Despite recent criticisms, straightforward interpretations of relative pathology frequencies in two or more past populations can generally be made from their cemeteries without concern for paradoxical interpretations.

Ortner (1992, 1998) notes that visible pathology forms slowly in bone. As a result, a skeleton without visible pathology might represent an individual who never suffered a particular disease or one who suffered so severely that (s)he died before the pathology affected the skeleton. Individuals displaying pathology might paradoxically be healthier and longer-lived than those who display no pathology. Visible pathology in cemeteries might increase not because more people got sick but because more people lived long enough for pathology to register in their skeletons.

The “osteological paradox” (Wood et al. 1992; see also Milner et al. 2000) argues that cemeteries are not necessarily fair samples of the living populations from which they are derived, because chance, differential frailty, and selective mortality may all affect the cemetery samples, so frequencies of pathology in cemeteries have no predictable relationship to the pathology of populations they represent. If this were true, no conclusions about pathology in a prehistoric population could be drawn from its cemetery.

Several arguments can be offered in rebuttal of both arguments, at least where comparisons between populations are attempted (see also Cohen 1997).

First, Goodman (1993) and Wright and Yoder (2003) suggest that using multiple independent lines of evidence may circumvent the problems of the so-called paradox. Good candidates are ethnographic observation and uniformitarianism. Cohen (1989) provides summary paleopathological data (as directly interpreted), modern epidemiology, and uniformitarian logic used to reconstruct ancient disease patterns. The three lines of evidence converge on the same conclusions, strengthening conclusions from the direct interpretation of skeletons. Skeletal pathology frequencies largely accord with expectations from other lines of analysis, suggesting that neither Ortner’s argument nor the paradox argument applies.

Second, the paradox argument overstates the importance of differential frailty and selective mortality on cemetery populations, at least with regard to the relatively chronic conditions that appear in skeletons and that may contribute to the probability of death without themselves being lethal.
Those with a specific pathology in any cemetery come from one of two groups: individuals whose deaths are hastened ("selected") by their chronic condition, and those whose deaths are random relative to a particular pathology. For example, if individuals with or without pathology are killed by bad luck or "outside events" with no regard to differential frailty (for example, a cave roof collapses), those dead will be a random sample of the living and will, on average, reflect the population's rates of pathology accurately. But those dying "selectively" will also reflect the frequency of pathology among the living because the selected group reflects the strength of selection multiplied by the original frequency of the pathology in the population. Despite selective mortality, the original pathology frequency in the living population exerts a powerful influence on its frequency in the cemetery (compare Wright and Chew 1998).

If the strength of selection changes dramatically from population to population, this argument might not hold. Epidemics of acute disease that kill rapidly without scarring the skeleton are prone to "paradoxical" interpretation. No skeletal pathology may reflect rapid death. But these diseases have widespread effects only relatively late in prehistory. Therefore, the skeletal invisibility of epidemics should result in underestimation—rather than negation—of the pattern of declining health commonly observed in history.

We can interpret stress markers of childhood events, such as enamel hypoplasia, in the same way. High frequencies of hypoplasia could indicate a high frequency of stress episodes. Hypoplasia can also be considered an indication of good health, as implied by Ortner (1992, 1998).

Adults who show no hypoplasia cannot have died before the stress marker could form: that adult must not have experienced the stress. Therefore, when adults are compared, few hypoplasias mean few stresses, whereas more hypoplasias mean more stresses. Ortner would argue that absence of hypoplasia among adults, however, reflects high childhood mortality instead of health. But it does not. The assumption that more LEH means more health problems, not survival, is almost certainly correct. Which is more likely—that children died of stresses that adults did not even record in teeth, or that they died of stresses that others survived but recorded? Surely the latter is more probable. More hypoplasia in adults must mean higher, not lower, childhood mortality.

Third, if cemetery populations are misleading samples, where are the paradoxical results? There ought to be many results that make no sense vis-à-vis the results of theoretical expectations. But paleopathological data repeatedly match the expectations of data from ethnoarchaeological studies and uniformitarian assumptions. Skeletal signs of anemia and infection (including tuberculosis, syphilis, and leprosy) increase when and where we would expect.