Pathological skeletal lesions and selective mortality in the Postclassic population of Cholula

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Abstract

The skeletal remains pathological lesions are difficult to interpret because of different factors related to the heterogeneity masked and selective mortality (Wood *et al.* 1992). A new multi-state model (Usher 2000) provides a means to address the selective mortality factor and thus determine the relationship between the presence of pathological lesions in skeletons and the risk of death for a given population. This model was applied to 309 skeletons from the city center Postclassic Prehispanic Cholula. The observations were made by searching cribra orbitalia, porotic hyperostosis, enamel hypoplasias incisors, canines, first and second molars, and proliferative lesions of long bones. While the presence of most of the lesions was associated with an increased risk of death in the town of Cholula, the model indicated that the canine enamel hypoplasias and fibula proliferative lesions had no effect on mortality.

Keywords: paleopathology, osteological paradox, selective mortality, Cholula.

Resumen

Las lesiones patológicas en restos esqueléticos son difíciles de interpretar debido a los diferentes factores relacionados con la heterogeneidad enmascarada y la mortalidad selectiva (Wood *et al.* 1992). Un nuevo modelo de estados múltiples (Usher 2000) proporciona un medio para direccionar el factor de mortalidad selectiva y así determinar la relación entre la presencia de lesiones patológicas en esqueletos y el riesgo de muerte para una población dada. Este modelo se aplicó en 309 esqueletos del Posclásico provenientes del centro urbano prehispánico de Cholula. Las obser-

vaciones se hicieron buscando criba orbitaria, hiperostosis porótica, hipoplasias del esmalte en incisivos, caninos, primeros y segundos molares, y lesiones proliferativas en huesos largos. Mientras la presencia de la mayoría de las lesiones fue asociada con un incremento en el riesgo de muerte en la población de Cholula, el modelo indicó que las hipoplasias del esmalte de caninos y lesiones proliferativas de la fíbula no tuvieron efecto sobre la mortalidad.

PALABRAS CLAVE: paleopatología, paradoja osteológica, mortalidad selectiva, Cholula.

INTRODUCTION

In 1992, Wood *et al.* published an article entitled "The osteological paradox", in which they discussed how the related problems of *hidden heterogeneity* and *selective mortality* could result in equivocal interpretations of pathological lesions. Although a number of other researchers (Ortner 1991; Cook and Buikstra 1979; Goodman and Armelagos 1988; Stuart-Macadam 1988) had previously pointed out that skeletal lesions take time to form and that they, therefore, represented at least short-term survival of a disease process, the implications of this realization had not been made explicit in the literature. Wood *et al.* (1992) argued that as a result of the related issues of hidden heterogeneity and selective mortality, skeletal lesions bear a complex relationship to health.

Individuals vary in their underlying frailty as a result of genetic, developmental, environmental, and socioeconomic factors (Vaupel and Yashin 1985; Vaupel *et al.* 1979; Alter and Riley 1989). *Frailty* in this sense refers to biological characteristics of individuals that are associated with their relative risk of becoming ill or dying (Vaupel 1990). While some variation in frailty is attributable to factors such as age and sex that are observable, other differences in frailty in a population result from less obvious and less measurable causes, such as genetic variation in susceptibility to diseases, differences in nutrition, or even differential exposure to environmental stresses. These unseen differences in the risk of death are what are referred to as *hidden heterogeneity* (Wood *et al.* 1992). When heterogeneity in frailty is hidden, it is exceedingly difficult to control for it in paleopathological analyses of skeletal remains. However, failure to take into account hidden heterogeneity in susceptibility to disease or death may result in erroneous interpretations of skeletal lesions (Wood *et al.* 1992).

As a result of heterogeneity in frailty, both seen and unseen, mortality is selective, meaning that the frailest individuals in a population have the greatest risk of death (Wood *et al.* 1992). In other words, a skeletal sample is not a random sample of the living population from which it was drawn. Within any birth cohort, those individuals with higher frailty are more likely to die and enter the mortality sample than others of their sex and age. Furthermore, those individuals in a population with higher frailty are likely to be selected out of the population at younger ages than those with lower frailty (Wood *et al.* 1992).

As a result of hidden heterogeneity and selective mortality, pathological lesions in skeletal remains cannot necessarily be interpreted in a straightforward manner. Because skeletal lesions take time to form, frailer individuals may die before infection or malnutrition can leave their marks (Wood *et al.* 1992). Individuals must have at least some degree of immunological or genetic resistance to these insults in order to survive long enough for paleopathological indicators of stress to become apparent. However, an individual who develops a pathological lesion clearly has a different health status than someone who was immunologically able to avoid or suppress the insult before it resulted in skeletal involvement, or someone who was not exposed to the stress at all (Wood *et al.* 1992).

Prior to Wood *et al.*'s article, most paleopathological analyses had assumed that skeletal populations with higher frequencies of pathological lesions were less healthy than those that had lower frequencies of such lesions. In contrast, the osteological paradox suggests that it might also be the case that populations with higher frequencies of lesions consisted of somewhat healthier individuals who lived at least long enough for the skeletal lesion to form. Populations with lower frequencies of lesions could consist of particularly healthy individuals that were able to avoid or fight off the disease before it could impact the skeleton, or they could consist of particularly unhealthy individuals that died before the disease could result in skeletal involvement. While the presence of pathological skeletal lesions does indicate something about the health of the individual, the message being conveyed is quite complicated. Without some way to model the distribution of frailty in the living population or to determine how the presence of skeletal lesions relates to mortality, the frequency of pathological lesions in a population is essentially uninterpretable (Wood *et al.* 1992; Milner *et al.* 2000).

The osteological paradox has not yet been completely resolved, but proposals exist for dealing with the issue of hidden heterogeneity (Usher 2000; Milner *et al.* 2000) and selective mortality has now been successfully addressed (Cook and Buikstra 1979; Goodman and Armelagos 1988; Boldsen 1991, 1997; Milner *et al.* 2000; Usher 2000; Boel 2001). Studies of selective mortality have demonstrated that the relationship between particular pathological lesions and mortality may be population specific (Usher 2000; Ferrell Thomas 2003; De Witte 2006). Thus, it is imperative that the exact nature of this relationship be examined in the course of paleopathological investigations of individual populations. In the current study, a model that specifies how pathological lesions affect the risk of death was applied to 309 Postclassic skeletons from the urban center of Cholula to identify how the presence of porotic hyperostosis, cribra orbitalia, enamel hypoplasias, and proliferative lesions of the skeleton influenced mortality in this population.

The Usher model

Bethany Usher (2000) has proposed a comprehensive multistate mortality model to address the issues of hidden heterogeneity, selective mortality, and nonstationarity. As skeletal collections are typically biased samples, it is impossible to extrapolate the frequency of pathological lesions in the living population directly from their frequency in the skeletons under study; therefore, paleodemographers need a means of "working backwards" from the biased skeletal sample to reconstruct the living population that produced it. The Usher model is just such a tool, allowing paleopathologists to overcome the osteological paradox by identifying the relationship between pathological lesions and the risk of death and to then make inferences about the frequency of skeletal lesions in the once-living population (Usher 2000; Milner *et al.* 2000). The following description of the model is derived from Usher (2000) and Milner *et al.* (2000).

The full model consists of four states: well, ill, healed, and dead. "Well" indicates that there are no observable pathological lesions on the skeleton, "ill" indicates active pathological lesions on the skeleton, and "healed" indicates healed pathological lesions on the skeleton. Of course, as many diseases do not leave lesions on the skeleton, the model

cannot test the effects of those illnesses. Rather, the model is evaluating the effects of lesions that can be observed by paleodemographers (Usher 2000). Individuals are born into the population in the "well" state through a process that takes into account the growth rate and resolves the issue of nonstationarity. Each newborn is randomly assigned a frailty level from a distribution of frailty. As they age, individuals can either move into the "ill" state or they can die without ever becoming "ill". If they become "ill", individuals can die from that state, or they can move into the "healed" state and eventually die from there. Transitions between the living states are dependent upon the individual's frailty, as are the hazard rates for moving from a living state to the dead state. The hazards of passing from the ill and healed states to the dead state are modeled as being proportional to the hazard of moving from the well to the dead state. This allows the issue of selective mortality to be addressed. Obviously, individuals are observed by the paleodemographer only once they have entered the "dead" state, so the age at death of each individual, as well as observations of active and healed lesions in the skeletal sample provide the data necessary to estimate the parameters of the model using maximum likelihood analysis (Usher 2000; Milner *et al.* 2000).

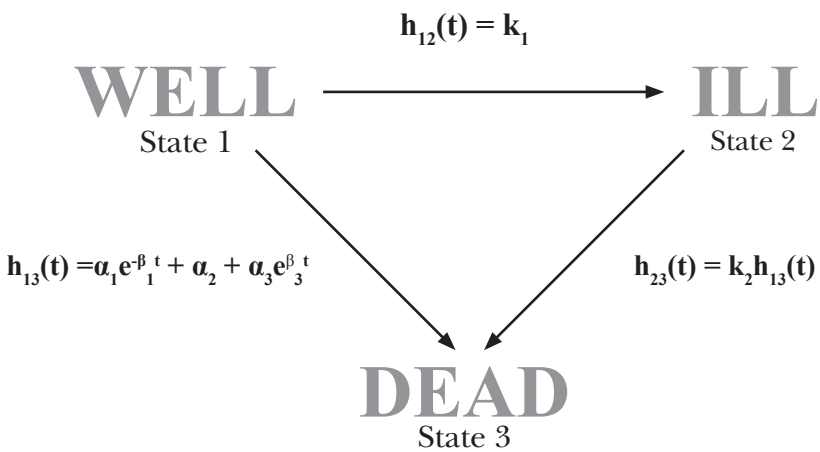

Figure 1. Usher's reduced model. The baseline risk of death, $h_{13}(t)$, is the Siler model. The constant k_1 is the hazard of acquiring a skeletal lesion, and k_2 represents the proportional risk of dying with a skeletal lesion (redrawn from Usher 2000).

Unfortunately, the full four-state model is very complicated and includes a significant number of parameters that must be estimated. Consequently, it has not yet been fully tested (Usher 2000). However, Usher (2000) did implement a reduced stationary three-state version of the model and was able to detect the impact of selective mortality on several different types of skeletal lesions. In this simplified model, frailty is not considered, so hidden heterogeneity cannot be addressed. The reduced model includes only the "well", "ill", and "dead" states (figure 1). No distinction is made between active and healed lesions, which precludes paleopathologists from determining if healed pathological lesions have a different relationship to mortality than unhealed lesions.

In this reduced model, Usher used the Siler model as the hazard of dying from the "well" state. The Siler model (Gage 1988, 1989, 1990, 1994; Gage and Dyke 1986) is a parametric model of mortality that offers a very generalized description of human mortality. It consists of three additive components:

$$h(t) = \alpha_1 e^{-\beta_1 t} + \alpha_2 + \alpha_3 e^{\beta_3 t}$$

where t refers to the age-at-death, and $h(t)$ refers to the hazard of dying at some age t. The first component ($\alpha_1 e^{-\beta_1 t}$) captures the high mortality of infancy and early childhood, which then declines rather rapidly. The second component (α_2) represents a constant age-independent mortality, and the third component ($\alpha_3 e^{\beta_3 t}$) is senescent risk, or the increasing risk of death with age (Gage and Dyke 1986; Gage 1988). In these equations, $\alpha_{1,2,3}$ and $\beta_{1,3}$ are parameters, or constants, that are to be estimated from the skeletal sample itself. The Siler model, therefore, provides us with a means to estimate $h(t)$ for skeletal samples, which is age-specific mortality, given survival to age t. Usher uses the Siler model to represent baseline mortality in the population.

The hazard of becoming "ill" at some age t is modeled as a constant, k_1, meaning that the risk of developing a lesion is assumed to be constant across all ages (Usher 2000). This assumption is simplistic, as some individuals undoubtedly had a greater risk of becoming ill than others. However, this simplified assumption provides a starting point for addressing the issue of selective mortality. The hazard of dying once "ill" (k_2) is modeled as proportional to the baseline risk of death from the "well" state, so the risk of

death is either increased or decreased, depending upon the effect of the pathological lesion (Usher 2000). The likelihood of dying with a skeletal lesion is, therefore, defined by the equation below, whose derivation can be found in Usher (2000: 24-29):

$$L_{a|\mathrm{I}=1} = k_1 k_2 h_{13}(a) (S_{13}(a))^{k_2} \int_0^a e^{-k_1 a_{12}} (S_{13}(a_{12}))^{a-k_2} \mathrm{d}a_{12}$$

where the subscripts refer to the transition between states and *a* refers to age. This reduced model was used to examine the relationship between mortality and the presence of certain skeletal lesions in the Postclassic population of Cholula.

The parameters of the Siler model, as well as the k_1 and k_2 constants are estimated using a maximum likelihood estimation program (*mle*) (Holman 2002). A value of k_2 significantly greater than one indicates that the presence of a pathological lesion increases the risk of death. A value of k_2 significantly less than one indicates that the pathological lesion reduces the risk of death, and a k_2 value of one indicates the lesion has no effect on mortality (Usher 2000). In tests of the reduced model using simulated data, Usher (2000) found that k_1 and k_2 values were accurately captured by the *mle* program. Paleopathological analyses that have included the Usher model can be found in Usher (2000), Ferrell Thomas (2003), De Witte (2006), De Witte and Wood (2008), De Witte and Bekvalac (2010), and Bullock Kreger (2010).

The osteological assemblage

The osteological assemblage used in this analysis consists of 78 Cholulteca II (AD 900-1325) skeletons and 231 Cholulteca III (AD 1325-1500) skeletons excavated from the archaeological site of Cholula during the 1967-1970 field seasons of the Proyecto Cholula (López *et al.* 1976). These burials were recovered from beneath house floors and plazas of a low-status Postclassic residential area that overlay Classic Period ceremonial structures near the Great Pyramid (López *et al.* 1976). Although ceremonial burials, sacrifices, and corporal segments were also excavated from this site, these skeletons were *not* included in the current study.

Previous paleodemographic and paleopathological investigations concerning culture, health, and living conditions have focused on both prehispanic Cholula, as well as on cultural evolution in prehispanic central Mexico (Camargo *et al.* 1999; Camargo and Partida 1998; Gómez de León 1998; Lagunas 1973, 1994; López 1972, 1973; López *et al.* 1970, 1976, 2002; López and Salas 1989; Márquez 1996; Márquez *et al.* 2002; Márquez and Hernández 2006, 2007). In general, these studies have found that cribra orbitalia, porotic hyperostosis, periostitis, osteomyelitis, enamel hypoplasias, and other pathologies were relatively frequent in the Cholula skeletal sample. Márquez *et al.* (2002) used the health index proposed by Steckel and Rose (2002) to evaluate the Cholula skeletons and compare health in the collection to that of other prehispanic Mexican populations. They concluded that the urban environment of Cholula negatively affected the health of its inhabitants. While the health index does offer a productive means of standardizing paleopathological observations, it does not specifically address the problems raised in the osteological paradox.

In the following article, I will refer to the results of the paleopathological analyses in reference to their implications for the "population of Cholula". However, the reader should keep in mind that this is merely shorthand and that the skeletal collection under study represents only one habitational zone within the urban center and not the entire Postclassic population of Cholula. Variation in demographic and pathological processes due to genetic variation, status differences, and living conditions within the city undoubtedly existed.

Paleodemographic analysis

An integral part of Usher's model is the age estimations of skeletons in the population. In order to identify how the presence of a skeletal pathology affects the risk of death, it is necessary to know the age at which each individual died. Only individuals whose age at death could be determined (280) were included in the model. Age determination in juveniles was based on dental development, the union of epiphyses, and, when necessary, diaphyseal length. As rates of dental development seem to be relatively invariant across populations (Smith 1991), tooth formation and eruption were preferentially used when possible. Dental ages were assigned to

juveniles in accordance with the criteria presented in Ubelaker (1989). Fetal and neonate ages were established using measurements of available skeletal elements (Fazekas and Kosa 1978).

Adult ages were estimated using transition analysis. Transition analysis, developed by Jesper Boldsen and George Milner (Boldsen *et al.* 2002), is an adult aging method that addresses the problem of age mimicry of the reference sample (Bocquet-Appel and Masset 1982; Konigsberg and Frankenberg 1992, 1994, 1997). Transition analysis relies on age-related information collected from five different cranial sutures, the pubic symphysis, and the iliac portion of the sacroiliac joint using the scoring system established by Boldsen *et al.* (2002). These data are then entered into a computer program (ADBOU Age Estimation software), which appropriately weights this osteological information and then calculates a maximum likelihood estimate of age using Bayes' Theorem and either a uniform prior distribution of f(a) or an external f(a).

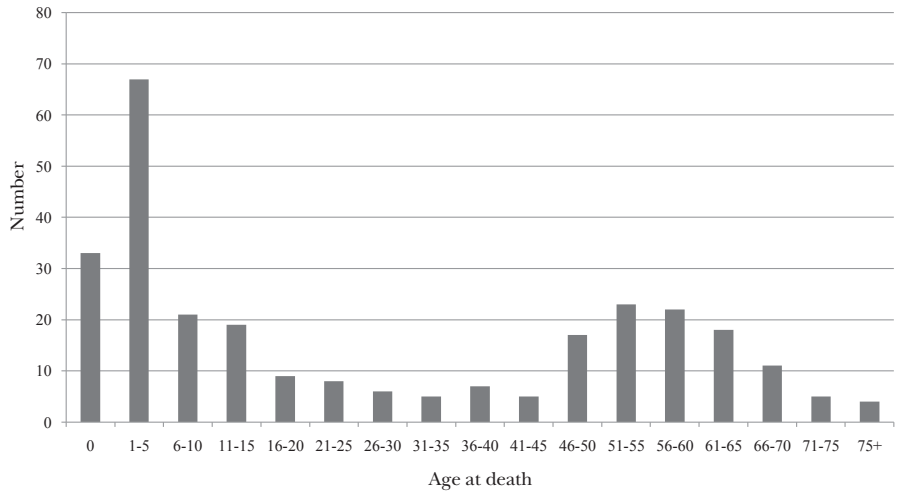

Figure 2. The age-at-death distribution for Postclassic Cholula (Bullock Kreger 2010).

Separate age-at-death distributions were generated for the Cholulteca II and Cholulteca III skeletons to determine if significant changes in mortality occurred over time. As no statistically significant differences exist between the two distributions, they were combined into a single age-at-death distribution for Cholula (figure 2). From an archaeological

perspective, combining Cholulteca II and III is, perhaps, the best course of action as questions have been raised about the validity of the ceramic chronology used to differentiate these two phases (McCafferty 1996). In addition, a sufficient sample size is an important consideration with the Usher model, and combining the two distributions increased the number of skeletons that could be included in the analysis. While, ideally, we would use skeletons drawn from much briefer time spans to ensure that the relationship between pathological lesions and selective mortality does not change over time, parameter values are much more difficult to capture with smaller sample sizes.

Pathological lesions in the Cholula osteological assemblage

A number of pathological lesions were recorded in the Cholula collection including porotic hyperostosis and cribra orbitalia, enamel hypoplasias, and proliferative lesions. These pathological lesions were chosen, in part, because they are among the most commonly recorded lesions in skeletal collections, and they occur with sufficient frequency in past populations to ensure enough observations of the condition to determine how their presence affects mortality. For all lesions considered, no distinction was made with regard to sex. Rather, adult males and females were combined into one sample. This was done because these pathologies are present in children as well as adults, yet the sex of children cannot be reliably determined from skeletal remains. Eliminating children and dividing males and females would significantly reduce the sample size. However, it should be remembered that pathological lesions may differentially affect the sexes, a possibility which should be addressed if sample sizes permit. Furthermore, no distinction was made between healed and unhealed lesions, although survival of the stress episode, as indicated by healing of the lesion, is likely to tell us something about selective mortality.

Porotic hyperostosis and cribra orbitalia

Porotic hyperostosis is characterized by porous lesions on the frontal, parietal, and occipital bones of the skull. The underlying condition that produces the lesions frequently produces similar porosities on the superior parts

of the orbits, a condition referred to as cribra orbitalia. Stuart-Macadam (1987) conducted a radiograph study of living populations, in which she linked porotic hyperostosis and cribra orbitalia to anemia. In response to the anemia, the body attempts to produce more red blood cells in the cranial diploë. The diploë expands and puts pressure on the outer table of the skull, causing it to thin and resulting in the porous appearance (Wright and Chew 1999: 925). As red marrow is not present in the cranial bones of adults, porotic lesions as an active response to anemia are limited to juveniles, although healed lesions may be present on adult skeletons (Stuart-Macadam 1985). Evidence of healing or remodeling of the bone indicates that the individual survived the stress episode.

In New World skeletons, iron-deficiency anemia, resulting from malnutrition, gastrointestinal infections, and parasites, has traditionally been assumed to be the cause of porotic hyperostosis and cribra orbitalia (El-Najjar *et al.* 1976; Stuart-Macadam 1987; Mensforth *et al.* 1978; Holland and O'Brien 1997; Palkovich 1987: 528-529). A more recent study suggests that while genetic anemia may be responsible for porotic hyperostosis, iron-deficiency anemia is an unlikely culprit (Walker *et al.* 2009). Instead, this investigation posits that megaloblastic anemia, caused by vitamin B_{12} deficiencies, is responsible for most cases of porotic hyperostosis observed in skeletal material. As B_{12} is primarily found in foods of animal origin, a diet with limited meat consumption, particularly if combined with intestinal parasites, could result in such deficiencies. Furthermore, the authors of this study argue that cribra orbitalia can reflect either B_{12} deficiencies or scurvy (figures 3 and 4).

Figure 3. Cribra orbitalia in an individual from Cholula. Photograph taken with permission from the Dirección de Antropología Física of the INAH.

Figure 4. Porotic hyperostosis in an individual from Cholula. Photograph taken with permission from the Dirección de Antropología Física of the INAH.

For the purposes of analyzing lesions of porotic hyperostosis, an attempt at diagnosing the underlying condition was not made because of the tentativeness of such diagnoses and the fact that nutritional deficiencies do not typically occur in isolation. Rather, if an individual is deficient in one nutrient, they are likely to be deficient in others as well, so it is possible for B_{12} deficiencies, scurvy, and iron-deficiency anemia to all be present. Therefore, all cranial lesions involving significant porosity were included in the following analysis.

Stuart-Macadam (1988) has suggested that porotic hyperostosis may, in fact, indicate a healthy response to nutritional deficiencies. In other words, it is possible that sicker individuals die before their bodies are able to mount a defense to the nutritional inadequacy. However, in her analysis of the Tirup skeletal sample, Usher (2000) found that individuals with cribra orbitalia in this medieval Danish village were almost five times more

likely to die than those without the lesions. Wood *et al.* (1992) and De Witte (2006) report similar findings regarding the relationship between porotic hyperostosis and mortality. These data, therefore, support the idea that this condition reflects poor health, but this connection must also be established in the Cholula skeletal sample.

The state of preservation of the orbits, frontal squama, parietal, and occipital bones in the Cholula collection was assessed following Buikstra and Ubelaker (1994). Lesions were recorded as present or absent, and, if present, as active or inactive. In addition, the severity of lesions was scored as described by Usher *et al.* (2000: 143). As cribra orbitalia is a bilateral condition, good preservation (75-100 % complete) of at least one of the orbits was sufficient for inclusion in the analysis. There were no cases in which both orbits were well preserved that cribra orbitalia was recorded on one side, but not the other. Healed and unhealed lesions were combined. The Usher model indicated no increase in mortality from cribra orbitalia ($p > .05$), however, the limited number of cases observed likely resulted in the maximum likelihood program being unable to capture the parameter values. The high standard error would also suggest that the small percentage of individuals with cribra orbitalia caused statistically insignificant results.

The effects of porotic hyperostosis on the risk of death were assessed for the parietal and occipital bones separately, and only bones with good preservation (75-100 % complete) were included. The frontal bone was not included in the statistical analysis as so few individuals (3) had frontal lesions. As porotic hyperostosis is also a bilateral condition, either the left or right parietal was included depending upon which was better preserved. There were no cases in which both parietals were relatively complete that porotic hyperostosis was observed on one side and not the other. The decision to treat the bones separately was simply a means to increase the number of observations that could be included in the analysis and was not motivated by any theoretical assumptions that the presence of porotic hyperostosis on one cranial bone might affect mortality differently than its presence on another bone. However, it is interesting to note that porotic hyperostosis is much less frequently observed on the frontal squama in the Cholula population than on the occipital or the parietal, and the individuals with frontal involvement tend to have more severe lesions on other parts of the cranium as well. This perhaps indicates that frontal involvement occurs only after the nutritional deficiency has been

present for some time, which could, indeed, have implications for the relationship between the location of the lesion and what the pathology indicates about mortality. Unfortunately, so few individuals were observed with frontal lesions that statistical analysis of this bone was not possible, so this hypothesis could not be tested. Alternatively, it may be the case that frontal lesions are caused by a different nutritional deficiency than lesions on the parietal and occipital, perhaps scurvy. This is, in fact, suspected for at least one of the individuals with frontal lesions, as sphenoidal and zygomatic involvement is also present (Ortner 1999, 2003).

The presence of porotic hyperostosis does appear to increase the risk of death in the Cholula population. The pathology is much more common in younger individuals than in older individuals. While the results of the likelihood analysis appear to concur that porotic hyperostosis increases the risk of death tenfold or more, the results were not statistically significant for either the parietal or the occipital ($p > .05$), possibly due to fairly limited number of observations of the pathology.

Enamel hypoplasias

Enamel hypoplasias are transverse grooves that appear on the lingual or buccal surfaces of teeth during their development, when nutritional deficiencies or disease interrupts enamel formation for an extended period of time (Roberts and Manchester 1997: 58-59; Skinner and Goodman 1992; Goodman *et al.* 1984). As these lesions form during the process of tooth development, they are indicative of childhood illnesses or malnutrition. As with porotic hyperostosis and cribra orbitalia, enamel hypoplasias may be observed in adults, but they do not occur after the process of crown formation has ended. Furthermore, enamel hypoplasias always represent survival of a stress episode, as the grooves result from enamel formation being interrupted and then beginning anew (Goodman *et al.* 1984). Unlike most other pathological lesions, the age at which enamel hypoplasias form can be determined. As teeth develop at fairly set rates across populations, the location of the enamel hypoplasia on the tooth crown can be used to estimate the age of the individual at the time that enamel formation was interrupted (Goodman and Rose 1990) (figure 5).

Four permanent teeth were included in the present study: the maxillary first incisor, the mandibular canine, and the first and second

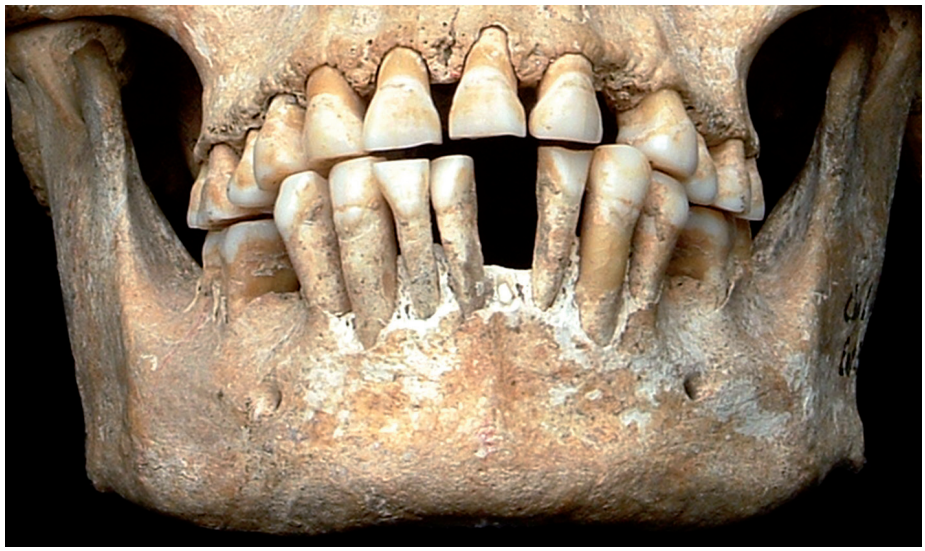

Figure 5. Enamel hypoplasias in the mandibular canines of an individual from Cholula. Photograph taken with permission from the Dirección de Antropología Física of the INAH.

mandibular molars. In each instance, the left tooth was scored, unless it was not present, in which case the right tooth was used. Deciduous teeth were not included because few had enamel hypoplasias. This finding is fairly typical since much of the deciduous dentition forms prior to birth when the fetus is buffered against stress by the mother (Goodman *et al* 1984: 26). Enamel hypoplasias were identified based upon a macroscopic examination of the tooth. Each tooth was scored as absent, present with no enamel hypoplasias, or present with at least one enamel hypoplasias (Usher 2000: 69-70). For each existing enamel hypoplasias, the location of the defect was also measured in order to determine the age at which it occurred (Goodman and Rose 1990). These measurements will be used in future studies of the Cholula population, as will be discussed later. For the present analysis, individuals were identified as "ill" if at least one enamel hypoplasia was present on the particular tooth being analyzed.

Tooth wear and tooth loss are concerns in the Cholula population, particularly with older individuals. Requiring that 100 % of the tooth be present for inclusion in the analysis resulted in small sample sizes con-

sisting exclusively of juveniles, which would have biased the results. As enamel hypoplasias do not typically occur in cuspal enamel (Goodman and Armelagos 1985), a decision was made to include in the current study those teeth in which at least two-thirds of the crown was present in order to maximize the sample size. Tooth wear and tooth loss resulted in fewer observations of older individuals, even allowing for the loss of up to one-third of the tooth. The extent of tooth loss and tooth wear does not become significant until around age 50, however, so the effects of hypoplasias on the risk of death should still be discernable. Analyses were also run requiring that 75 % of the tooth be present and that only 50 % of the tooth be present, and results similar to those discussed below were obtained.

Previous studies examining the relationship between enamel hypoplasias and mortality have resulted in inconsistent findings. While Goodman and Armelagos (1988) and Goodman (1996) established that the existence of enamel hypoplasias on the maxillary central incisors, the mandibular canines, and the maxillary first molars was linked to an increase in mortality, Usher (2000) found that the presence of enamel hypoplasias on only the first molar was associated with an elevated risk of death. She, in fact, reports that hypoplasias on the incisors were linked to a *reduction* in mortality in the Tirup population. These conflicting results may indicate that populations differ in their response to stress because of genetic or environmental variability. Consequently, examining the relationship between the presence of particular pathological lesions and the risk of death in each population is a necessary step in understanding health in past populations.

The results of the likelihood analysis indicate that enamel hypoplasias on the incisors, and the mandibular first and second molars are associated with a statistically-significant increase in the risk of death ($p < .05$). The k_2 constant for the mandibular canine indicates that there was no statistically-significant change in the risk of death associated with the presence of enamel hypoplasias ($p > .05$). As the sample size for the canine was similar to that of the others, it does not appear that an insufficient sample size or a limited number of observations of the pathological lesion is to blame for the lack of an effect on mortality. Indeed, the pathology occurs at a fairly consistent frequency in all age groups, supporting the results of the likelihood analysis that indicate that presence of enamel hypoplasias on the canine has no effect on mortality. In fact, very few individuals in the population did *not* have at least one enamel hypoplasia on the canine.

Interestingly, Usher (2000) also found that enamel hypoplasias on the canine had no effect on the risk of death in the Tirup population.

Proliferative lesions

Skeletal indicators of nonspecific infections were also assessed. As the skeleton can only respond to stress in a limited number of ways, namely through the proliferation or resorption of bone, diagnosing particular infectious diseases from skeletal remains is often impossible. Periostitis and osteomyelitis are general terms that refer to proliferative changes in bone caused by systemic or localized infections. Periostitis occurs when a bacterial infection or traumatic injury causes an inflammatory response in the periosteum, the thin membrane covering the bone. The inflammation stimulates osteoblasts to lay down new bone, which is porous in appearance (Ortner and Putschar 1981). Localized infections may result in unilateral skeletal involvement, while systemic infections tend to cause bilateral proliferative lesions. Osteomyelitis is a more severe form of bone infection in which bacteria enter the medullary cavity, again as a result of either a systemic infection or direct injury to the bone (Ortner and Putschar 1981). Unlike the other pathological lesions considered here, proliferative lesions may occur at any time during life. Healing of the lesions indicates that the individual survived the disease episode. In the current investigation, no distinction was made between periostitis and osteomyelitis; instead, all proliferative lesions were grouped together (figure 6).

Macroscopic examination of the skeleton was used to assess the presence or absence of skeletal lesions associated with infections. The degree of preservation of all the bones of the skeleton was recorded following Buikstra and Ubelaker (1994). Proliferative lesions were scored for presence or absence according to Usher (2000: 143). For the purposes of the likelihood analysis, no distinction was made between active and healed lesions, nor was the severity of the lesion considered; however, these data were collected and will be considered in future studies. The relationship between proliferative lesions and risk of death was assessed for the ulna, the femur, the tibia, and the fibula. To be included in the analysis, the diaphysis of the bone had to be well-preserved (75-100 %). Other bones were not included in the analysis of proliferative lesions due to the relatively small number of observations of this pathology elsewhere on the skeleton.

Figure 6. Proliferative lesions in an individual from Cholula.
Photo taken with permission from the Dirección de Antropología Física of the INAH.

The Usher model indicates that proliferative lesions on the ulna, femur, and tibia are associated with an increased risk of death ($p < .05$). Similar results were reported by Usher (2000) in her analysis of proliferative lesions of the femur in the Tirup population. The estimates of the k_2 parameter for the fibula do not indicate a significant increase in the risk of death as a consequence of proliferative lesions on this bone ($p > .05$). The maximum likelihood results for the fibula appear to be reflecting a genuine phenomenon, as a significant number of observations were recorded for this bone. Proliferative lesions on the fibula apparently have little effect on mortality.

In general, it appears that the majority of pathological lesions tested in the Cholula population were associated with an increase in the risk of death. While the k_2 estimates for cribra orbitalia were not found to cause a statistically significant increase in mortality, small sample sizes and an insufficient number of observations likely resulted in invalid results, as supported by the large standard error associated with the parameter value. The likelihood results for the canine, when viewed in conjunction with the distribution of canine enamel hypoplasia by age, does suggest that enamel hypoplasia on this particular tooth do not affect the risk of death in the Cholula population. Similarly, proliferative lesions on the fibula do not appear to increase the risk of death.

At this time, we can only speculate as to why enamel hypoplasia on the canines and proliferative lesions on the fibula did not affect mortality

among residents of Postclassic Cholula. With respect to enamel hypoplasias on the canines, it could be the case that the population of Cholula was genetically susceptible to these lesions, meaning that they formed easily in response to only minor insults, but the population was sufficiently healthy that most individuals were able to survive these minor stresses. Alternatively, the age at which these insults typically occurred may have resulted in them having little to no effect on the risk of death (Ferrell Thomas 2003) –a hypothesis currently being tested. Proliferative lesions on the fibula may primarily (although not exclusively) be associated with a condition that carries with it a low risk of death, such as yaws, or proliferative lesions of this long bone may only be forming in select individuals whose immune systems are not able to clear the infection but are healthy enough to survive the chronic condition (table 1).

Table 1
Table showing the number of observations of each pathological lesion and the k_2 value (Bullock Kreger 2010).

Pathological lesions in the Cholula skeletal assemblage					
Pathology	*present*	*absent*	*unobserv*	k_2	*SE*
Cribra orbitalia	12	113	154	3.2901	3.6820
Porotic hyperostosis					
Parietal	17	164	98	9.9999	6.5231
Occipital	11	143	129	9.9999	10.0446
Enamel hypoplasias					
Incisor	58	50	172	9.9999	4.5807
Canine	94	15	170	8.8819	5.2601
1st molar	44	76	160	7.3197	3.3072
2nd molar	41	66	172	9.9999	4.9857
Proliferative lesions					
Ulna	22	129	128	2.1940	0.6883
Femur	30	124	126	4.5353	2.0501
Tibia	76	97	105	5.1573	2.0230
Fibula	48	109	121	1.5347	0.4176

It is important to note that the assumption that is made in the Usher model, by virtue of the way the k_1 and k_2 parameters are specified, is that all individuals, regardless of age, are at equal risk of acquiring and dying from a particular pathology. The results of the likelihood analyses discussed above, therefore, indicate the relationship between pathological lesions and mortality at the aggregate level. However, age-related variations in selective mortality are almost a certainty. For example, examining the distribution of proliferative lesions on the long bones, it is notable that relatively few infants were observed to have this pathology. Quite probably, most of these individuals, as they are among the frailest in their cohort, simply die of the infection before the lesions have a chance to form. The presence of proliferative lesions indicative of infection in an infant may, therefore, indicate a somewhat "healthier" (relatively speaking) individual, who was, at least, able to survive long enough for the lesions to develop. However, in other age groups the relationship between proliferative lesions and the risk of death may not be the same.

Some studies have, in fact, indicated age-related differences in the risk of death associated with pathological lesions. Goodman (1996) found enamel hypoplasia forming between ages one to two were associated with longer-term survival than enamel hypoplasia forming between ages two and four. Ferrell Thomas (2003) found that the presence of a particular type of enamel defect was associated with an increase in the risk of death, except when it occurred between the ages of two and four. Clearly, it is necessary for paleopathologists to study not only aggregate-level relationships between pathological lesions and mortality, but also the age-related pattern of risks associated with particular pathological conditions. Enamel hypoplasia afford just such an opportunity, as the age at which the defect formed can be estimated. In future studies of the Cholula assemblage, an examination of the age at formation of enamel hypoplasia and the associated risk of death will be completed to determine if the age at which the stress occurred had an effect on mortality in this population.

In this study, the Usher reduced model of health was used to look at the relationship between several pathological lesions and the risk of death for the Postclassic skeletal sample from Cholula. Enamel hypoplasia on the incisors and first and second mandibular molars were shown to be associated with increased mortality, as were proliferative lesions on the ulna, the femur, and the tibia. Enamel hypoplasia on the canine and pro-

liferative lesions on the fibula had no effect on mortality. The parameter estimates for other pathologies are more equivocal because of small sample sizes or small numbers of observations. More research must be done in order to model underlying frailty in populations and, thereby, resolve the osteological paradox. While we cannot yet draw any conclusions about the health of the Postclassic residents of Cholula, being able to specify the relationship between observed pathological lesions and the risk of death in this population brings us one step closer to that goal.

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References

Alter, G. and J. Riley
1989 Frailty, sickness, and death: Models of morbidity and mortality in historical populations, *Population Studies,* 43: 25-45.

Bocquet-Appel, J. and C. Masset
1982 Farewell to Paleodemography, *Journal of Human Evolution,* 11: 321-333.

Boel, L.
2001 *Adult bone mass and physiological stress: A paleoepidemiological study*, thesis, University of Aarhus, Aarhus.

Boldsen, J.
1991 Ageing and dental attrition in a Medieval Rural Danish population, *International Journal of Anthropology,* 6: 217-224.

1997 Estimating patterns of disease and mortality in a Medieval Danish Village, R. Paine (ed.), *Integrating Archaeological Demography: Multidisciplinary approaches to Prehistoric Population*, Center for Archaeological Investigations, Southern Illinois University, Carbondale: 229-241.

BOLDSEN, J., G. MILNER, L. KONIGSBERG AND J. WOOD
2002 Transition analysis: A new method for estimating age from skeletons, R. Hoppa and J. Vaupel (eds.), *Paleodemography: Age distributions from skeletal samples*, Cambridge University Press, Cambridge: 73-106.

BUIKSTRA, J. AND D. UBELAKER
1994 *Standards for data collection from human skeletal remains*, Arkansas Archaeological Society, Fayetteville.

BULLOCK KREGER, M.
2010 *Urban population dynamics in a Preindustrial New World City: Morbidity, mortality, and immigration in Postclassic Cholula,* thesis, Pennsylvania State University, Philadelphia.

CAMARGO, L., L. MÁRQUEZ AND M. PRADO
1999 Paleodemografía del México prehispánico, R. Benítez and R. Ornelas (eds.), *Hacia la demografía del siglo XXI,* Sociedad Mexicana de Demografía, Mexico: 227-250.

CAMARGO, L. AND V. PARTIDA
1998 Algunos aspectos demográficos de cuatro poblaciones prehispánicas de México, L. Márquez and J. Gómez de León (eds.), *Perfiles demográficos de poblaciones antiguas de México***,** Instituto Nacional de Antropología e Historia, Mexico: 77-94.

COOK, D. AND J. BUIKSTRA
1979 Health and differential survival in Prehistoric Populations, *American Journal of Physical Anthropology,* 51: 649-64.

DE WITTE, S.
2006 *The Paleodemography of the Black Death 1347-1351*, thesis, Pennsylvania State University, Philadelphia.

DE WITTE, S. AND J. BEKVALAC
2010 Oral health and frailty in the Medieval English Cemetery of St. Mary Graces, *American Journal of Physical Anthropology,* 142: 341-354.

DE WITTE, S. AND J. WOOD
2008 Selectivity of Black Death mortality with respect to preexisting health, *Proceedings of the National Academy of Sciences,* 104: 1 436-1 441.

EL-NAJJAR, M., D. RYAN, C. TURNER AND B. LOZOFF
1976 The etiology of porotic hyperostosis among the prehistoric and historic Anasazi Indians of the Southwest United States, *American Journal of Physical Anthropology,* 44: 477-478.

FAZEKAS, I. AND F. KOSA
1978 *Forensic Fetal Osteology*, Akademiai Kiado, Budapest.

FERRELL THOMAS, R.
2003 *Enamel defects, well-being, and mortality in a Medieval Danish Village*, thesis, Pennsylvania State University, Philadelphia.

GAGE, T.
1988 Mathematical hazard models of mortality: An alternative to model life tables, *American Journal of Physical Anthropology,* 76: 429-441.
1989 Bio-mathematical approaches to the study of human variation and mortality, *Yearbook of Physical Anthropology,* 32: 185-214.
1990 Variation and classification of human age patterns of mortality: analysis using competing hazards models, *Human Biology,* 62: 589-614.
1994 Population variation in cause of death: level, gender, and period effects, *Demography,* 31: 271-296.

GAGE, T. AND B. DYKE
1986 Parameterizing abridged mortality tables: The Siler Three-Component Hazard Model, *Human Biology,* 58: 275-291.

GÓMEZ DE LEÓN, J.
1998 Análisis paleodemográfico de poblaciones antiguas de México, algunas estimaciones y comentarios metodológicos, L. Márquez and J. Gómez de León (eds.), *Perfiles demográficos de poblaciones antiguas de México*, Instituto Nacional de Antropología e Historia, Mexico: 106-130.

GOODMAN, A.
1996 Early life stresses and adult health: Insights from dental enamel development, C. Henry and S. Ulijaszek (eds.), *Long-term consequences of Early-Environment: Growth, development and the lifespan developmental perspective*, Cambridge University Press, Cambridge: 163-182.

GOODMAN, A., D. MARTIN, G. ARMELAGOS AND G. CLARK
1984 Indicators of stress from bone and teeth, M. Cohen and G. Armelagos (eds.), *Paleopathology at the origins of agriculture*, Academic Press, New York: 13-50.

GOODMAN, A. AND G. ARMELAGOS
1985 Factors affecting the distribution of enamel hypoplasias within the human permanent dentition, *American Journal of Physical Anthropology,* 68: 479-493.
1988 Childhood stress and decreased longevity in a Prehistoric Population, *American Anthropologist,* 90: 936-944.

GOODMAN, A. AND J. ROSE
1990 Assessment of systematic physiological perturbations from dental enamel hypoplasias and associated histological structures, *Yearbook of Physical Anthropology,* 33: 59-110.

HOLLAND, T. AND M. O'BRIEN
1997 Parasites, porotic hyperostosis, and the implications of changing perspectives, *American Antiquity*, 62: 183-193.

HOLMAN, DARRYL
2002 Mle: A programming language for building likelihood models, Version 2.1, Seattle.

KONIGSBERG, L. AND S. FRAKENBERG
1992 Estimation of age structure in Anthropological Demography, *American Journal of Physical Anthropology*, 89: 235-256.
1994 Paleodemography: "Not quite dead", *Evolutionary Anthropology,* 3: 92-105.
1997 Regress what on what? Paleodemographic age estimation as a calibration problem, R. Paine (ed.), *Integrating Archaeological Demography: Multidisciplinary approaches to Prehistoric Population*, Center for Archaeological Investigations, Southern Illinois University, Carbondale: 64-88.

LAGUNAS, Z.
1973 La trepanación suprainiana en cráneos de Cholula, Pue., *Comunicaciones,* 8: 47-48.
1994 Las prácticas funerarias en Cholula prehispánica, *Mirada antropológica,* 1: 82-94.

López, S.
1972 La cremación y los entierros en vasija de la zona arqueológica de Cholula, Puebla, *Religión en Mesoamérica: XII Mesa Redonda de la Sociedad Mexicana de Antropología*, Sociedad Mexicana de Antropología, Mexico: 375-379.
1973 Enterramientos humanos prehispánicos de Cholula, Puebla, *Comunicaciones,* 8: 43-45.

López, S. and M. Salas
1989 Los antiguos habitantes de Cholula: algunos elementos del perfil físico, *Notas Mesoamericanas,* 11: 5-18.

López, S., Z. Lagunas and C. Serrano
1970 Sección de Antropología Física, I. Marquina (ed.), *Proyecto Cholula*, Instituto Nacional de Antropología e Historia, Mexico: 143-152.
1976 *Enterramientos humanos de la zona arqueológica de Cholula, Puebla*, Instituto Nacional de Antropología e Historia, Mexico.
2002 *Costumbres funerarios y sacrificio humano en Cholula prehispánica*, Universidad Nacional Autónoma de México, Mexico.

Márquez, L.
1996 Paleoepidemiología en las poblaciones prehispánicas mesoamericanas, *Arqueologia Mexicana*, IV (22): 4-11.

Márquez, L. and P. Hernández
2006 Demografia y salud en Mesoamérica, L. Márquez and P. Hernández (eds.), *Salud y sociedad en el México prehispánico y colonial*, Consejo Nacional para la Cultura y las Artes, Instituto Nacional de Antropología e Historia, Mexico: 59-73.
2007 La transición demográfica en la prehistoria de Mesoamérica, P. Espinoza, C. Serrano Sánchez and F. Pedraza (eds.), *VII Congreso de la Asociación Latinoamericana de Antropología Biológica*, Instituto Nacional de Antropología e Historia, Mexico: 37-48.

Márquez, L., R. McCaa, R. Storey and A. del Ángel
2002 Health and nutrition in Prehispanic Mesoamerica, R. Steckel and J. Rose (eds.), *The backbone of history health and nutrition in the Western Hemisphere,* Cambridge University Press, Cambridge: 307-340.

McCafferty, G.
1996 The ceramics and chronology of Cholula, Mexico, *Ancient Mesoamerica,* 7: 299-324.

Mensforth, R., C. Lovejoy, J. Lallo and G. Armelagos
1978 The role of constitutional factors, diet and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children, *Medical Anthropology,* 2: 1-58.

Milner, G., J. Wood and J. Boldsen
2000 Paleodemography, M. Katzenburg and S. Saunders (eds.), *Biological Anthropology of the Human Skeleton,* Wiley-Liss, New York: 467-497.

Ortner, D.
1991 Theoretical and methodological issues in paleopathology, D. Ortner and A. Aufderhheide (eds.), *Human Paleopathology: Current syntheses and future options,* Smithsonian Institution Press, Washington: 5-11.
1999 Probable evidence of scurvy in subadults from archaeological sites in Peru, *American Journal of Physical Anthropology,* 108: 321-331.
2003 *Identification of pathological conditions in Human Skeletal remains,* Academic Press, San Diego.

Ortner, D. and W. Putschar
1981 *Identification of pathological conditions in Human Skeletal remains,* Smithsonian Institution Press, Washington.

Palkovich, A.
1987 Endemic disease patterns in paleopathology: Porotic hyperostosis, *American Journal of Physical Anthropology,* 74: 527-537.

Roberts, C. and K. Manchester
1997 *The archaeology of disease,* Cornell University Press, New York.

Skinner, M. and A. Goodman
1992 Anthropological uses of developmental defects of enamel, S. Saunders and M. Katzenburg (eds.), *Skeletal biology of past peoples: Research methods,* Wiley-Liss, New York: 153-174.

SMITH, B.
1991 Standards of human tooth formation and dental age assessment, M. Kelley and C. Larsen (eds.), *Advances in dental anthropology*, Wiley-Liss, New York: 143-168.

STECKEL, R. AND J. ROSE (EDS.)
2002 *The backbone of history*, Cambridge University Press, Cambridge.

STUART-MACADAM, P.
1985 Porotic hyperostosis: Representative of a childhood condition, *American Journal of Physical Anthropology,* 66: 391-398.
1987 New evidence to support the anemia theory, *American Journal of Physical Anthropology,* 74: 511-520.
1988 Porotic hyperostosis: Changing interpretations, D. Ortner and A. Aufderhheide (eds.), *Human paleopathology: Current syntheses and future options*, Smithsonian Institution Press, Wahington: 36-39.

UBELAKER, D.
1989 *Human skeletal remains: Excavation, analysis, and interpretation*, Taraxacum, Washington.

USHER, B.
2000 A *multistate model of health and mortality for Paleodemography: Tirup Cemetery*, thesis, Pennsylvania State University, Philadelphia.

VAUPEL, J.
1990 Relative's risks: Frailty models of life history data, *Theoretical Population Biology,* 37: 220-234.

VAUPEL, J. AND A. YASHIN
1985 Heterogeneity's ruses: Some surprising effects of selection on population dynamics, *The American Statistician,* 39: 176-185.

VAUPEL, J., T. JOHNSON AND G. LITHGOW
1979 The impact of heterogeneity in individual frailty on the dynamics of mortality, *Demography,* 16: 439-454.

WALKER, P., R. BATHURST, R. RICHMAN, T. GJERDRUM AND V. ANDRUSHKO
2009 The causes of porotic hyperostosis and cribra orbitalia: A reappraisal of the iron-deficiency-anemia hypothesis, *American Journal of Physical Anthropology,* 139: 109-125.

WOOD, J., G. MILNER, H. HARPENDING AND K. WEISS
1992 The osteological paradox: Problems of inferring health from skeletal samples, *Current Anthropolgy,* 33: 343-370.

WRIGHT, L. AND L. CHEW
1999 Porotic hyperostosis and paleoepidemiology: A forensic perspective on anemia among the ancient Maya, *American Anthropologist,* 100: 924-939.