in terms of decreased activity and learning may be signaled by growth stunting.

A wealth of studies have shown that poor growth is related to an increased risk of mortality, the ultimate indicator of adaptive failure (see Alam et al. 1989; Chen et al. 1980; Lerberghe, 1988; Smedman et al. 1987). Alam and co-workers (1989) show that children in Bangladesh with small arm circumferences are 12 times more likely to die than children with more adequate arm circumferences. Lerberghe sums up his recent review of the relationship between childhood stunting and mortality by stating: "If both stunting and mortality are different outcomes . . . of a succession of stresses on children's health, then the measure of the height of children in a community is an even more valuable tool for evaluation" (1988:259). He continues: "The measurement of the prevalence of stunted children then becomes an operationally fairly feasible, be it indirect, measurement of poverty related disease frequency. . . . The prevalence of stunted children in a community appears to be a good overall indicator of the health status of a community of children" (1988:259).

Martorell persuasively summarizes the interacting scientific and political difficulties of the small-but-healthy hypothesis by asserting the following four points. First, children and adults in developing countries are short as a result of chronically poor diets and frequent infection during development. To consider stunting to be adaptive is to intimate that its causes—impoverished diets and increased exposure to disease—are desirable. Second, growth retardation, "rather than an innocuous response to environmental stimuli, is a warning signal of increased risk of morbidity and mortality" (1989:15). Third, the conditions that cause stunting also affect other functions such as cognition and work capacity. Finally, stunted girls who grow up short are at increased risk of delivering growth-retarded infants with a greater chance of dying (1989:15).

In thinking about how Seckler could have made such a nearsighted interpretation of the adaptive consequences of stunting, Pelto and Pelto (1989) suggest that he fell victim to the "quitting early" problem (also see Harris 1988). They propose that many scientists are guilty of quitting before they have evaluated the long-term causes and consequences of apparent adaptations.

The implication that can be derived from the "small but healthy" debate is that many paleonutritionists also quit too soon. We quit before systemically analyzing the implications of disease patterns on the individual and society. In a contemporary context, Chavez and Martinez (1982) have shown that moderately malnourished children may be sick as much as half their childhoods. It is difficult to comprehend the burden that such a level of illness must place on a mother in a developing community (Leatherman 1987), and there is no reason to suggest that the burden of illness was any less in most past societies. Just as LEH is imperfectly related to nutrition and health status, linear growth is also an imperfect measure. Nonetheless, they are both useful.



Just as anemia is not a cost-free homeostatic adaptation, small body size does not come without costs to other functional systems.

## Conclusions: Paleonutrition and Vulgar Adaptationism

Don't look for the meaning, look for the use.

—Wittgenstein's Aphorism

Don't look for the use, look for the abuse.

—Callahan's Corollary of Wittgenstein

The study of the diets of prehistoric peoples is a relatively benign undertaking. One need not worry that faulty theories or methods might lead directly to human suffering. The work of reconstructing prehistoric diets does not have much to do with how to choose a president or even with current infant mortality rates. A mistake, unlike the improper administration of anesthesia, will not lead to coma. On the other hand, there are links between the human condition now and in the past. Convictions about the past interact with convictions about the present. They reinforce each other. The purpose of this paper has been not only to explore connections between ideas about past and present nutritional status but also to consider implications of commonly held interpretations.

Recent interpretations of the meaning of nutritional-status indicators have in common an underlying assumption that physiological processes are mechanistic and homeostatic. Thus, dietary inadequacy and all that might follow from it can be adjusted to with little or no cost. Neiburger (1990) is befuddled by the lack of a clear and consistent relationship between dietary deficiency and the formation of enamel defects. Stuart-Macadam (1992) suggests that the cause of porotic hyperostosis is complex but misinterprets the organism's ability to withhold iron (and by extension porotic hyperostosis) as an adaptation to parasitism. These new explanations could be viewed as benign reinterpretations of what are commonly referred to as "stress indicators," reflecting a worldview in which physiology is seen as no more complex than house plumbing. However, I suggest that they are not totally benign, rather that they lead to an excess of emphasis on the organism's ability to adjust (after all it is still alive!) and an attribution of adaptation before all the costs have been accounted for.

Does it make a difference whether nutritional-status indicators such as LEH, porotic hyperostosis, and anthropometry are considered signs of stress or of adaptation? On one level, the difference might not be so great if both perspectives considered the costs of adjustment and the cause of the need to adjust, that is, if the biological process were considered in full social and political-economic context. However, such uses, or one should say misuses, of adaptation tend to cast a narrow beam of light, whereas a focus on stress casts a broader beam. A focus on stress redresses an imbalance of excess adaptationism by concentrating on the costs and limits of adaptation (Goodman et al.

1988). Lastly, to paraphrase Martorell (1989), it is ludicrous to focus on adaptation when the conditions are of sociopolitical exploitation and impoverishment. Clearly, the only adaptation is to change the system, not to adjust to it.

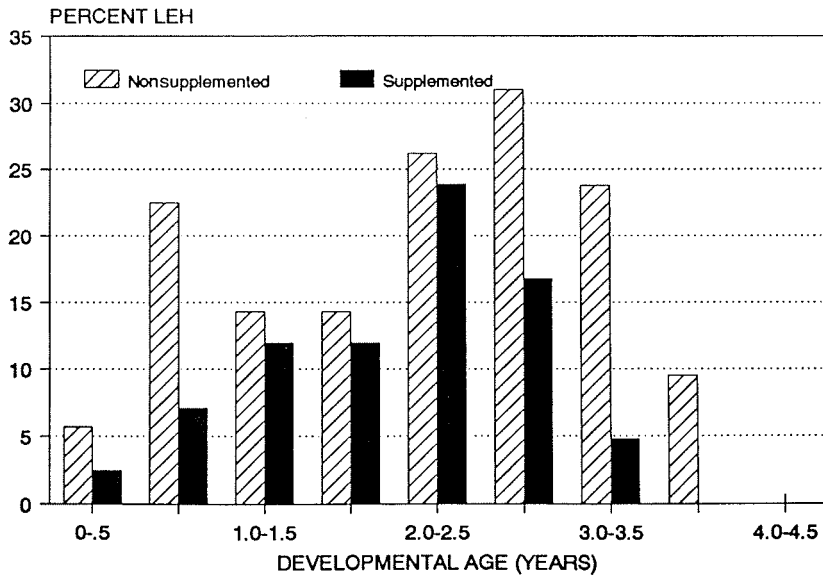
Fortunately, it is possible to test whether these signs of undernutrition are adaptive. Data can resolve the differences in perspective. For permanent and deciduous enamel developmental defects, Goodman and Armelagos (1988) and Blakey and Armelagos (1985), respectively, have found that defects at Dickson Mounds are associated with decreased survival. What is significant about their work is the effort to see if the indicator is associated with a change in other measures of morbidity and mortality. In the Dickson context, it would be problematic to see enamel developmental defects as signs of adaptation, unless one were to say that death is an adaptation. Conversely, Harris lines are infrequently associated with increased morbidity (Goodman et al. 1984). For those reasons one might question their use as a stress indicator. Clearly, more research should be done on the potential morbidity and mortality consequences associated with LEH, anemia, and growth faltering in prehistoric populations.

Returning to ethnoarchaeology and the original theme of this paper, I find that one of the exciting things about the three indicators discussed is that they all have parallels in contemporary nutrition work. Growth and LEH can be studied in both prehistoric and contemporary individuals, and one can make inferences between blood measures of iron status and skeletal markers of iron deficiency. Thus, by studying the context and meaning of nutritional-status indicators in contemporary settings, it may be possible to make surer inferences about their meaning in prehistory.

A brief ethnoarchaeological example involves work on enamel defects among Nahuatl children in highland Mexico (Goodman et al. 1991). Our research was conducted with the same children who had been involved in the previously discussed dietary supplementation study of Chavez and Martinez (1982). We found that nonsupplemented children had approximately twice the LEH frequency of the supplemented children. Differences were especially great during the first and fourth years (Figure 11-1). The data suggest that change in nutritional intake can greatly decrease the frequency of LEH. Interestingly, LEH rates around ages two and three in the supplemented group were quite high, possibly due to endemic parasitism.

The prevalence of defects in the nonsupplemented children is similar to rates often observed in prehistoric groups. In Tezonteopan, that prevalence of LEH is associated with high respiratory and diarrheal morbidity between the ages of one and three years, growth stunting, and effects on childhood activity and learning. From those data we can begin to pose the question of whether similar rates in prehistoric populations might infer comparable functional consequences.

Stressful environmental conditions, which are frequently linked to political and economic processes, can lead to an increase in recoverable signs of stress. Those signs are significant to interpreting adaptation because they suggest a concurrent lowering of resistance to disease, decreased work and productive



**Figure 11-1.** *Frequency of linear enamel hypoplasias on permanent maxillary central incisors by half-year developmental periods, nonsupplemented vs. nutritionally supplemented children, Tezonteopan, Mexico (Goodman et al. 1991).*

capacity, less interest in and ability to engage in social and discretionary activities, and decreasing fertility and fecundity (Allen 1984; Buzina et al. 1989). If anything, however, they suggest maladaptation instead of adaptation. But, it is not the organism that is maladapted; rather, the sign of disrupted development more accurately suggests that the social system has failed to buffer against stress.

Paleonutrition studies can provide unique insights into the dynamics of past societies and the evolution of the human condition. Unfortunately, however, those insights are at risk because of a narrow focus on presumed short-term adaptations. Paleonutritionists need to focus on the full process, including the conditions—which often involve political-economic inequalities—that lead to the need to adapt and the long-term consequences of adjustments.

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## Notes

1. I interpret Baer to mean by "adaptation" a response that increases tolerance or adjustment to a given environment. That response may have survival value. I prefer to call such responses "adjustments" or "coping strategies" rather than adaptations, unless the long-term consequences are known to be "adaptive."

2. In private correspondence Stuart-Macadam has made clear that she agrees with the costs of anemia. However, that is less clear from published material.

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