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On the Interpretation of Health from Skeletal Remains[1]

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Wood et al. (CA 33:343–58) argue that unambiguous health inferences from paleodemographic and paleopathological data are impossible because of the inherent, ubiquitous, and essentially unresolved dual problems of selective mortality and hidden heterogeneity in frailty risks. They conclude that "lesion frequencies cannot be interpreted in a straightforward fashion" (p. 354) and, for example, that "the skeletal evidence pertaining to the transition from hunting-and-gathering to settled agriculture is equally consistent with an improvement in health and a deterioration in health" (p. 343; see also p. 357). In also concluding, in contradiction to the above, that "Better health makes for worse skeletons" (p. 356), they seem to turn upside down the reconstruction of health from skeletal evidence.[2] Is the reconstruction of health from skeletal remains as paradoxical as proposed?

I suggest that what they consider paradoxical only appears so, because they focus on single rather than multiple indicators of health and misinterpret the goals of paleoepidemiology, and that the models they construct do not reflect biological realities and credible cultural contexts. Instead of providing a useful critique of method and theory in paleoepidemiology and paleodemography, Wood et al. demonstrate the dangers of scientific snobbery and present an example of a biological anthropology that does not consider cultural contexts. What appears as an osteological paradox is in fact not one.

The premise of selective mortality is that "we never have a sample of all the individuals who were at risk of disease or death at a given age, but *only of those who did in fact die at that age*" (p. 344, emphasis mine). The chief implication is said to be that "the observed frequency of pathological conditions should overestimate the true prevalence of the conditions in the general population."[3] This inference, the basis of nearly all to follow, is an oversimplification at best and incorrect at worst. Most indicators are actually represented by lesions that signify survival for some time after the morbidity event. This is true for activity-induced lesions, for periostitis and porotic hyperostosis (Larsen 1987, Martin, Goodman, and Armelagos 1985, Mensforth 1991), and for enamel hypoplastic defects (Goodman and Rose 1990). As an indication of the confusion surrounding this concept, I note that several commentators cite Ortner's (1991) proposition that skeletons with infectious lesions are from healthy individuals (see esp. Eisenberg, p. 359, and Katzenberg, p. 361) as similar to Wood et al.'s explication of selective mortality, whereas Ortner's hypothesis is diametrically opposed to it. For Ortner, pathology indicates survival of a stress or disease event;[4] for Wood et al., pathology indicates nonsurvival.

That the dead are a "select sample" was fully recognized by Cook and Buikstra (1979; see also Cook 1981, 1990), who found a positive relationship between age at death and enamel defect frequencies (older individuals initially had more defects) and the reverse association at subsequent ages (younger individuals had more defects) and attributed it to the decreasing probability that those who were exposed to severe stress would die before forming a defect. Cook (1981) points to the importance of understanding biological process (the age-related probability of survival of a stress severe enough to cause an enamel defect) and mortality patterns in the study of enamel defects. When assessing lesions in skeletal series, one is constrained by the fact that archaeologically derived bones and teeth are observed at one point in

1. I thank the following for helpful comments on this essay: George Armelagos (Emory University), Debra Martin (Hampshire College), Robert Mensforth (Cleveland State University), Jerome Rose (University of Arkansas), Alan Swedlund (University of Massachusetts), R. Brooke Thomas (University of Massachusetts), and Dennis Van Gerven (University of Colorado).

2. There is a fundamental inconsistency here; Wood et al. suggest that we cannot yet interpret population-level changes in morbidity and mortality because the data are equally consistent with opposite interpretations but also that "better health makes for worse skeletons" (that is, the data are apt to be interpreted in a counterintuitive way). It is this interpretation that is taken away by at least one commentator (see Eisenberg, p. 365).
3. It is not clear what Wood et al. mean by "condition." Seemingly a "condition" is either a pathological process or an environmental stress or/constraint. Nonetheless, it is true to either interpretation that we often have survivors of the "condition" and therefore will often undernumerate (not overnumerate) those who were exposed.
4. Except for cases of perimortem violence and the like, the osseous marker is related to a biological response to the stress (Ortner 1991). Ortner proposes, therefore, that signs of infection are indications of adaptation. While I agree that responses are evidence of survival, I do not accept the idea that pathologies are best interpreted as signs of "health" and "adaptation" (see Goodman et al. 1988, Goodman 1991); tests of this hypothesis do not tend to support it (Mittler et al. 1992, Rose 1985).

time, when the individual died. The key question is how that which is observable at the time of death relates to processes during life. For example, stature and long bone lengths are useful general and cumulative measures of nutritional status even though they may not clearly reflect nutritional status at a specific time (Bogin 1988, Gibson 1990, Jelliffe and Jelliffe 1989, Sutphen 1985, Tanner 1986, Zerfas, Jelliffe, and Jelliffe 1985).[5] In contrast, indicators of growth disruption such as linear enamel hypoplasia provide a time-specific indication of past physiological perturbations (Sarnat and Schour 1941). The two measures produce different but entirely complementary information. Examination of multiple indicators of health and nutrition helps distinguish among otherwise seemingly paradoxical interpretations. The bones and teeth of the dead reflect conditions at death *and* conditions during life. They do tell tales about life processes, albeit ones that can be difficult to interpret.

Wood et al. miss the mark in their explication of the dynamics of selective mortality partly because they are committed to the notion that the goal of paleoepidemiology is understanding cause of death. Paleoepidemiologists are in fact seldom concerned with cause-of-death analysis,[6] which is exceedingly difficult for a number of reasons and is not at all essential to saying something about health and adjustment in past populations. Further, whereas Wood et al. consider disease of interest because it affects an individual's risk of death, paleoepidemiologists find disease worth studying even when it has no real or measurable effect on mortality (Alland 1970, McElroy and Townsend 1989). Minor infirmities and decrements in nutritional status can affect such things as work capacity and reproduction and have widespread cultural and social repercussions (Allen 1984, Haas 1991, Leatherman 1987). Wood et al.'s demographic emphasis seems to have impeded their understanding of the range of uses of morbidity information derived from skeletons.

According to Wood et al., the key implication of hidden heterogeneity in risks is that it is "virtually impossible to interpret aggregate-level age-specific mortality rates in terms of individual risks of death" (p. 345) and "there is no way to infer individual risks of death from aggregate mortality data in the absence of prior theory that specifies the way in which frailty varies among individuals" (p. 349).[7] Here they overstate the uninformativeness of group frailty. Clearly, a change in group frailty of *x* increments does not signify that all individuals change in frailty by *x* increments or even that there is any change in frailty of any individuals. There is, however, a mathematical tethering of individual and group frailty: if group frailty changes, then either the size of subgroups or the frailty of one or more subgroups must change. Furthermore, one can begin to interpret the individual significance of aggregate frailty if one has a theory about the distribution of frailty and some idea of how groups might change in size and how exposures might change subgroups' health risks—in short, contextual information.[8] In any case, paleoepidemiologists are rarely interested in individuals. While there is certainly interesting and informative frailty variation on an individual level, it is not key to the interpretation of group differences.[9] Wood et al. have overlooked the development of an epidemiological approach to the study of prehistoric health and nutrition including, among other things, a change in focus from disease in *individuals* to disease in *groups*.

In summary, Wood et al.'s premise that those working with prehistoric skeletal remains "presuppose that direct relationships exist between statistics calculated from archaeological skeletal series (e.g., skeletal lesion frequencies and mean age at death) and the health status of the past populations" (p. 343) is a stale characterization of paleoepidemiology. It has long been known that many issues can complicate and cloud interpretations (see, e.g., Huss-Ashmore, Goodman, and Armelagos 1982, Gilbert and Mielke 1985, Iscan and Kennedy 1989, Saunders and Katzenberg 1992). The interpretation of

5. There is some debate over the interpretation of achieved size as measured in cross-sectional growth surveys (Bogin 1988, Eveleth and Tanner 1976, Gibson 1990, Sutphen 1985, Tanner 1978). While anthropometry is generally considered to reflect nutritional status, dietary intake interacts with morbidity, genetics, and other factors in mercurial fashion. Weight-for-age is usually considered more reflective of undernutrition near the time of measurement (current), whereas height-for-age is usually considered reflective of chronic undernutrition (Gibson 1990, Sutphen 1985, Zerfas et al. 1985).

6. Wood et al. unfairly criticize Cohen for comparing incidences of disease in skeletal populations by suggesting that this violates laws of proportional mortality. Cohen was discussing morbidity, not causes of death. Whereas causes of death are mutually exclusive (one only dies once), diseases are not. Finally, the focus on cause of death presupposes that one dies from only one thing. What is usually entered as "cause of death" on a death certificate is only the proximate cause, not the sole or underlying cause of death.

7. Wood et al. refer to both Vaupel, Manton, and Stallard's (1979) definition of frailty as "an individual's relative risk of death compared to a standardized cohort risk" and Vaupel's (1990) more general "individual *biological characteristics* [emphasis mine] associated with persistent differences among individuals in susceptibility, propensity, or relative risk with respect to disease or death" (n. 4). Finally, they define frailty (p. 345) as simply variation in "susceptibility to disease and death," which may arise from both genetic and environmental conditions. Is frailty a biological (genetic) characteristic, or is it also subject to environmental conditions? If the latter, how does it differ from risk relative to cohort risk?

8. Many of Wood et al.'s examples are designed to show that mortality will shift the aggregate frailty of a group by selecting out the most frail. One apparently paradoxical situation is that infant mortality is higher than childhood mortality. However, risk of diarrheal disease follows an almost inverse age pattern (initially low and then high). They use this example to suggest that the more frail die earlier (having been exposed to less disease). At older ages there are fewer deaths (despite high disease) because the most frail have been culled out. Of course, this interpretation does not address age-related patterns of resistance to disease and death (older individuals are more resistant because they have more fully developed immune and other defense systems).

9. Greater clarity on the formation of subgroups has long been an important challenge to archaeology and bioarchaeology (Saitta 1992). Many researchers have tried to examine the health of different subgroups, such as males and females and different social classes (see, e.g., Powell 1988, Rathbun and Scurry 1991, Wilkinson and Norelli 1981).

paleoepidemiological data is indeed fraught with difficulties, but the issues raised by Wood et al. are less new, less significant, and less troublesome than they suggest.

Wood et al.'s nihilistic assessment of paleoepidemiology and paleodemography is made to appear probable by a series of paradoxical examples. A reassessment of these examples makes it clear, however, that they only seem paradoxical because of incomplete information and untenable assumptions.

Their first example is that of an imaginary population in which three groups can be distinguished: group A never experiences stress and has no skeletal lesions, group B experiences moderate stress, sufficient to cause skeletal lesions, but most individuals survive, and group C suffers heavy stress, resulting in death before lesions are formed. The groups' stress exposures range from low (group A) to high (group C). The paradox, according to Wood et al., is that in terms of lesion frequencies group C appears to have experienced low stress (and is indistinguishable from group A). They use this example to introduce the difficulty of reconstructing population prevalences of pathological conditions from skeletal lesions and the paradoxical "possibility that individuals displaying lesions may actually be healthier than at least some individuals without lesions" (p. 345).[10]

Can this paradox be resolved? Wood et al. provide information on mortality but do not use it. Individuals in group C died before they could mount a bony response, whereas individuals in the other two groups survived. The distribution of ages at death distinguishes group C from the other two groups (and the above-noted lesion distribution distinguishes groups A and C from group B).[11] Combining the two indicators, we find that the low-stress group A is correctly identified by the combination of low morbidity and low mortality, the intermediate-stress group B is correctly identified by high morbidity and low mortality, and the high-stress group C is correctly identified by low morbidity and high mortality (table 1). The key lesson here is the importance of examining lesion frequencies in relationship to mortality and, more generally, of the use of multiple indicators of stress.

A second apparent paradox concerns the relationship between stature and poor health. As previously noted, growth stunting is often considered as indicating undernutrition secondary to factors such as poor dietary intake and high disease loads in both contemporary (Haas 1990; Martorell 1980, 1989; Sutphen 1985) and past (Huss-Ashmore, Goodman, and Armelagos 1982, Johnston and Zimmer 1989, Martin, Goodman, and Armelagos 1985, Saunders 1992) populations. On the basis of the inverse association between size at a given age and probability of dying, Wood et al. suggest, in contrast, that mean stature decreases with decreased environmental stress (p. 351) and intimate broad applicability: "In general, the frequency of apparent 'stunting' among the dead is uninformative about the distribution of stature or relative health among the living. . . . Once again, proportional mortality is a poor guide to population prevalence" (p. 351).

TABLE 1
Lesion Prevalence and Mortality Profiles of Three Differentially Stressed Groups

	Group A	Group B	Group C
Presumed stress intensity	Low	Moderate	High
Lesion prevalence	Low	High	Low
Mortality	Low	Low	High

The fundamental flaw here is an altogether unsubstantiated view of the relationship between environmental conditions, human growth response, and mortality. Wood et al. cite inverse associations between size and probability of dying to suggest that short children are inherently frailer than tall children. Stunted children do indeed consistently suffer from increased morbidity and mortality (Alam, Wojtyniak, and Rahaman 1989, Chen, Chowdhury, and Huffman 1980, Kielman and McCord 1978, Lerberghe 1988, Smedman et al. 1987), and this is one of the reasons that anthropometric data are attributed significance for public health (Martorell 1989). However, the association between stature and mortality is confounded by the causal link of both with impoverished and stressful living conditions. Stunting on a group level is often a sign of a growth-sparing response to environmental conditions (Acheson 1960, Tanner 1986). Whereas there is an increased risk of death in stunted individuals, the association between stunting and mortality is based on the prerequisite that stunted individuals have been exposed to environmental stress that has robbed them of nutrient reserves (Acheson 1960, Martorell 1989, Tanner 1986).[12]

Wood et al.'s mistake of not focusing on the bio-ecological process is akin to the implication that those who visit a physician are frail because of some inherent propensity to meet with a physician. The associational snapshot ignores the process leading individuals to seek medical care. Similarly, Wood et al. mistakenly assume causality between stunting and frailty and subsequently miss the confounding of environmental influences on probability of stunting *and* mortality. Their static view

10. It should be clear that this is an imaginary example; the actual dynamics of the relationship between stress, lesion frequency, and mortality are far from constant.

11. Because of demographic nonstationarity, mean age at death is usually difficult to interpret in terms of life expectancy at birth, but this problem can be solved in large part by estimation of growth rates (Johansson and Horowitz 1988) and need not apply to this theoretical example.

12. There are many interesting and important issues surrounding the relationship between growth, environmental conditions, and mortality. For example, it is not yet clear how much the association between small size (at birth or early in life) and increased mortality is due to inconsistent differential exposure to stress or early "damage." This controversy, in fact, is part of what stimulated the study of Goodman and Armelagos (1988) highlighted by Wood et al.

of the relationship between stunting and mortality overlooks the significant biological and social processes leading to this association.

A third example is drawn from real data reviewed by Wood et al. in order to suggest a counterintuitive explanation. Using data from Dickson Mounds in Illinois, Goodman and Armelagos (1988) present results showing an inverse association between enamel hypoplasias and mean age at death that is strongest in the Middle Mississippian, a fully agricultural group. The enamel hypoplasias developed between approximately 3.5 and 7.0 years, and all individuals were adolescents or adults when they died. Individuals with two or more enamel hypoplasias (who are thought to have survived multiple bouts of physiological disruption) have a mean age at death of 21.8 years, whereas those without a defect have a mean age at death of 37.5 years. Multiple enamel defects are associated with a decreased mean age at death of 15.7 years during the Middle Mississippian. Three processes are suggested as possible explanations of the association between early stress (indicated by enamel hypoplasias) and decreased mean age at death: (1) consistent individual-level variation in biological susceptibility to physiological perturbation (individuals who are most susceptible to stress during childhood also being most susceptible during adolescence and adulthood),[13] (2) a biological-damage hypothesis (early stress decreases physiological resistance), and (3) a social-status-differentiation/social-susceptibility process (both high hypoplasia and high mortality are due to lower social status throughout life). The last of these is considered the most likely; the cultural context of the study, excluded from Wood et al.'s discussion, suggests the possibility of increased social differentiation through time (Rothschild 1979). Wood et al. advance a fourth hypothesis—that the individuals with hypoplasias and lower mean age at death were the *less* frail and more socially advantaged.

This hypothesis is supported by the results of a mathematical model with the following assumptions and starting points: The population at any point in time is made up of two groups, one advantaged and the other disadvantaged. The advantaged group "did not entirely escape childhood insult" and survived to develop enamel defects. It went on to have high fertility and an increase in mean life expectancy. "Individuals with observable lesions were *principally* [emphasis mine[14]] from the less frail (i.e., advantaged) group, and they had lower mean age at death than the disadvantaged group" (p. 355).

The fatal flaws of this model are that nearly all members of the advantaged group have linear enamel hypoplasia and that there are "no dental signs of stress among the survivors of childhood in the disadvantaged population" (p. 355). These make the results anything but counterintuitive. Beyond its obvious circularity, the model has other flaws. First, the life-expectancy differences and especially the fertility differences are extreme, especially given that all individuals in the analysis survived to adolescence. Second, the model outputs an intrinsic rate of increase of .029 in the advantaged group, which is unusually high for even an "advantaged" prehistoric population. Third, the outputted mean ages at death for individuals more than 15 years old are 44 years for the disadvantaged group and 53 years for the advantaged, both figures far higher than the observed mean age at death of 32.5 years (Goodman and Armelagos 1988:939). Fourth, the model outputs that the advantaged group initially comprises 10% of the total population and after 150 years comprises almost the whole of the population.[15]

Is this scenario probable? The short answer is an emphatic no. I know of no situation in which a clearly advantaged group, living or past, has more hypoplasia than a disadvantaged group, not to mention any situation in which an advantaged group has exceedingly high hypoplasia rates (greater than 90%) and the disadvantaged group has none. On the contrary, enamel hypoplastic defects have repeatedly been shown to be more prevalent under conditions of lower socioeconomic status, increased exposure to disease, and decreased access to food and other basic resources (see reviews by Goodman and Rose 1990, 1991). For example, work in two communities in Mexico has shown that enamel hypoplasias are inversely related to economic status (Goodman et al. 1992) and dietary intake at the time of enamel formation in living children (Goodman, Martinez, and Chavez 1991). Lukacs presents data showing that males in a situation of son preference have fewer enamel defects than the disadvantaged (neglected) females (p. 362; see also Lukacs and Joshi 1992). The conclusion of this section, "Better health makes for worse skeletons" (p. 356), is unsupported because Wood et al. fail to account for biological processes. Their model is mathematically possible but unlikely because it has no biological basis.

A last example is provided in Wood et al.'s reply to commentators. Their point in introducing it is to show that what they call "decrepitude" may not be informative of risk of death (or frailty). They state that the presettled San had high infant and childhood mortality although they "appear[ed] to be in good condition" (p. 367), whereas the settled San had reduced early-life mortality but were "decrepit." From this example one is led to believe that there is a paradoxical relationship between decrepitude and mortality. Although this is possible, it does not mean that health is uninformative when it is not clearly associated with mortality. We are interested in health for a number of reasons, only one of which is its relationship to mortality (Alland 1970, Armelagos, Goodman, and Jacobs 1978). Health can pro-

13. Interestingly, this process might be phrased in terms of variation in genetic frailty.

14. Lesions come not "principally" but *entirely* from the advantaged group.

15. Wood et al. are not precise about the input parameters for their model. From the time = 0 results of figure 9 it appears that all members of the advantaged group have enamel defects. From the results at time = 150 years it appears that the advantaged group comprises over 90% of the population.

vide information about local and political-economic conditions, and it can have functional significance independent of its relationship to mortality (see Haas 1990, Leatherman 1992). Interest in mild-to-moderate malnutrition is based not so much on its links to mortality as on its effect on functional capacity and quality of survival.

Further, Wood et al. would surely chastise paleoepidemiologists were they to base their analyses upon skeletal "decrepitude." Nomadic !Kung were often described as in good physical condition in the 1960s and 1970s (Howell 1979), but, of course, it is likely that researchers saw what they presupposed (Wilmsen 1989). In fact, examination of some of the empirical evidence, such as growth rates and adult statures, suggests that the San were not in such good physical condition (Truswell and Hansen 1976, Wilmsen 1989). Conversely, researchers today, concerned about the recent proletarianization of the San, may see them through lenses of decrepitude. The point here is not whether the San are healthier now or in the past but that this notion is too vague and subjective to be taken seriously.

Finally, we are given only enough information to make the situation appear paradoxical. For example, we are told nothing about differences in diets and nutritional status of the settled and pre-settled !Kung. No effort is made to solve the paradox or, at minimum, to rule out alternative hypotheses. Wood et al. seem to provide data only if it heightens the sense of paradox. This is an abuse of model building.

Efforts to draw inferences from osseous signs of morbidity and mortality face a number of challenges. In their concluding section "Is There Hope?" Wood et al. suggest four tasks in the interpretation of skeletal data and, despite protestations to the contrary, are utterly pessimistic about the possibility of their fulfilment. The bone that is thrown to archaeological osteologists—the fourth task, to be shared with "all anthropologists"—is to develop better understanding of the cultural context of frailty. This is a flawed view of the current state of bioarchaeology. Wood et al. propose a noninteractive and nondynamic vision of the scientific process; it is no wonder that so many of the commentators were discouraged by their article. Prior to its "revelations," paleoepidemiology faced calls for more precise diagnosis in considering the significance of pathological conditions for groups and cultures and calls for reburial of most human remains. Now Wood et al. add that it must wait for the assembly line of science to produce frailty equations from which it can choose.

Health data from past populations can provide insight into current conditions as much as research on contemporary groups can inform studies of the past. The flow of knowledge is two-way.[16] Furthermore, it is unlikely that geneticists, physiologists, and epidemiologists will work in the service of understanding health in the past unless this is part of their own research agenda. The trend, overlooked by Wood et al., is for bioarchaeologists themselves to be expert in fields such as epidemiology, nutrition, and physiology. Rather than standing by as passive consumers, they are actively studying skeletal and dental health measures in contemporary contexts.

Although Wood et al. do not provide any counsel for working out apparent paradoxes, paleoepidemiologists have been actively doing so. I end this commentary by focusing on some of the research that is addressing the data needs to which Wood et al. point. Although they pay lip service to "significant changes in orientation and sophistication" in paleopathology since the 1960s, they do not say much about these changes other than that paleopathology has moved from "particularistic concerns with individual lesions" to a "population-based perspective" (p. 344). In addition to turning to populations, three key advances are (1) a multiple-indicators approach to reconstructing prehistoric health, (2) the development of models for contextualizing skeletal indicators of stress, and (3) the development of multiple lines of research to help clarify the cultural contexts of skeletal lesions and the biological processes leading to their development.

The inference that health declined at Dickson Mounds at the time of agricultural intensification is justified because data from different methods and sources provide cross-confirmation (Goodman, Lallo, Armelagos, and Rose 1984). The agricultural group's skeletons display not only dramatically more enamel hypoplasias but also dramatically more Wilson bands, growth stunting, porotic hyperostosis, periostitis, trauma, osteoarthritis, and osteophytosis. Porotic hyperostosis and periostitis in subadults increase three-to-fourfold through time, and there is an approximate doubling in the prevalence of enamel defects. Thus we can examine stress in both survivors and nonsurvivors. Given these archaeological and paleoecological data, one can begin to think about the processes that produced them. Whereas data from a single source are likely to be open to a number of equally consistent interpretations, the use of multiple types of data greatly reduces the number of likely interpretations and consequently the chances of error in interpretation.[17]

16. This can, of course, be overdone. For example, many researchers, including Wood et al., point to problems in overstating similarities between "generic hunter-gatherers" of the 20th century and "generic hunter-gatherers" of the prehistoric past. Specific contexts are key.

17. Flaws can be consistent across measurements. For example, I contend that Cohen (1989) has overstated the case for declining health as due to agricultural intensification (Goodman 1991). The data are rather consistent in showing that health declines with intensification of agriculture, but increasing social stratification is also associated with agricultural intensification. Multiple sources support the agricultural-intensification-and-social-stratification hypothesis. If we could do a better job of understanding markers of social status, then it would be theoretically possible to know who benefited and who was disadvantaged by agricultural intensification. A supposition is that agricultural populations might, for the sake of modeling, be divided into two groups, a small elite that gains greater access to resources (and greater health and nutritional status) and one or more other classes that likely suffer from the advent of agriculture. Thus, a population-level decline in health could be due to the expansion in size of the "underclass" or a change for the worse in its mean frailty.

Hooton's (1930) work is so widely referenced in part because he adopted a multiple-indicators approach to health at Pecos Pueblo. Since his time nearly all key studies have used multiple approaches. To name but a few, this perspective is central to the work of Armelagos (1969) in Nubia, Angel (1984) in the eastern Mediterranean, Buikstra (1984) and Cook (1984) in the Lower Illinois River Valley, Storey (1992) at Teotihuacan, Larsen (1990) at Mission Santa Catalina de Guale, Martin (Martin et al. 1991) on Black Mesa, Ubelaker (1984) in Ecuador, Walker (Walker and Hollimon 1989) on the California coast, and Powell (1988) at Moundsville. Wood et al.'s view that the most important role of paleoepidemiology is the examination of health change with the origins of agriculture overlooks the exciting uses of skeletal data in reconstructing contact-period and historical-period health (Rose 1985, Verano and Ubelaker 1992).

Wood et al. seem to imagine that paleoepidemiologists operate without thinking about the context of their data—about how it fits together in a model. They cite Keyfitz's aphorism "No model, no understanding" and imply that it fits paleopathology, but the opposite is true. Buikstra (1977) was perhaps the first to model how bioarchaeological data might be used and interpreted within a regional framework of archaeological research, and researchers at the University of Massachusetts (Huss-Ashmore, Goodman, and Armelagos 1982; Goodman, Martin, Armelagos, and Clark 1984; Martin, Goodman, and Armelagos 1985; Goodman et al. 1988) have developed a model (cited by Wood et al.) specifically designed for interpreting multiple signs of stress in skeletons.

I fully agree with Wood et al. that further work needs to be done on osseous responses to disease. Bioarchaeologists such as Magennis (1990) have explored the formation and resorption of Harris lines as seen in serial radiographs in children from Denver. L. Barbian (University of Florida) is exploring the consequences of arthritis in an elderly contemporary population with a view to better understanding the consequences of arthritis in the past. Mensforth has examined vertebral fractures in a historical series (Mensforth and Latimer 1989). Stuart-Macadam (1989, 1992) has tried to understand the degree of anemia that will cause porotic hyperostosis and cribra orbitalia in the crania of contemporary individuals. The majority of my own research has been devoted to studies of enamel developmental defects in living populations in Mexico (Goodman, Martinez, and Chavez 1991), Guatemala (May, Goodman, and Meindl 1992), and Egypt (Goodman et al. 1993).

In conclusion, Wood et al. make a number of errors in their discussion of the significance of selective mortality and hidden heterogeneity in frailty. They grossly overstate the significance of hidden frailty because they fail to understand the diminished importance in the paleoepidemiological agenda of individual-level analysis and of linking morbidity to mortality. Whereas they are correct in the notion that the dead are a select group, they fail to grasp the details of the process linking events in life to the chances of displaying a skeletal lesion at death. Their contribution to the literature is less than they assume. Their models ignore cultural processes, contradict known biological processes, leave out key information, and depend on false assumptions and ultimately prove mathematically possible but biologically highly improbable. Although it is useful to point out possible paradoxes and counterintuitive explanations, it is important to separate the probable from the suspect. Scientific snobbery is a poor substitute for a well-grounded critique.

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