Chapter 2

Low Bone Mass in Past and Present Aboriginal Populations

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1. Introduction

A slight and gradual loss of bone mass is characteristic of all aging primates, if they live long enough (Garn, 1970; Burr, 1980). Nevertheless, the observation of reduced bone mass among ancestral human skeletal remains is limited to relatively recent populations. Since the domestication of plants roughly 12,000 years ago, skeletal remains from disparate parts of the world have occasionally shown low bone mass. Perhaps earlier populations did not suffer age-related bone loss because they died at young ages (Pfeiffer, 1990), or perhaps their diet or lifestyle facilitated effective bone maintenance. Past human populations were more dependent on local natural resources and their own physical labor for subsistence, a cultural pattern maintained by only a few geographically isolated aboriginal groups today. These "anthropological populations" have been portrayed as natural paradigms whose dietary habits might be studied as representations of our species' natural "set point" for nutritional requirements, and against which we might evaluate modern regimens and their biological consequences (Eaton *et al.*, 1988; Eaton and Nelson, 1991).

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With regard to calcium and bone mass, it has been argued that our mammalian, primate and earlier hominid ancestors consumed a diet much higher in bioavailable calcium than did our more recent ancestors of the last ten millennia. One scenario traces a 200 million year evolutionary sequence from insectivory, through frugivory, to omnivory, the latter stage incorporating increasingly greater amounts of scavenged or hunted meat with the appearance of the genus *Homo* some 2 million years ago (Eaton and Nelson, 1991; Leonard and Robertson, 1992). The suggestion that these calcium-rich diets were compatible with achieving a high peak bone mass is consistent with data documenting skeletal robusticity in early forms of *Homo* (Ruff, 1988; McHenry, 1992).

The study of cortical and trabecular bone mass and symptomatic fracture in aboriginal populations can help us understand the phenomenon of low bone mass as it appears in modern, industrial populations. Aboriginal peoples can have less varied diets, less genetic diversity, comparatively low socioeconomic variability, and more homogeneous—albeit sexually dimorphic—mechanical histories (Ruff, 1992). Thus, causal links between low bone mass and environmental variables should be more clearly perceived. This might allow more specific hypotheses to be formulated prior to testing in contemporary clinical settings.

Nevertheless, there are obvious limitations to the analysis of diet and bone mass in skeletal populations, even beyond those imposed by their intrinsically cross-sectional research designs. The attribution of fundamental parameters such as age and sex to individual adult skeletons is imprecise. Also, we may have only general information regarding a particular group's dietary regimen. The methods of bone mass quantification applied in the clinical arena may not be easily transferable to dry, long-buried bone tissue. Indeed, the burial environment may significantly alter skeletal tissues at both the microscopic (Hanson and Buikstra, 1987) and elemental (Hancock *et al.*, 1989) levels. The prehistoric dietary habits represented may themselves have become extinct, leaving no modern analogs and thereby relegating information about osteopenic side-effects to simple curiosity. Finally, where living populations are concerned, there is the possibility of confounding age- or sex-specific secular change in bone mass associated with culture contact and acculturation.

In the following sections, we first consider the methods used to infer or measure low bone mass in anthropological populations. This is followed by a summary of the evidence from prehistoric populations and historic Arctic groups, both of which are described as having skeletal mass below expected values. Nutritionally-based hypotheses dominate the explanations of low bone mass in these populations; however, such models now seem at odds with epidemiological data from modern populations (Hegsted, 1986).