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Notes and Comments

Paleoepidemiological Inference and Neanderthal Dental Enamel Hypoplasias: A Reply to Neiburger

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In his recent comment, Neiburger (1990) proposes that Ogilvie and co-worker's (1989) study is "an error prone interpretation of Neanderthal dental hypoplasias to infer foraging success" and more generally that enamel hypoplasias are "a poor indicator of dietary stress" (1990:231). Ogilvie and Trinkaus (1990) have addressed Neiburger's remarks that were pointed at their original research (Ogilvie et al., 1989). The purpose of this comment is to clarify the more generic issues raised by Neiburger (1990) on the specificity of dental enamel hypoplasias and their use in paleoepidemiological inference.

Neiburger's initial problem stems from his failure to distinguish among terms such as dietary intake (he uses the term "dietary stress," an ambiguous term that he never defines), nutritional stress, nutritional status, physiological stress, and foraging success. These terms involve different levels of specificity and refer to processes operating at different levels. Though related, they cannot be used interchangeably. For example, *dietary intake* refers to nutrients consumed, whereas *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), the end result of numerous factors affecting access to and utilization of nutrients. Failure to understand these concepts and the distinctions among them is likely to have contributed to Neiburger's bewilderment about the inferences drawn by Ogilvie et al. (1989). I agree that there are few situations in which enamel hypoplasias could be unambiguously related to the intake of a specific nutrient. However, there are many situations in which enamel hypoplasias have been associated with decreased nutritional status (Goodman and Rose, 1990).

Neiburger backs up his assertion that enamel hypoplasias are a poor indicator of "dietary stress" by citing a text from 1962 and two articles from the 1940s (both improperly referenced), and including a list of seven potential causes of enamel hypoplasias. The listing is particularly ineffectual. The first and fifth items on the list respectively refer to hereditary factors and local trauma. Both of these causes can be differentially diagnosed—a fact that Neiburger should be aware of as their differential diagnosis was included in Weinmann and coworkers (1945), one of his references. Items two through four and six include a mixed bag of causes, mainly related to nutrition and disease. All items comfortably fall into a group of factors contributing to nutritional status. Neiburger's seventh item refers to the use of dental appliances such as orthodontic braces, the use of which might cause a blemish that mimics a hypoplasia. Such blemishes may also be differentially diagnosed. Of course, most researchers would presumably agree that orthodontic appliances were likely to have infrequently adorned the teeth of even the most advanced Neanderthals.

The most important corrective is that the issue of specific etiology that Neiburger focuses upon is actually a non-issue. Following nearly all research on enamel hypoplasias in prehistoric populations, Ogilvie and coworkers acknowledged that these defects result from a wide variety of conditions (Cutress and Suckling, 1982). Their use of enamel hypoplasias is not a measure of a specific nutrient deficiency, or other specific condition, but as a permanent record of the achievement of a *level of general metabolic disruption sufficient to disrupt ameloblastic physiology* (Kreshover, 1960).

Having established the frequency and pattern of these defects in Neanderthals, Ogilvie and co-workers (1989) suggest that nutri-

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tional stress may have been an important etiological factor. This inference is based on sound differential diagnosis. The pattern of defects suggests systemic stress (rather than hereditary or local causes). Further, the demographic and skeletal data from the Neanderthals show that infectious disease may have been rare. Ogilvie and co-workers make a logical inference from these data to a nutritional etiology. Neiburger, on the other hand, rather than proposing an alternative etiology based on some aspect of Neanderthal biology, culture, and environment, provides a laundry list of causes, without reference to the conditions of the study population. Ogilvie and Trinkaus rightly responded to Neiburger by proposing that interpretation of paleoepidemiological patterns is more likely to benefit from careful consideration of likely alternatives, rather than producing a "mere listing of all the possible sources of the lesion" (1990:233).

Subsumed in the above is a larger misunderstanding of population versus individual levels of analysis and the degree to which nutritional status measures, including enamel hypoplasias, can be confidently linked to specific causes. Fortunately, Neiburger's statement that hypoplasias cannot be predicted with reliability (I assume he meant accuracy or validity) is false. In fact, the better one knows individuals' illness and nutritional histories, the better one could predict the development of enamel defects (a proposition that goes back at least to Sarnat and Schour [1941]). While the predictions are unlikely to be perfect (there are too many factors that contribute to ameloblastic disruption and the expression of an enamel defect) the lack of perfect prediction from a measurement in individual cases should not preclude its use on a population level (Goodman and Rose, 1990). It is also very difficult to know the precise cause of growth faltering (Sutphen, 1985). However, inferences can be made on a population level and growth monitoring remains a key tool for evaluating nutritional status (Sutphen, 1985).

In summary, much of Neiburger's commentary stems from his lack of familiarity with the current literature on paleoepidemiology and dental enamel development, and a parallel lack of appreciation of basic concepts and perspectives in epidemiology and anthropology. Questioning basic assumptions and comparing perspectives should provide a basis for a useful exchange, and clinical training and experience could provide useful insights into the nature of prehistoric health. However, these insights will be slow in coming unless clinicians make an effort to understand anthropological concepts and perspectives and to stay abreast of developments in the relevant literatures.

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I appreciate Dr. Goodman's interest and thoughtful response concerning this complicated subject. His letter typifies the great danger we face as scientists in producing

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