The Effects of Urbanism on the Health and Demography of the Postclassic Population of Cholula, Puebla

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Site: Cholula

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Introduction

The debate regarding the effects of urbanism on the health of preindustrial populations has a long history, which began in the 17th century when an individual named Graunt conducted an early demographic study of London using parish records of baptisms and burials. Based on his findings, he concluded that the unhealthy conditions of the urban environment resulted in a mortality rate so high that deaths regularly exceeded births in the city, leading to a natural decrease in population, and that immigration must have been significant to account for the population growth being experienced in London at the time (cited in Galley 1998). Since Graunt, a number of other demographers and anthropologists have repeated the assertion that the high population densities and unsanitary living conditions present in preindustrial cities would have caused elevated morbidity and mortality rates in urban areas (see, for example, Wrigley 1967, 1969; Cohen and Armelagos, eds. 1984; Cohen 1989). However, other researchers have argued that the high mortality rates experienced in some preindustrial Old World cities were largely confined to particular demographic groups, namely young juveniles and immigrants, that were most susceptible to the ravaging effects of epidemic diseases (Sharlin 1978; Landers 1992; Galley 1998). Preindustrial urban health in New World populations is an issue that is central to resolving this debate because it provides a glimpse of urban mortality in the absence of epidemic diseases. Unfortunately, it is also an issue that is difficult to accurately assess given the lack of written demographic records. While several osteological and paleopathological investigations (Storey 1985; 1992; Marquez et al. 2002; Cohen 1989) have made valuable attempts to understand how the process of urbanization could have affected the health of New World urban residents, they have met with overwhelming methodological challenges that make interpreting their findings difficult.

The Foundation for the Advancement of Mesoamerican Studies, Inc., (FAMSI) generously provided a grant to realize a demographic and paleopathological study of Postclassic burials from the prehispanic city of Cholula, Puebla, in central México, in order to address the issue of New World urban health using new methods that overcome the methodological difficulties faced by previous studies. As part of this research, a technique of determining adult age-at-death, which corrects many of the problems associated with traditional aging methods, was utilized. In addition, the relationship of particular pathologies to mortality in the population will be analyzed in order to address the issue of selective mortality, which is a confounding factor in paleopathological studies (Wood et al. 1992). The results presented here are still preliminary and analysis and research are ongoing. However, the data at this time suggest that mortality in New World urban centers may differ from urban health in preindustrial Old World cities.

Mortality in Preindustrial Old World Cities

The majority of studies focusing on urban health have been demographic investigations of preindustrial Old World cities, particularly London, that have relied exclusively on documents such as parish records of baptisms, marriages, and burials to reconstruct vital events (Wrigley 1967, 1969; Finlay 1981; de Vries 1984; Sharlin 1979; Landers 1992; Galley 1998). The demographer Wrigley (1967, 1969) has been one of the most prominent proponents of Graunt's early assertion that mortality in cities was greater than fertility. Like Graunt, he argues that for the population of cities to have increased, immigration must have been substantial. Wrigley attributes the high mortality in cities to a number of factors. First, the size of the population in Old World cities was sufficient to permit epidemic diseases like smallpox and measles to become established. Since epidemic diseases typically cause rapid death or, should the individual survive, grant lifetime immunity to subsequent infection by that particular pathogen, a sufficient number of new susceptibles must enter the population, either through birth or immigration, for the disease to become endemic. Cities enable these diseases to thrive because they supply the requisite population size.

Second, the large populations and crowded living conditions in cities facilitated the transmission of infectious diseases. Airborne or droplet infections like tuberculosis and other respiratory diseases are common causes of morbidity and mortality in preindustrial and developing urban areas because of the density of habitation. Diseases that rely on fecal-oral transmission are aided by the contaminated water supplies and unsanitary conditions found in many preindustrial cities. For example, in London, human wastes were frequently disposed of in a boarded-up cesspool beneath the houses of the lower classes (Landers 1992). Water contamination was similarly a problem in London, as some of the inhabitants drew their water directly from the Thames. Finlay (1981), in fact, states that parishes near the Thames had higher mortality rates than neighborhoods in other parts of the city.

Third, as inhabitants of preindustrial cities in Europe did not generally produce their own food, they were dependent on the market. In years of poor harvests, Wrigley argues that high grain prices, combined with declining real wages for occupational specialists who are unable to sell their goods, resulted in malnutrition for urban residents. Furthermore, those who lived in the city would have been unable to supplement their diet with scavenged wild resources. Malnutrition would have made city dwellers even more susceptible to succumbing to infectious disease.

Finally, trade and displacements caused by war or famine facilitated the spread of diseases into cities, as urban areas were economic centers. The Black Plague, for example, was introduced into Europe in the 14th century as a result of long-distance trade.

Thus, Wrigley presents preindustrial cities as having such high mortality rates that they are only able to sustain their numbers through large-scale rural-to-urban migration. However, this idea that cities are demographic "sinks" for the surrounding rural

population (Wrigley 1969) has been challenged by subsequent research. As other historical demographers have shown, the demography of preindustrial Old World cities is more complicated than the "natural decrease" model presented by Wrigley.

In 1978, Sharlin argued that the "natural decrease" observed in cities was largely due to the high mortality rates of immigrants. Many rural migrants were poor, and, therefore, may have been forced into overcrowded, unsanitary conditions once they reached the city. Furthermore, their low socioeconomic status may have led to many of them being malnourished and, hence, more susceptible to many infectious diseases. Most importantly, these individuals may not have had previous exposure to the epidemic infections that were endemic in cities. In addition to their high mortality rates, Sharlin argues that migrants contributed little to fertility in preindustrial cities and were unable to replace themselves. He bases his conclusions on the assumption that migrants were only in the city temporarily and did not marry or reproduce there. As migrants were largely apprentices and servants, cultural and sometimes legal restrictions dissuaded them from marrying while in the city. To support his argument, he compares citizens (which he equates to permanent residents) and non-citizens (which he equates to migrants) from the city of Frankfort-am-Main. His data indicate that while baptisms surpass burials among citizens, the opposite is true for non-citizens. Therefore, migrants were the primary reason for the apparent natural decrease in cities.

Another study of London from the 16th to the 19th century conducted by Finlay (1981), compared the demography of four urban parishes, two of which were poor parishes and two of which were of a higher socioeconomic status. Finlay's results were based on partial family reconstitutions, and, therefore, he was only able to address infant and childhood mortality and marital fertility rates. He found that infant and childhood mortality was high compared to English villages and that poorer parishes had higher mortality rates than wealthier parishes. Interestingly, infant mortality within the parishes was lower than expected when compared to childhood mortality. Finlay speculates that this may be due to the protective effects of breastfeeding for infants, particularly in regards to the partial immunity provided to diseases like smallpox by maternal antibodies. Finlay reconstructed marital fertility rates from birth intervals and found that marital fertility was high in the city. However, he suggests, based on a study of the comparative ages of marriage for immigrants and permanent residents, that immigrant women married later and had fewer children than permanent residents. As a result, total fertility in London was not high enough to surpass the high mortality rates, resulting in natural decrease.

Landers (1992), using both aggregative data and partial family reconstitution, reconstructed mortality rates in London in the 18th century. His data indicated that infant and childhood mortality rates among the London Quakers in the city were particularly high. Furthermore, the large number of burials among late adolescents and young adults supports the idea that migrants to the city had particularly high mortality rates. As cause of death is provided in some of the records used by Landers, he is also able to demonstrate that smallpox and unspecified fevers were major causes of death for both children and young adults. Since most individuals born in the city would have contracted

smallpox during childhood, the high mortality rates from this disease among young adults suggest that there was a great deal of immigration into London at this time, and that many of the migrants had not previously been exposed.

Galley (1998) relied on partial family reconstitution to determine fertility and mortality rates for York during the 17th and 18th centuries. Examining the total number of burials and baptisms from approximately the 16th century to the 19th century, he points out that the burials exceed baptisms only from the mid-17th century to the mid-18th century. Prior to that time, the Black Death caused periodic mortality crises in which large percentages of the population died, but in non-crisis years, baptisms exceeded burials. From the late 18th century on, the city again experienced natural increase. Therefore, natural decrease is not necessarily an inherent characteristic of preindustrial cities: They could and did experience a surplus of births.

Galley then examines the demography of York to explain why the decrease occurred from the mid-17th century to the mid-18th century. Infant and childhood mortality rates in the city were higher than those in smaller rural villages, with approximately 50% of all children dying before they reached adulthood. However, infant and childhood mortality rates were lower than those of London, suggesting that mortality may, in fact, have a density dependent component. Like Landers, he concludes that smallpox was a major cause of death for both children and young adults, again suggesting a substantial migrant population. Mortality in York increased during the 17th and 18th centuries, but not enough to account for the apparent surplus of burials over baptisms. This increase in mortality must have been accompanied by a decline in fertility for natural decrease to have occurred. Examining marital fertility rates, he found that while marital fertility was high, it declined during the 17th century perhaps because of reduced economic opportunities resulting in later marriages.

Based on these studies, the natural decrease that Wrigley and others postulated for preindustrial cities does not seem to have been inherent feature of urban life, even in the Old World. As indicated by Galley's data, natural increase did occur in some cities. Furthermore, a number of factors can confound the analysis of urban demography including socioeconomic status and microenvironmental variability within the city itself. While mortality rates of preindustrial Old World cities were generally higher than those of rural areas, elevated mortality was largely confined to infants and children and to migrants who had not previously been exposed to the epidemic diseases present in the city.

These findings seriously call into question whether New World cities would have experienced a similar demographic regime for a number of reasons. First, rural adults migrating to urban areas would not likely have been confronted with diseases to which they had never been exposed. While it could be argued that the endemic gastrointestinal and respiratory diseases that would have been present in New World cities may have still increased the mortality of immunologically vulnerable individuals, it is unclear if they would have had the same effect as epidemic diseases, particularly among young adults.

Second, the particular culture history of European cities clearly affected their demography and contributed to the occasions of natural decrease, as these cultural factors strongly influenced not only the demographic characteristics of migrants, but also when and if they married, thereby affecting fertility levels in cities. Migrants to New World cities may have immigrated for different reasons, at least in part, and may not have faced the same obstacles to marrying. As a result, fertility levels among migrants may not have differed that much from that of permanent residents. This information is simply not known.

The Anthropological Debate over the Relationship between Health and Cultural Evolution

In recent years, the effects of urbanism on preindustrial population dynamics have been part of a larger debate in anthropology regarding the relationship between cultural evolution and the health of past populations. Citing arguments similar to those used by Wrigley, a number of anthropologists have argued that the elevated population densities that accompany the transition to agriculture and the urban revolution result in the presence of a greater number of infectious diseases and parasites in a population because of the increased number of potential hosts. In addition, sedentism creates unsanitary living conditions that are conducive to the transmission of many of these pathogens. Simultaneous reductions in the adequacy of the diet induce malnutrition, which only serves to heighten susceptibility to these infectious diseases. As a result, they argue, health declines and mortality from infectious diseases rises as societies become more socially, politically, and economically complex (Cohen and Armelagos 1984; Cohen 1989; Swedlund and Armelagos 1990). A number of paleopathological studies involving farming populations have been presented as empirical evidence that the transition to agriculture was indeed accompanied by increases in malnutrition and disease for prehistoric people (Cohen and Armelagos 1984; Cohen 1989; Steckel and Rose 2002). Some studies have also focused on how the process of urbanization affected past populations (Lewis 1995; Storey 1992; Marquez *et al.* 2002; Brothwell 1994; Cohen 1989).

However, a number of questions have been raised about the theoretical soundness of the position that cultural evolution is inevitably tied to declining health and about the validity of the conclusions drawn from these osteological analyses. The majority of these paleopathological studies have assumed a straightforward relationship between the frequency of skeletal lesions and the health of populations, so high frequencies of pathological lesions are considered to be indicative of poor health. Unfortunately, skeletal lesions may not be so readily understandable. Wood *et al.* (1992) caution that issues such as hidden heterogeneity in risks, selective mortality, and population non-stationarity can hinder the interpretation of morbidity and mortality data from skeletal remains. As a result, an increase in the frequency of pathological lesions is not *necessarily* indicative of declining health. Furthermore, a theoretical model presented by Wood (1998) that investigates how well-being varies with economic change does not support the idea that the health of populations is negatively associated with increasing

social complexity. Questions have also been raised about whether an increase in the number of different infectious diseases present in a population inevitably translates into higher mortality from infectious diseases, particularly if the population is not suffering malnutrition (Wood 1998: 118-119). An osteological analysis of the Cholula population that addresses some of the problems associated with previous paleopathological and paleodemographic analyses can contribute to the resolution of this debate.

Figure 1. Map of México showing study area.

Cholula

Located in the state of Puebla in Central México, the archaeological site of Cholula had a long history of urban occupation. During the early Classic Period, Cholula developed rapidly into a politically important ceremonial center that may have exercised hegemony over sites in the southern Basin of México after the fall of Teotihuacán (Muller 1973; Sanders 1971). At the end of the Classic Period, some sort of catastrophe seems to have befallen the site, resulting in a depopulation of the area and a decline in the political importance of the city (Muller 1973: 20-21). In the Early Postclassic, changes in the ceramic tradition and the style of burials at the site indicate that another ethnic group may have populated the site at this time, possibly following a conquest or political takeover at the end of the Classic (Muller 1973: 21). The Middle Postclassic, or Cholulteca II (A.D. 900-1325) ushered in a new era of expansion for Cholula, and by the Late Postclassic (Cholulteca III, A.D. 1325-1500) the city reached its maximum size with an urban population estimated to have been between 30,000 and 50,000 individuals in an area of 8 km^2 (Muller 1973; Sanders 1971). As a religious and mercantile center, much of the urban population would have been comprised of occupational specialists who would have depended on the market for food. In addition, the population density of the site would have resulted in typical urban problems regarding waste disposal and

contamination. Thus, Cholula is an appropriate object of study to assess urban demographics of New World cities.

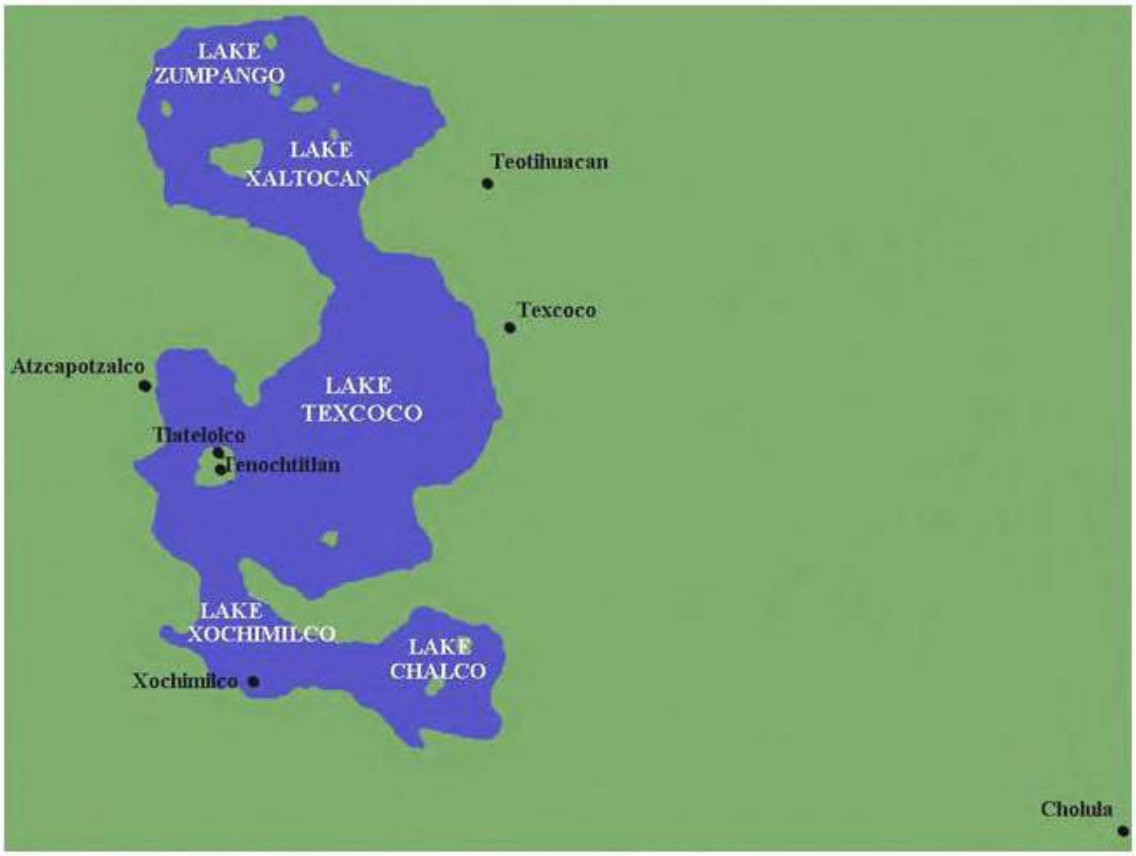

Figure 2. Map showing Cholula and sites mentioned in the text.

The skeletal sample from Cholula used in this analysis consists of 84 Cholulteca II skeletons and 257 Cholulteca III skeletons excavated during the 1967-1970 field seasons of the Cholula Project. 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 (Lopez *et al.* 1976). A number of ceremonial burials were also encountered during these excavations, but this material is not included in the data presented below, as the vast majority were clearly sacrificial victims. These skeletons are housed at the National Museum of Anthropology and History in México City, and special thanks goes to the Department of Physical Anthropology at the museum and Maestro José Jimenez, who facilitated this study.

Mortality in the Postclassic Cholula Population

An age-at-death distribution will be constructed for the Cholulteca II and Cholulteca III skeletons, both to assess mortality in the urban population and to aid in the interpretation of skeletal lesions. In living populations, mortality rates are often used to estimate the overall health of a population, since high instances of malnutrition and disease can increase the risk of death (Roberts and Manchester 1997: 27-28). However, calculating age-specific mortality rates for osteological samples has proven to be considerably problematic. The first issue with which paleodemographers must contend is inaccuracies in age determination. Although juveniles can be aged within a limited range of error, adult age estimation is less precise, particularly for older individuals (Milner *et al.* 2000: 476-477). When ages are assigned to adult skeletons, they are frequently presented as a range of possible ages. While this practice acknowledges the inaccuracies inherent in aging adult skeletal remains, it also assumes that all age intervals can be estimated with the same degree of error, which is not the case (Milner et al. 2000: 476-477). In addition, concerns have been raised that the age-at-death distribution of a skeletal sample may mimic that of the reference collection from which the aging techniques were derived (Bocquet-Appel and Masset 1982).

A second problem with paleodemographic analyses is that factors such as particular mortuary treatments, differential preservation, and biased recovery may bias osteological collections (Milner *et al.* 2000: 473-475). Juveniles, in particular, may be under-enumerated in skeletal collections because their smaller, less-dense bones often do not preserve as well as those of adults. Their remains may also be missed by excavators either due to inexperience in identifying the bones of children or due to special burial treatments that isolate them from the remains of adults. The underrepresentation of juveniles in osteological collections is particularly troublesome given the significance of infant and childhood mortality in the age-at-death profiles of preindustrial populations.

Third, the population growth rate can have a profound influence on the age-at-death distribution for a population, but the population growth rate for an archaeological skeletal sample is seldom known (Sattenspiel and Harpending 1983). In constructing model life tables for skeletal samples, paleodemographers have, in the past, assumed that the population had a zero growth rate, unchanging age-specific fertility and mortality rates, closure to migration, and an equilibrium age distribution (Milner *et al.* 2000: 479-480). This is a particularly problematic assumption because in nonstationary populations (i.e. populations with a nonzero growth rate), it has been demonstrated that small changes in fertility may have a drastic effect on the age-at-death distribution while large changes in mortality have little impact (Sattenspiel and Harpending 1983; Johansson and Horowitz 1986). This pattern is attributable to the fact that the age-at-death distribution, in part, reflects the number of individuals entering the population. Therefore, a change in the number of infants born affects the number of individuals that can potentially die at each subsequent age (Milner *et al.* 2000: 480-481). Being able to estimate the population growth rate is, therefore, an essential component of understanding the age-at-death distribution of skeletal sample.

Possibly as a result of the problems mentioned above, paleodemographic age-at-death distributions typically show an abundance of young adults and very few older individuals, a mortality pattern which is not found in the demography of either historic or modern populations. Nancy Howell (1982) has pointed out that if this mortality pattern were true, it would cause a variety of cultural difficulties including children being orphaned at a high frequency and a lack of cultural continuity due to a dearth of older adults. She strongly suggests that what is being observed in paleodemographic age-at-death distributions is, in fact, the result of preservation bias or most likely methodological errors in determining adult ages of skeletons.

Numerous attempts have been made to correct some of these problems or to find alternate methods to determine mortality for skeletal populations (Milner *et al.* 1989; Konigsberg and Frankenberg 1992; Gage and Dyke 1986; Gage 1988). A new method of determining adult age-at-death, referred to as transition analysis, was used to estimate the age of Cholula adults. Transition analysis as proposed by Boldsen *et al.* (2002) attempts to resolve some of the problems traditionally associated with adult age estimation by eliminating the biasing effects of the reference sample and by improving the estimation of ages for older adults. It also provides a statistically valid means of combining age-related data from the cranial sutures, the pubic symphysis, and the auricular surface. This information is used to generate maximum likelihood estimates of age.

Another proposed solution for studying mortality in past populations is the use of various parametric models to understand the age-at-death distribution of the population under study. One such mortality model is the Siler model, which consists of three components. The first component ($K_1e^{-2_1t}$) captures the high mortality of infancy and early childhood, which then declines rather rapidly. The second component (K_2) represents a constant "baseline," or age-independent, mortality, and the third component ($K_3e^{2_3t}$) is senescent risk, or the increasing risk of death with age (Gage and Dyke 1986; Gage 1988). In these equations, $K_{1,2,3}$ and $2_{1,3}$ are parameters or constants that are to be estimated from the skeletal sample itself. The Siler model does have some limitations in that it does not capture the "accident hump" associated with late adolescence and early adulthood, and it assumes that all individuals in a population have the same risk of death (Wood *et al.* 2002). However, in general it models human mortality relatively well.

Gage (1988; 1989; 1990; 1994; Gage and Dyke 1986) has done an extensive amount of research concerning the Siler model, and Wood *et al.* (2002) have also discussed the use of this model (as well as others) in the construction of age-at-death distributions. The parameters of the Siler model (as well as other mortality models) can be calculated using a maximum likelihood analysis program created by Darryl Holman (2003). Use of the Siler or other parametric mortality models allows paleodemographers to address some of the problems associated with skeletal samples including incomplete or inadequate data (Wood *et al.* 2002). For example, Usher (2000: 47-48) used the Siler model in order to approximate the number of missing juvenile skeletons in Tirup skeletal sample. In addition, parametric mortality models are essential to estimating the growth rate of a population and thereby correcting for nonstationarity (Wood *et al.* 2002). Wood

and Holman (cited in Usher 2000: 52), in particular, have developed a likelihood equation that uses the Siler model in order to estimate population growth from skeletal samples.

The age-at-death distributions of the Cholulteca II and Cholulteca III populations appear below. Interestingly, these age-at-death distributions do not show an overabundance of young adults typical of other paleodemographic samples, suggesting that transition analysis may, in fact, correct many of the problems described above. As would be expected, infant and child mortality in the populations appears to be high, as is the case in most preindustrial populations. Mortality among young adults is low, indicating that the elevated mortality rates that immigrants to Old World cities experienced may not have been present in New World urban centers. While further analysis must be completed, the age-at-death distributions of the Cholula population suggest that the demography of this city may have differed from that described for preindustrial European cities. Additional analyses planned for this data are discussed below.

Further analysis to be completed with the age-at-death distributions of the Cholula population:

1. The number of individuals to be included in the analysis of urban health at Cholula will be finalized. As mentioned, the above age-at-death distribution does not include information for burials from ceremonial areas, nor does it include a complex multiple burial for which the minimum number of individuals has yet to be determined. In addition, because many of the burials were close in space, some of the burials include skeletal elements of additional individuals. In cases in which it can be verified that these additional elements do not belong to individuals from nearby burials, they will be added to the analysis. Thus the number of skeletons and the age-at-death distribution of the population in future publications may differ slightly from that presented here.
2. The Siler model of mortality will be applied to the age-at-death distribution of both the Cholulteca II and the Cholulteca III population.
3. The population growth rate of the population will be estimated using the method discussed above.
4. Models will be constructed to estimate how the age-at-death distribution might be affected by different levels of migration.
5. The age-at-death distributions of the Cholulteca II and Cholulteca III populations will be compared with each other to determine if there were significant changes over time, and they will also be compared to those of Old World cities.

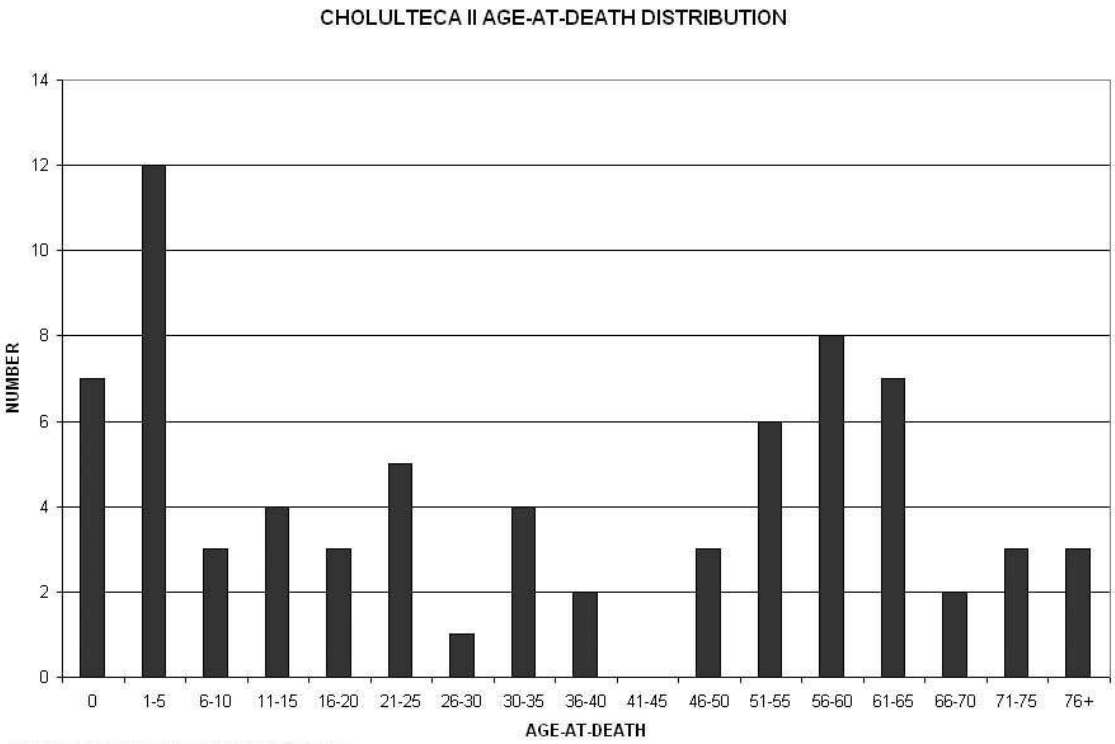

Table 1. Cholulteca II age-at-death distribution.

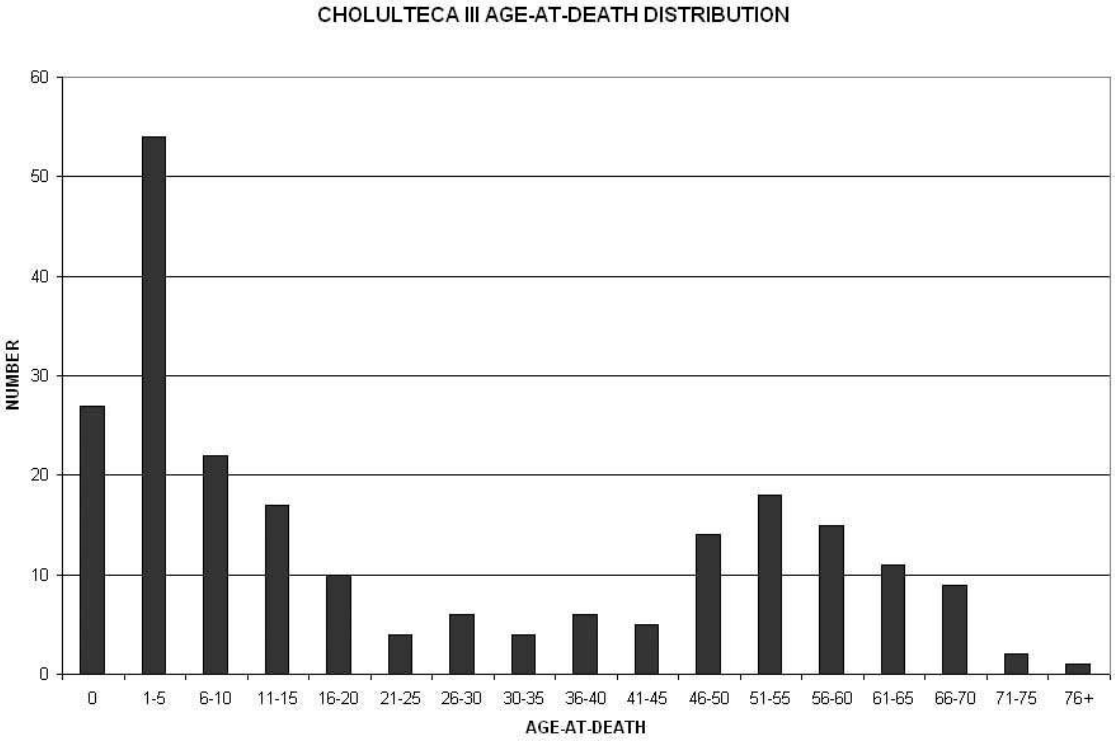

Table 2. Cholulteca III age-at-death distribution.

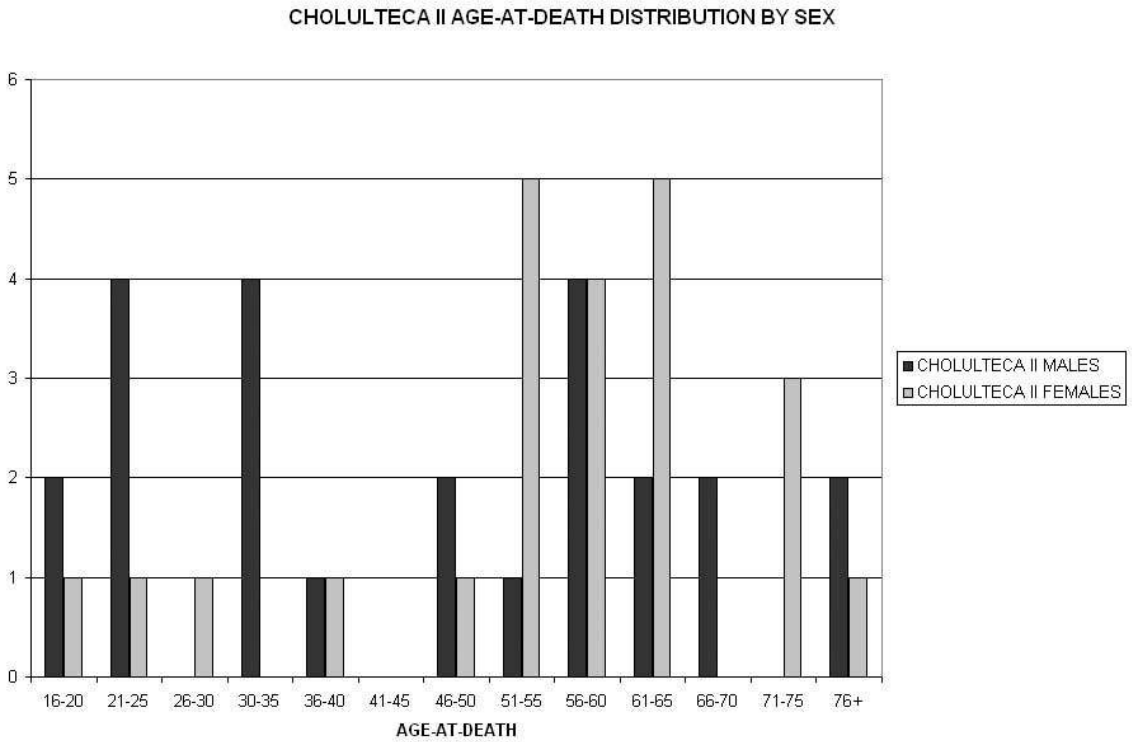

Table 3. Cholulteca II age-at-death distribution by sex.

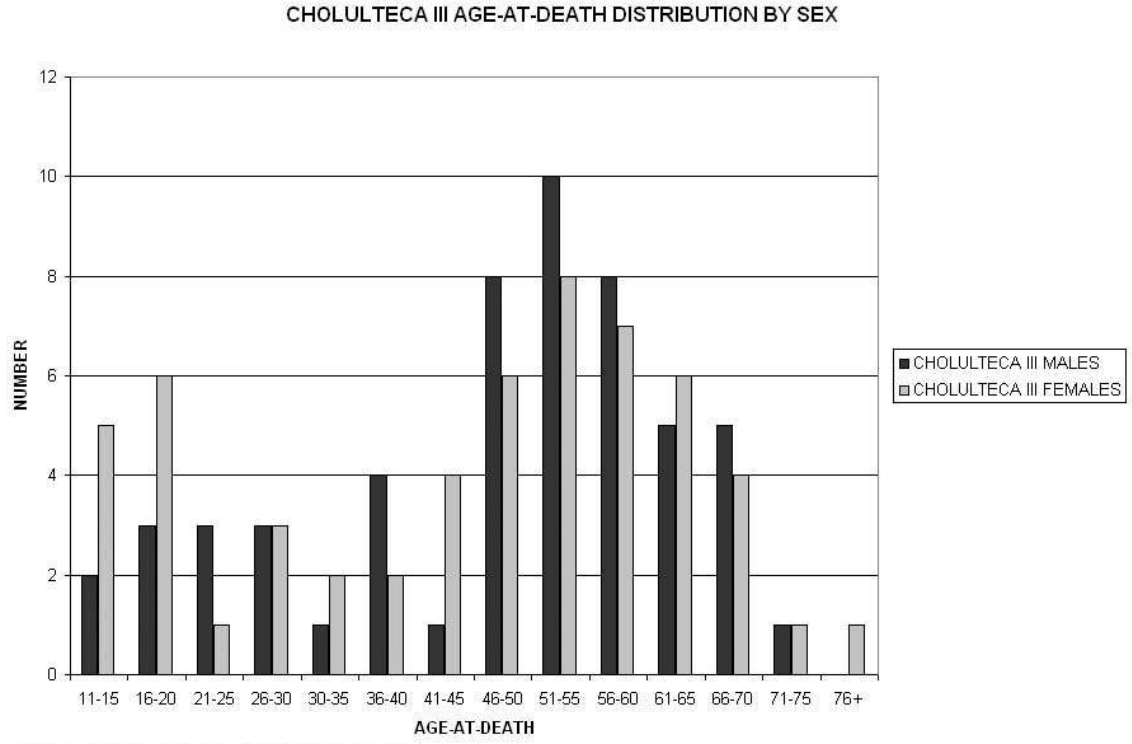

Table 4. Cholulteca III age-at-death distribution by sex.

Pathological Lesions and Mortality

The pathological lesions to be analyzed include porotic hyperostosis, periostitis and osteomyelitis, and enamel hypoplasias. Although paleopathological indicators of health are one of the few means available to determine if the urban residents of Cholula were, in fact, subject to nutritional stress or a heavy disease burden, these skeletal lesions are very difficult to interpret. Most diseases do not affect the skeleton or only do so after being present for a significant amount of time. Therefore, in many cases, diseases may not be evident at all from the skeletal sample or will only be observed in individuals who survived long enough for lesions to form. As a result, Wood *et al.* (1992) have suggested that hidden heterogeneity in risks and selective mortality are two factors that can confound the interpretation of skeletal lesions because healthier individuals may actually develop more skeletal lesions than those individuals in poorer health. A method of modeling hidden heterogeneity and selective mortality has been proposed that addresses some of these concerns and this model will be used to analyze the health of the Cholula population once it is fully tested (Usher 2000; Wood *et al.* 2002). Until that time, a maximum likelihood statistical analysis, as implemented by Ferrell (2003), of the relationship between particular pathological lesions and age-at-death will be completed to determine if the presence of certain pathologies indicate an increased risk of death, thus addressing the issue of 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 clearly 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 diploe. The diploe 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 a 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 is likely to be the cause of porotic hyperostosis and cribra orbitalia. The iron deficiency anemia that causes the skeletal lesions is thought to be the result of several factors including malnutrition, infections, and parasites, as demonstrated by the studies of El-Najjar *et al.* (1976); Stuart-Macadam (1987); Mensforth *et al.* (1978); and Holland and O'Brien (1997). A synergistic relationship exists between malnutrition and infection. Malnourished individuals often have suppressed immune systems and are, therefore, more susceptible to infections. In turn, infections, particularly gastrointestinal infections, can lead to further malnutrition because iron absorption is diminished (Palkovich 1987: 528-529). Parasites have also

been implicated as a possible cause of porotic hyperostosis, since some parasites cause intestinal bleeding and others interfere in the absorption of iron in the intestine (Wright and Chew 1999: 925).

Therefore, at least in theory, porotic hyperostosis and cribra orbitalia can reflect the nutritional status, infectious disease experience, and parasite burden of a population, but as with most skeletal lesions, this pathology can be difficult to interpret. Stuart-Macadam (1988) has suggested that porotic hyperostosis may, in fact, indicate a healthy response to anemia. In other words, it is possible that sicker individuals would die before their bodies could mount such a defense. However, in her analysis of the Tirup skeletal sample, Bethany Usher (2002) found that individuals with cribra orbitalia in the medieval Danish village were almost five times more likely to die than those without the lesions. The graphs below show the number of individuals in each age category with lesions of porotic hyperostosis or cribra orbitalia. These preliminary data from the Cholula population indicate that the presence of these lesions does correspond to an increased risk of death. Further investigation of this issue is planned to verify these results.

Further analysis to be completed regarding porotic hyperostosis and cribra orbitalia in the Cholula population:

1. A maximum likelihood statistical analysis will be done to determine how the presence of these lesions affects mortality in the Cholulteca II and Cholulteca III populations.
2. A comparison of the Cholulteca II and Cholulteca III populations will be done to determine if there was change over time.

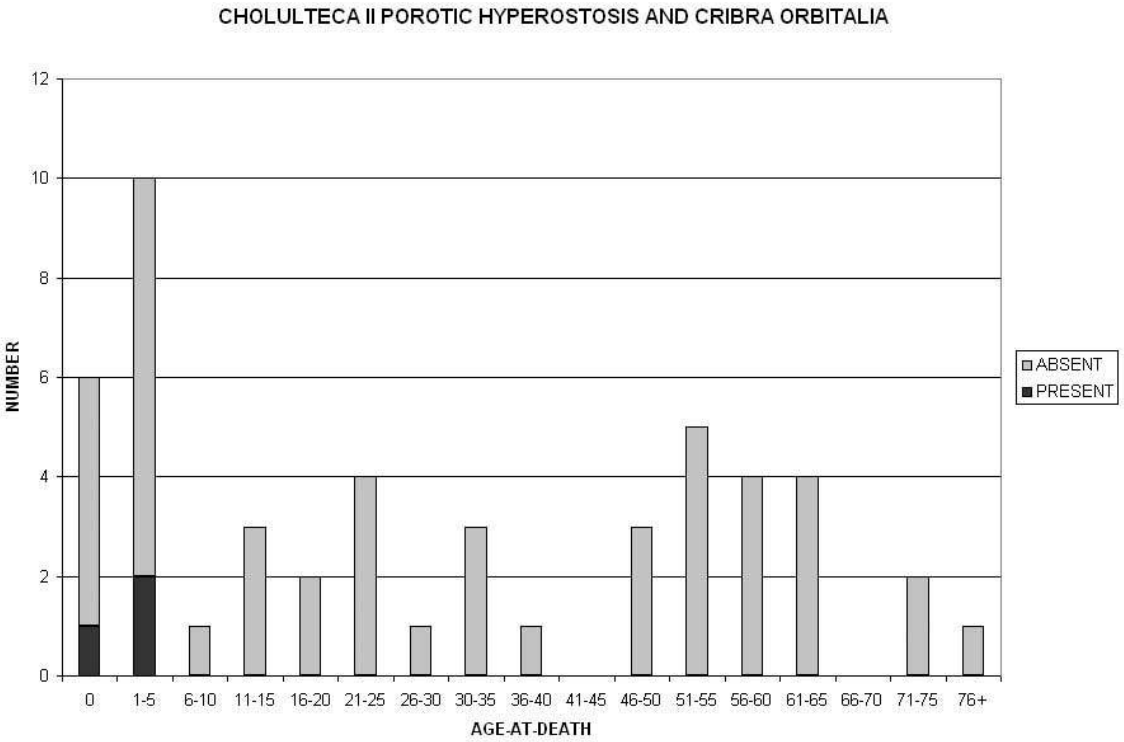

Table 5. Cholulteca II porotic hyperostosis and cribra orbitalia.

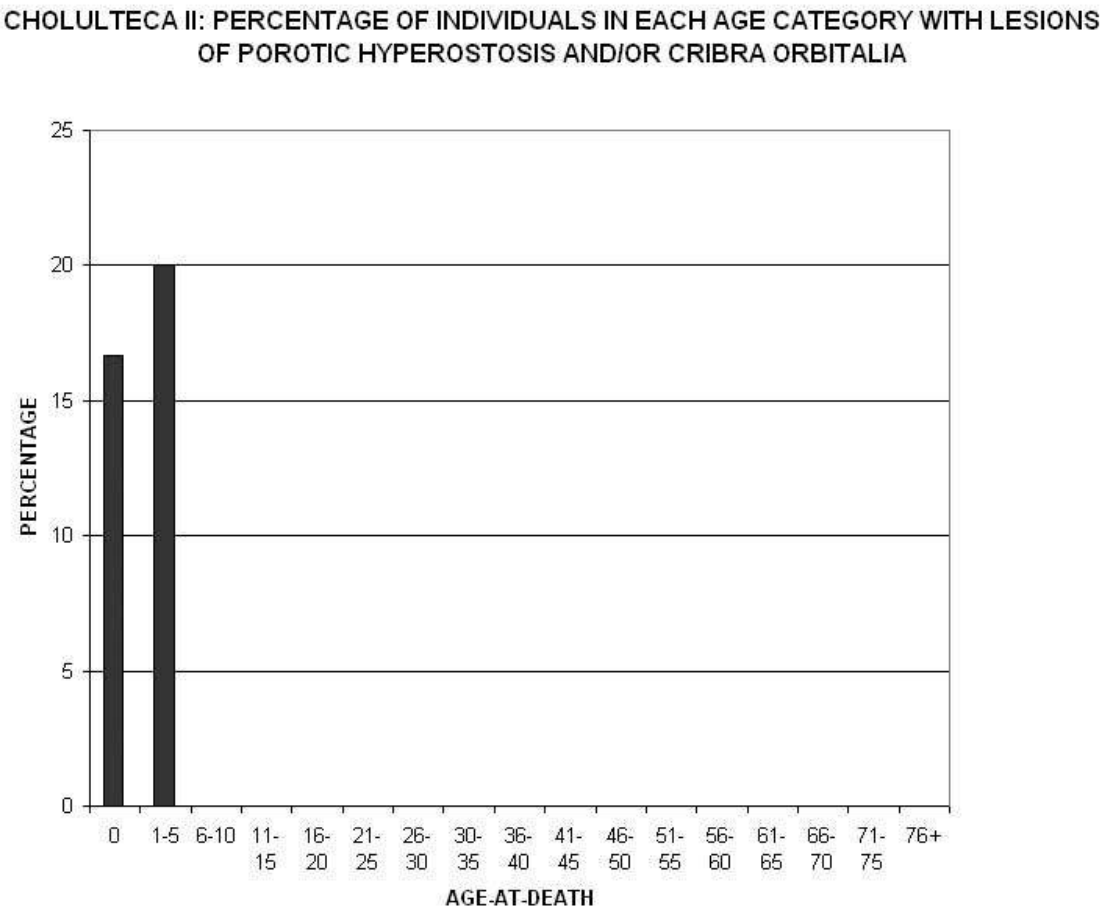

Table 6. Cholulteca II: Percentage of individuals in each age category with lesions of porotic hyperostosis and/or cribra orbitalia.

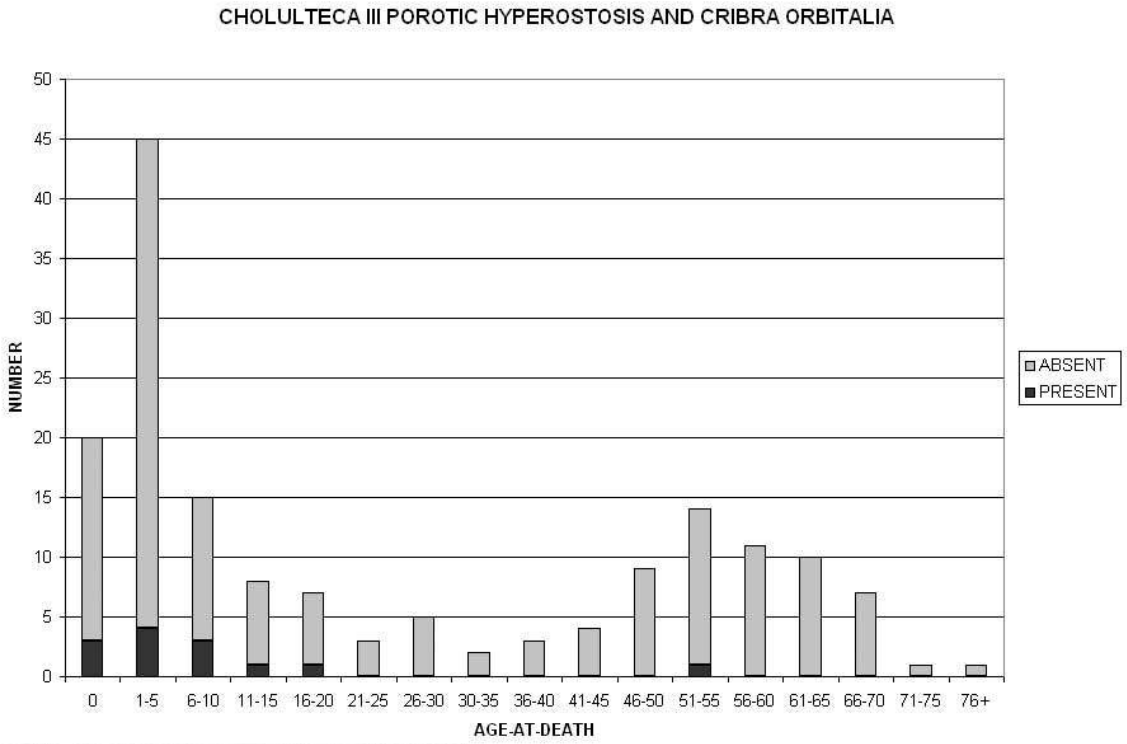

Table 7. Cholulteca III porotic hyperostosis and cribra orbitalia.

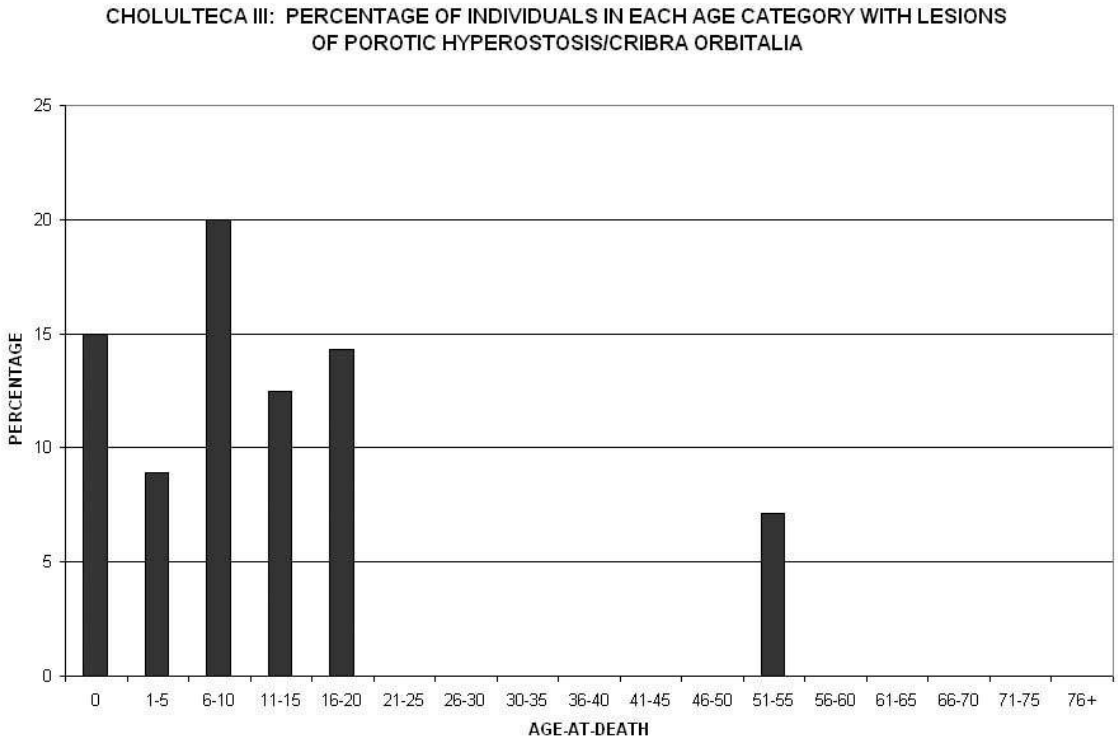

Table 8. Cholulteca III: Percentage of individuals in each age category with lesions of porotic hyperostosis and/or cribra orbitalia.

Infectious Lesions

Various skeletal indicators of specific and non-specific infections will also be assessed. As the skeleton can only respond to stress in a limited number of ways, namely 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 changes in bone consistent with the presence of infection. Periostitis occurs when a bacterial infection (or in some cases 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. In cases in which an infection is particularly severe, multiple parts of the skeleton may display lesions and the normal shapes of the bones may be somewhat distorted. Less severe infections produce smaller, more localized lesions (Roberts and Manchester 1997: 129-130). Osteomyelitis is a more severe form of bone infection in which bacteria enter the medullary cavity as a result of either a systemic infection or direct injury to the bone. Osteomyelitis, which tends to affect the tibia more frequently than other bones, can be differentiated from periostitis by the presence of cloacae (which allow for the discharge of pus), involucrum, and changes in the marrow cavity (Ortner and Putschar 1981). Preliminary data from the Cholulteca II and Cholulteca III populations is shown below. Although statistical analysis is required to form any definitive conclusions, it appears that the presence of infectious lesions in this population may indicate that, at least for particular ages, the individual actually has a decreased risk of death, as very young individuals are less likely to have these lesions. In other words, the youngest individuals in the population likely die of the infection before skeletal lesions have a chance to form.

Further analysis to be completed regarding infectious lesions in the Cholula population:

1. A maximum likelihood statistical analysis will be done to determine how the presence of these lesions affects mortality in the Cholulteca II and Cholulteca III populations.
2. A maximum likelihood statistical analysis will be done to determine if infectious lesions in different bones have different effects on mortality.
3. A maximum likelihood statistical analysis will be done to determine if healed and unhealed lesions have different effects on mortality.
4. A comparison of these results for the Cholulteca II and Cholulteca III populations in order to determine if there was change over time.

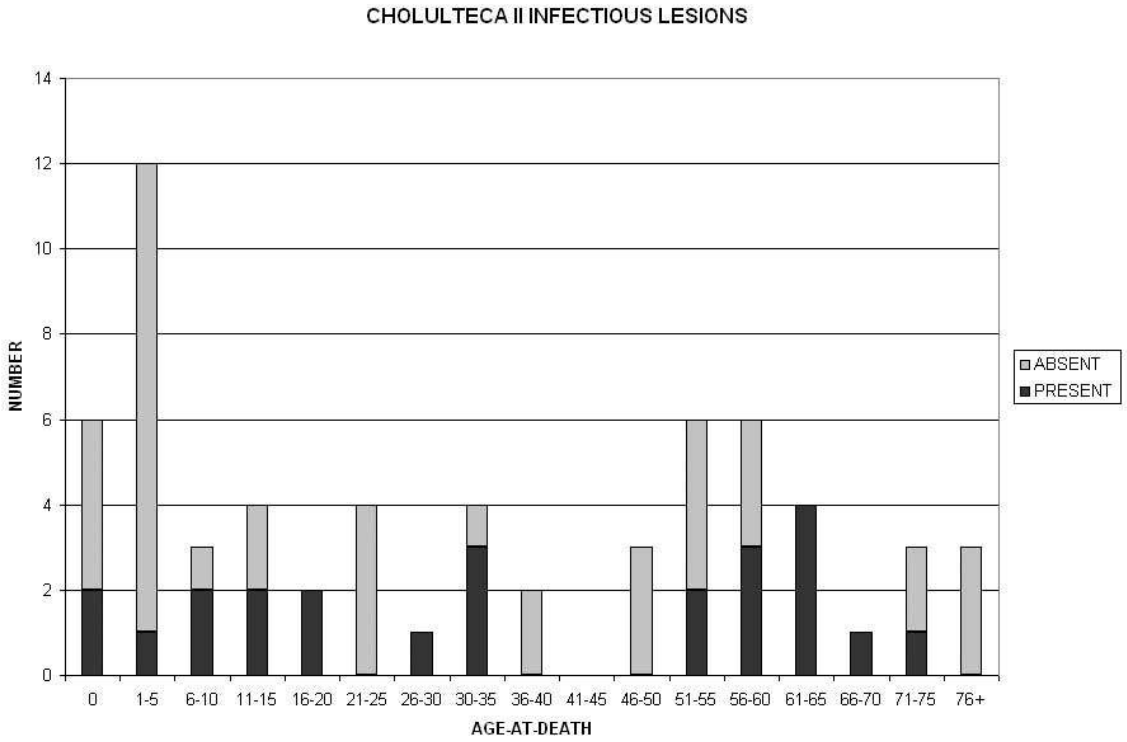

Table 9. Cholulteca II infectious lesions.

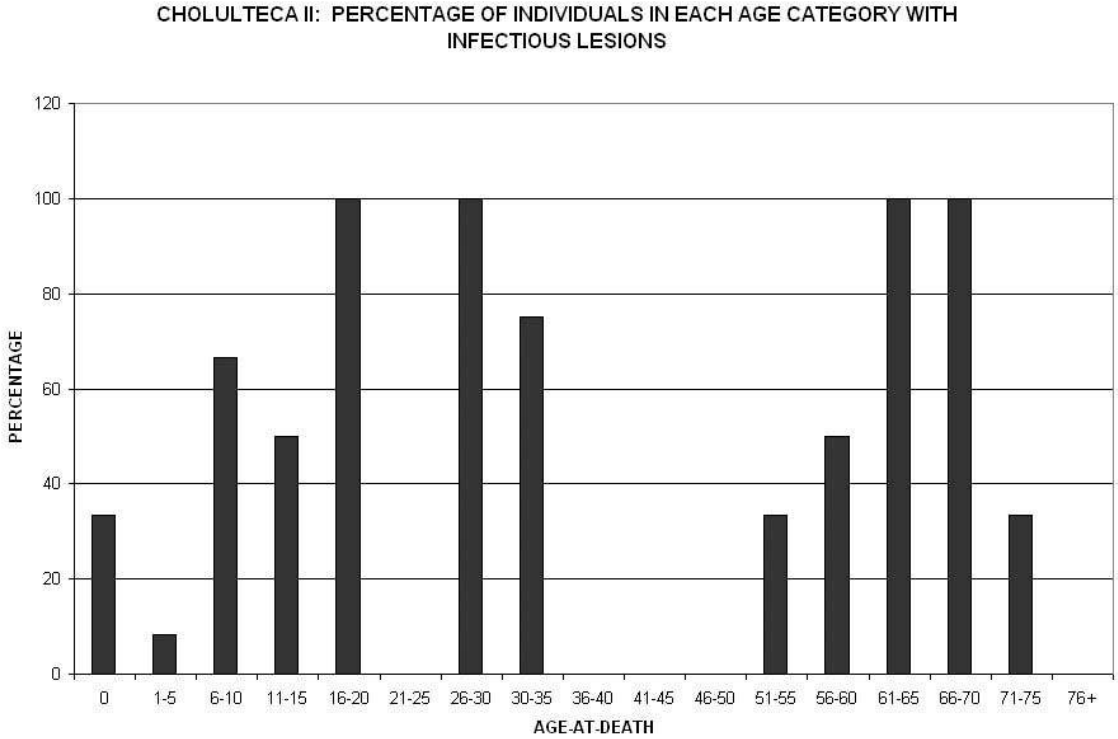

Table 10. Cholulteca II: Percentage of individuals in each age category with infectious lesions.

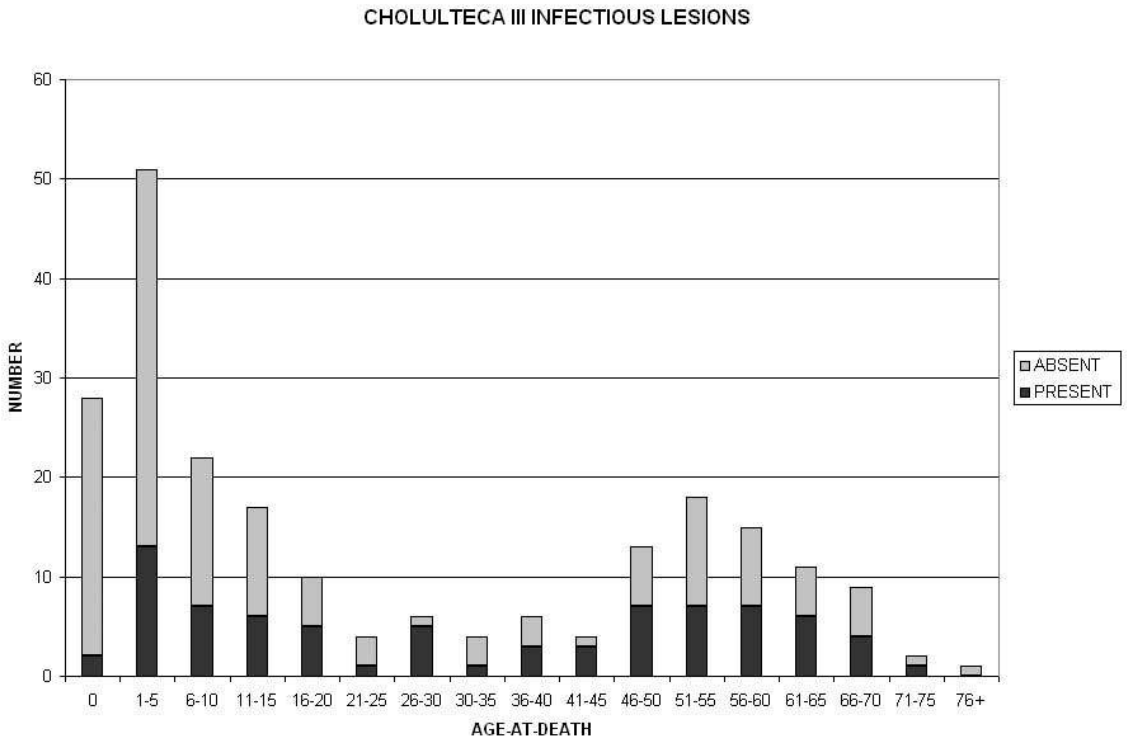

Table 11. Cholulteca III infectious lesions.

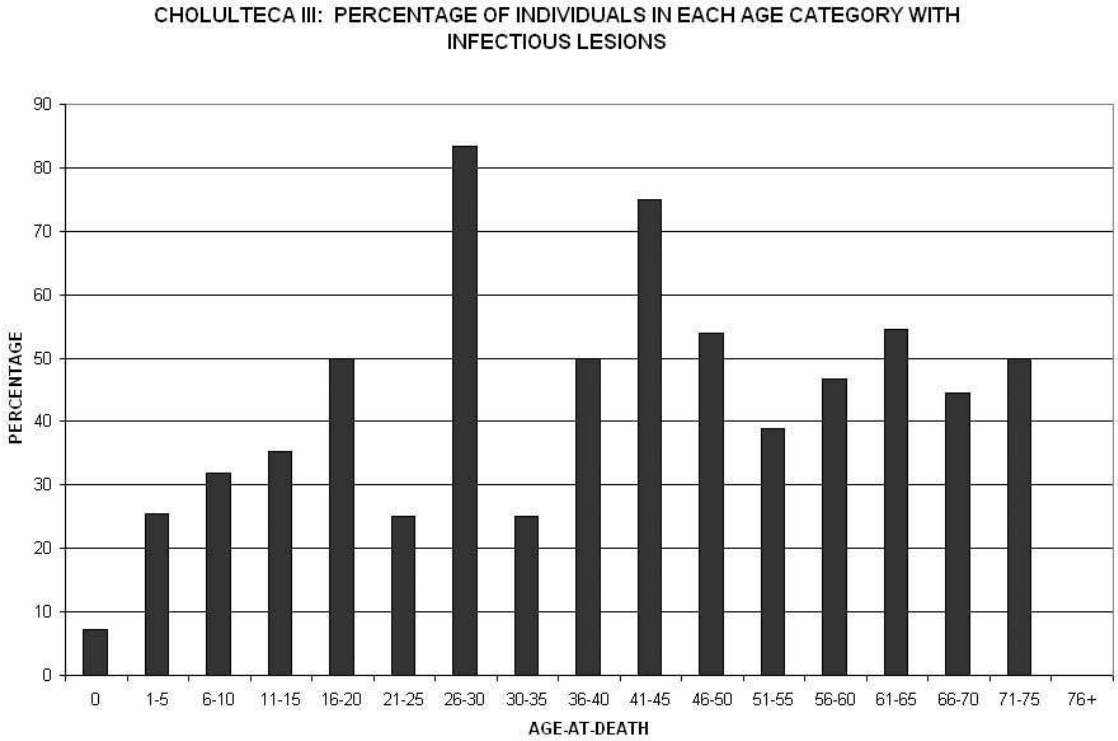

Table 12. Cholulteca III: Percentage of individuals in each age category with infectious lesions.

Enamel Hypoplasias

The final pathology that will be considered is enamel hypoplasia. Enamel hypoplasia is a pathology that appears on the teeth as transverse lines or grooves, primarily on the buccal surfaces of teeth. These lines occur during the development of the teeth, when some sort of stress interrupts enamel formation (Goodman *et al.* 1984). Enamel hypoplasias can be caused by either nutritional deficiencies or childhood illnesses, but the stress must last anywhere from a few weeks to two months for enamel formation to be disrupted (Roberts and Manchester 1997: 58-59; Skinner and Goodman 1992). For the Cholula population, hypoplasias were scored for the maxillary central incisor, the mandibular canine, and the mandibular first, second, and third molars. The data for the Cholulteca II and Cholulteca III populations is shown below for permanent teeth. It appears the hypoplasias on the canine, and possibly the incisor, have no effect on the risk of death, as they are present in most individuals regardless of the age-at-death. Hypoplasias on the molars, on the other hand, appear to be associated with an increased risk of death, given that they are more common among individuals dying at younger ages. More analyses are intended to confirm these findings.

Further analysis to be completed regarding enamel hypoplasias in the Cholula population:

1. A maximum likelihood analysis will be done for each tooth (incisor, canine, first molar, second molar, and third molar) to determine how the presence of hypoplasias affects mortality in the Cholulteca II and Cholulteca III populations and to identify how hypoplasias in particular teeth may differently affect mortality.
2. The age at which individual hypoplasias occurred will be calculated and a maximum likelihood statistical analysis will be used to determine how the age at which a hypoplasia occurred affected mortality in the Cholulteca II and Cholulteca III populations.

3. A comparison of these results for the Cholulteca II and Cholulteca III populations to determine if change occurred over time at the site.

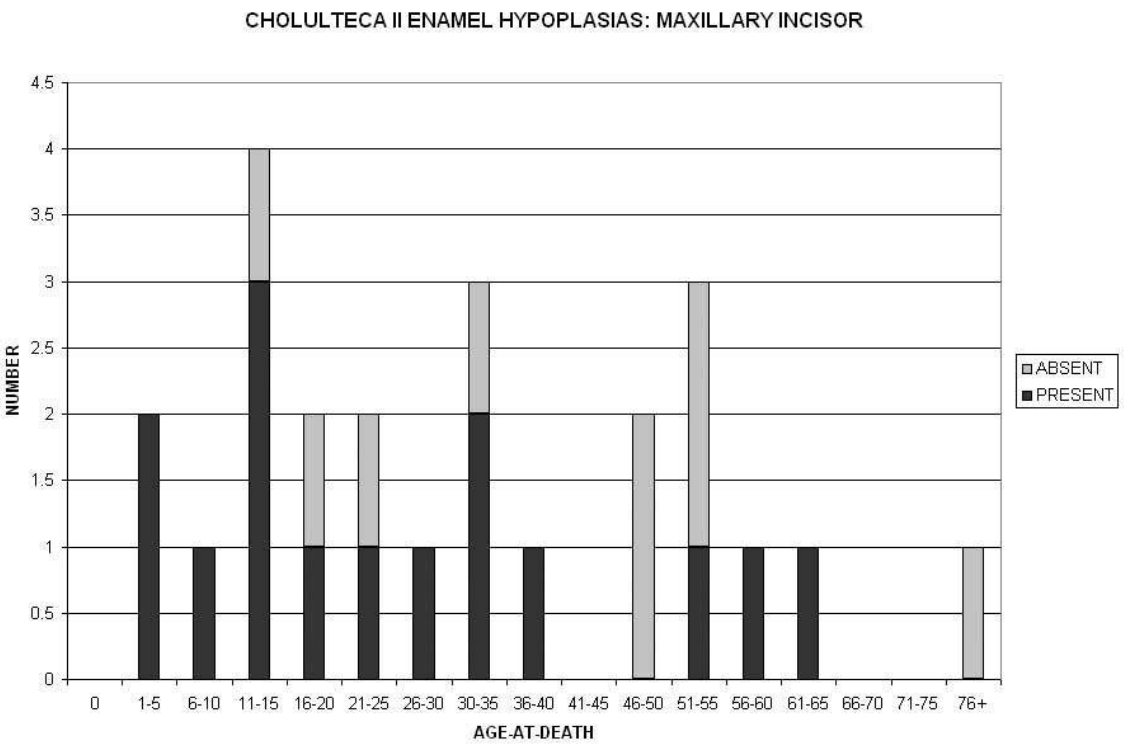

Table 13. Cholulteca II enamel hypoplasias: maxillary incisor.

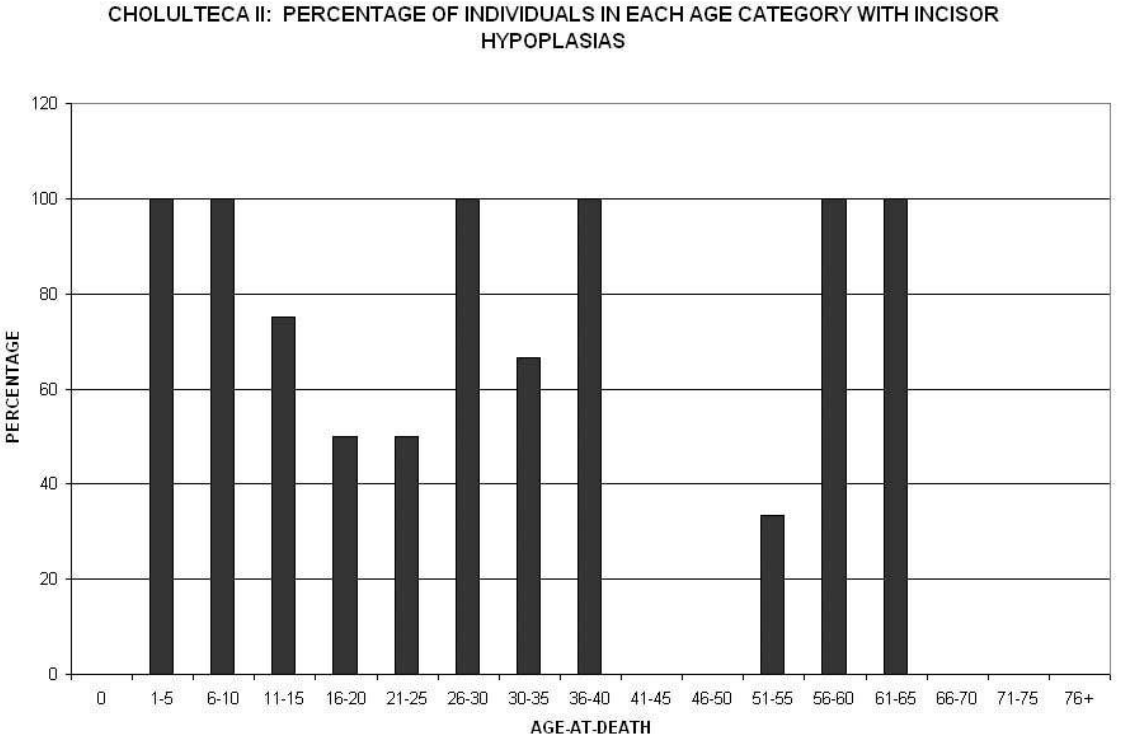

Table 14. Cholulteca II: Percentage of individuals in each age category with incisor hypoplasias.

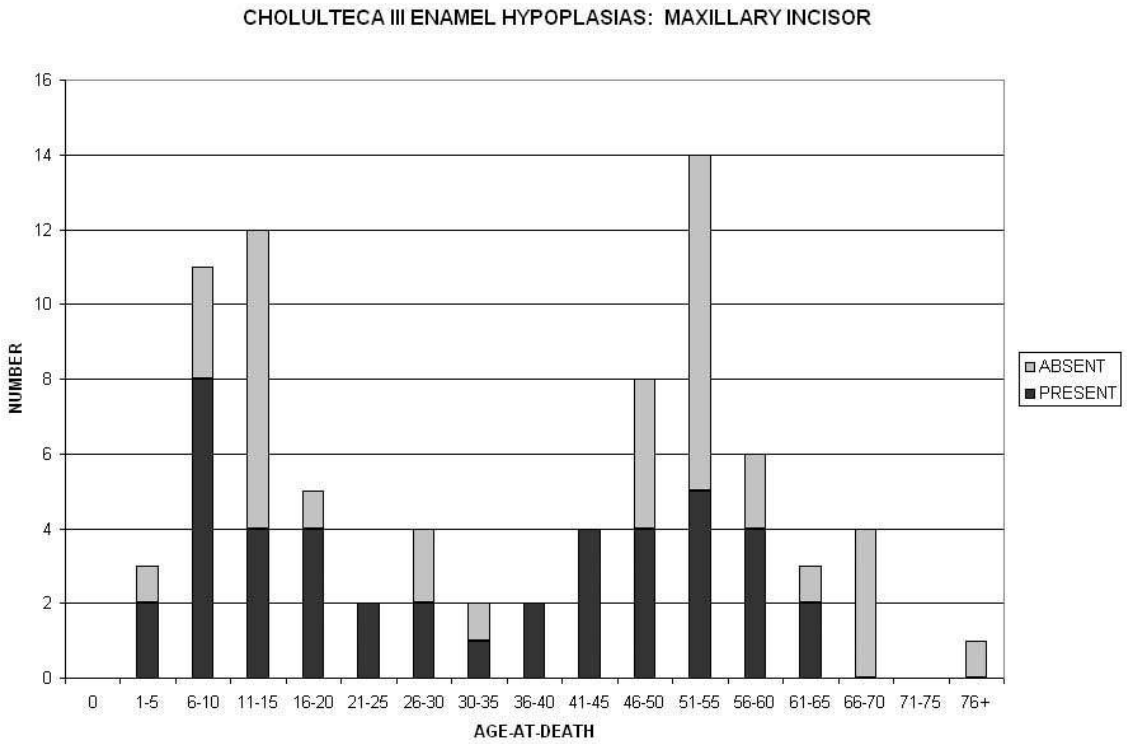

Table 15. Cholulteca III enamel hypoplasias: maxillary incisor.

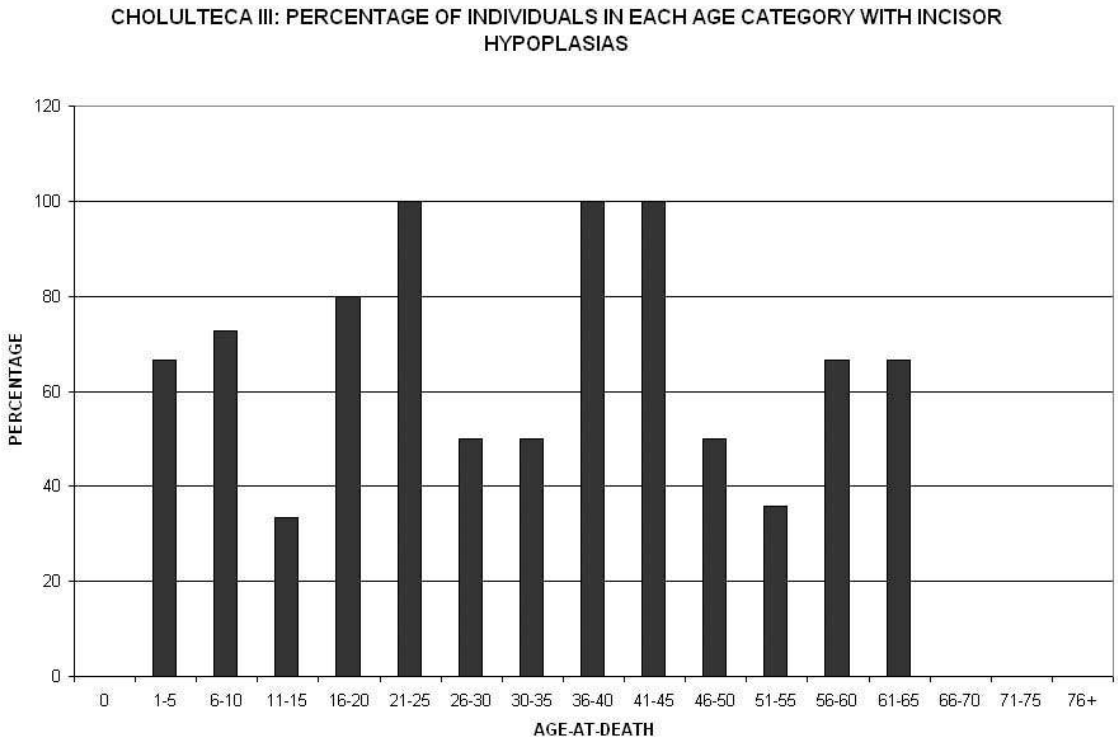

Table 16. Cholulteca III: Percentage of individuals in each age category with incisor hypoplasias.

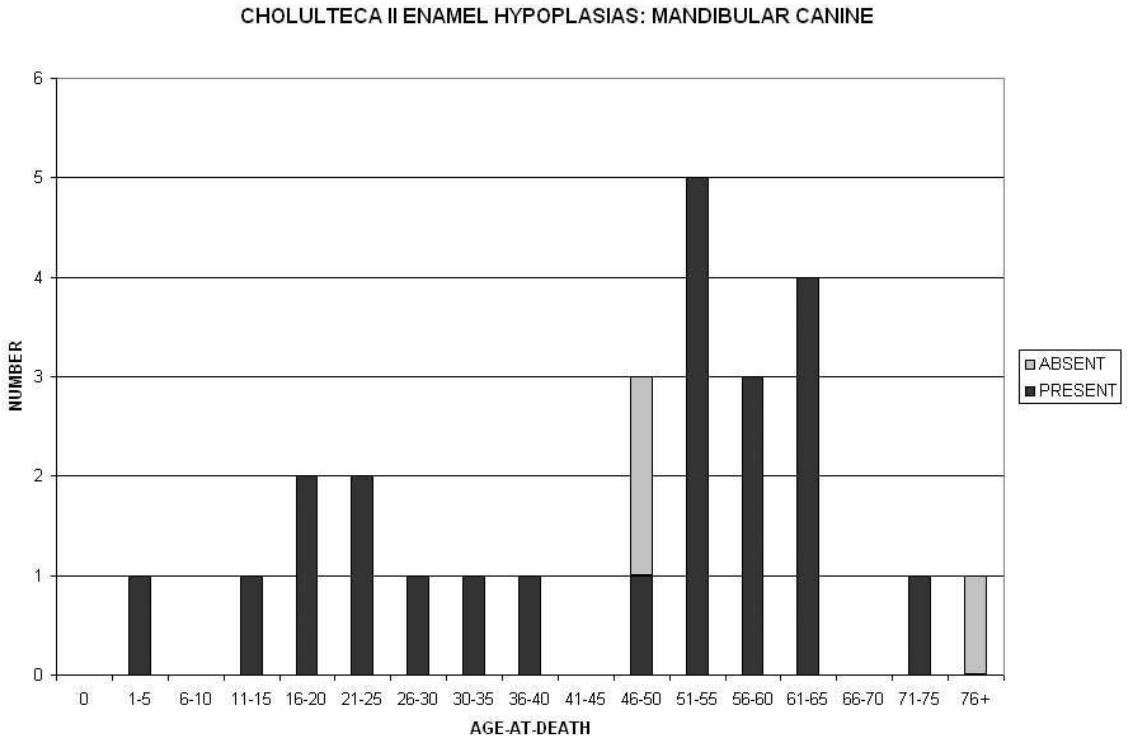

Table 17. Cholulteca II enamel hypoplasias: mandibular canine.

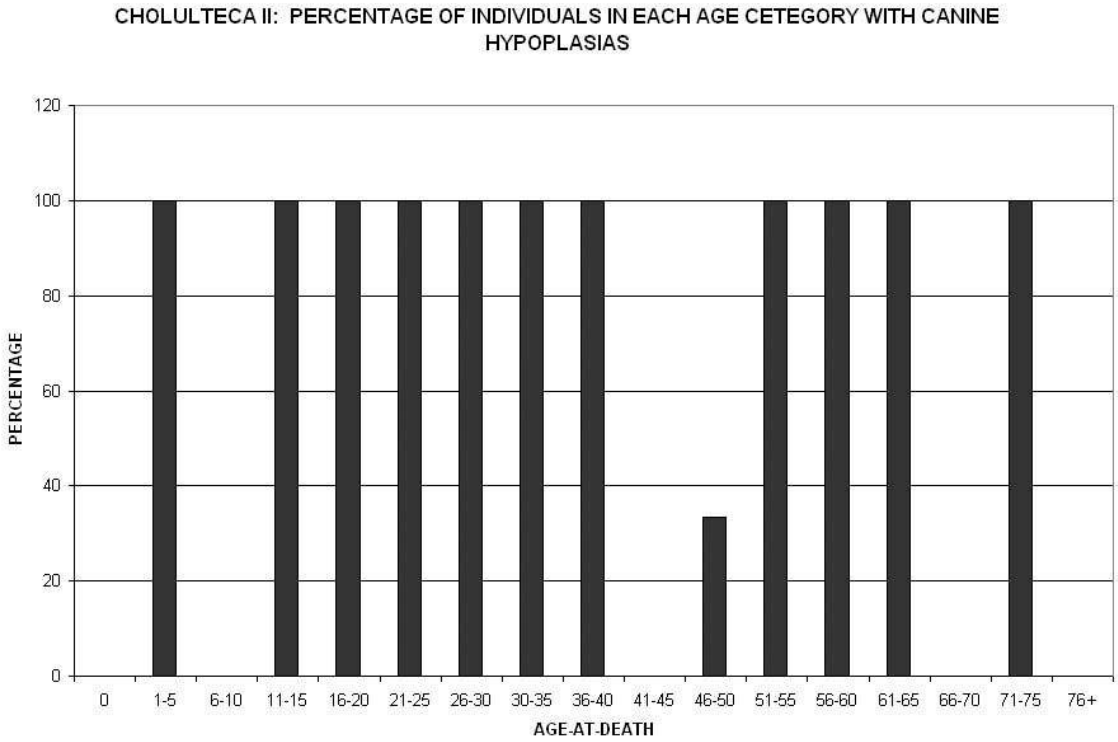

Table 18. Cholulteca II: Percentage of individuals in each age category with canine hypoplasias.

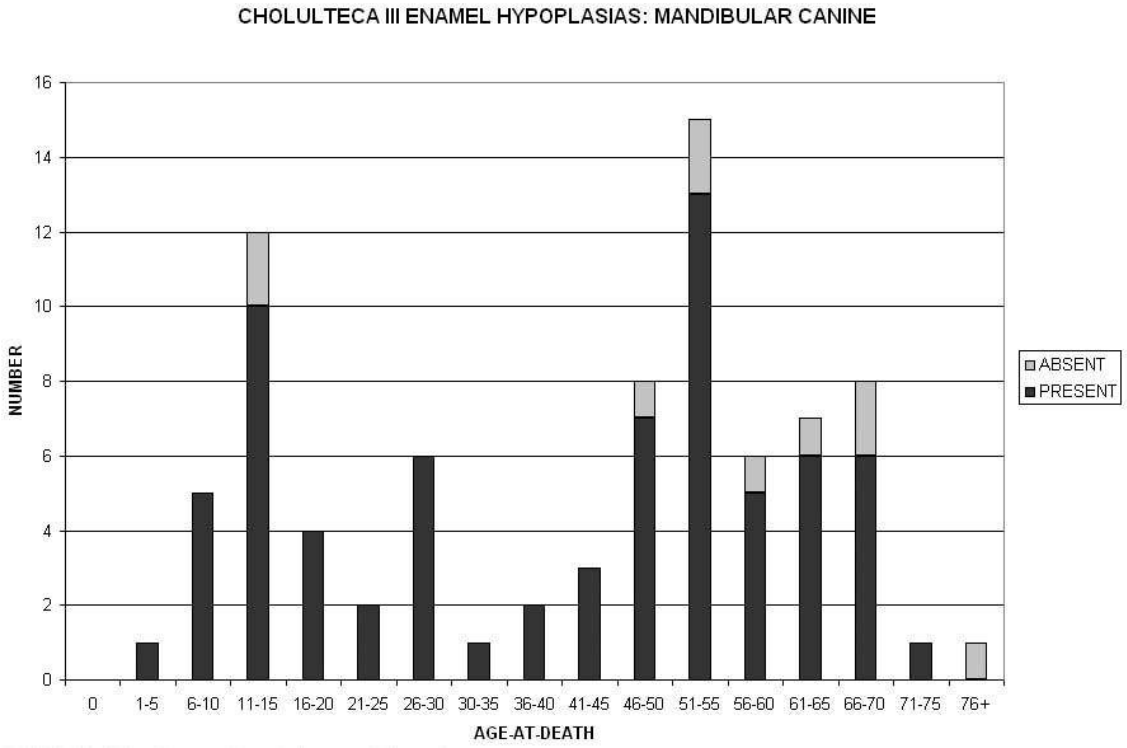

Table 19. Cholulteca III enamel hypoplasias: mandibular canine.

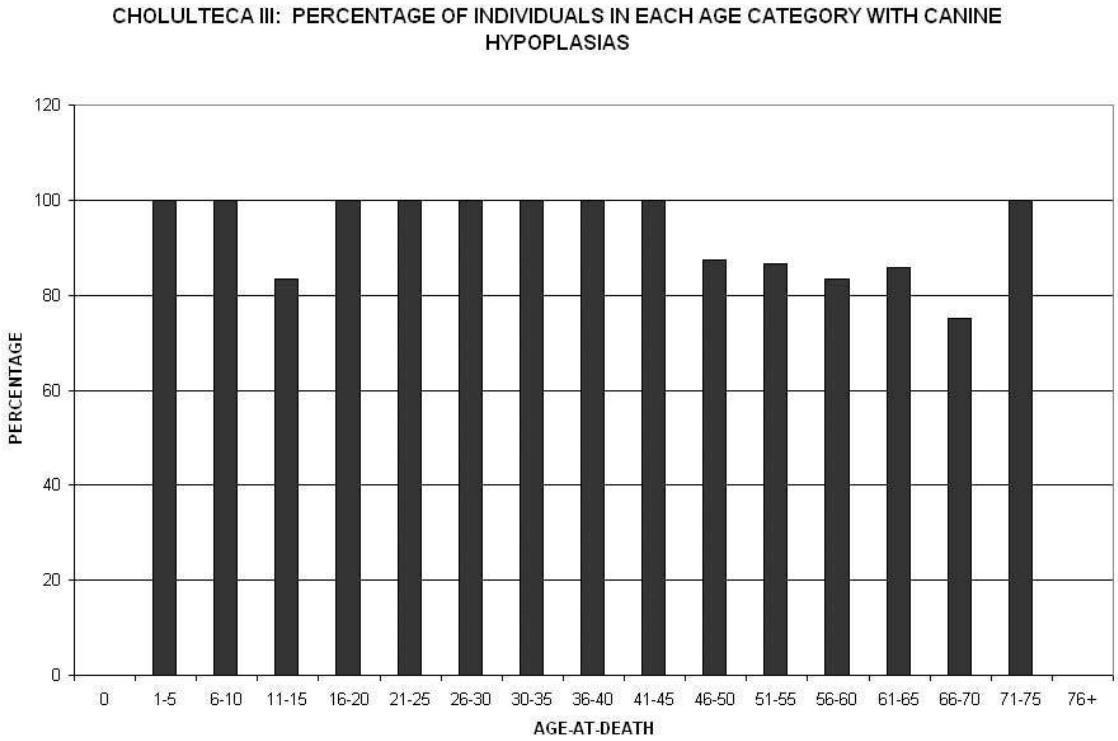

Table 20. Cholulteca III: Percentage of individuals in each age category with canine hypoplasias.

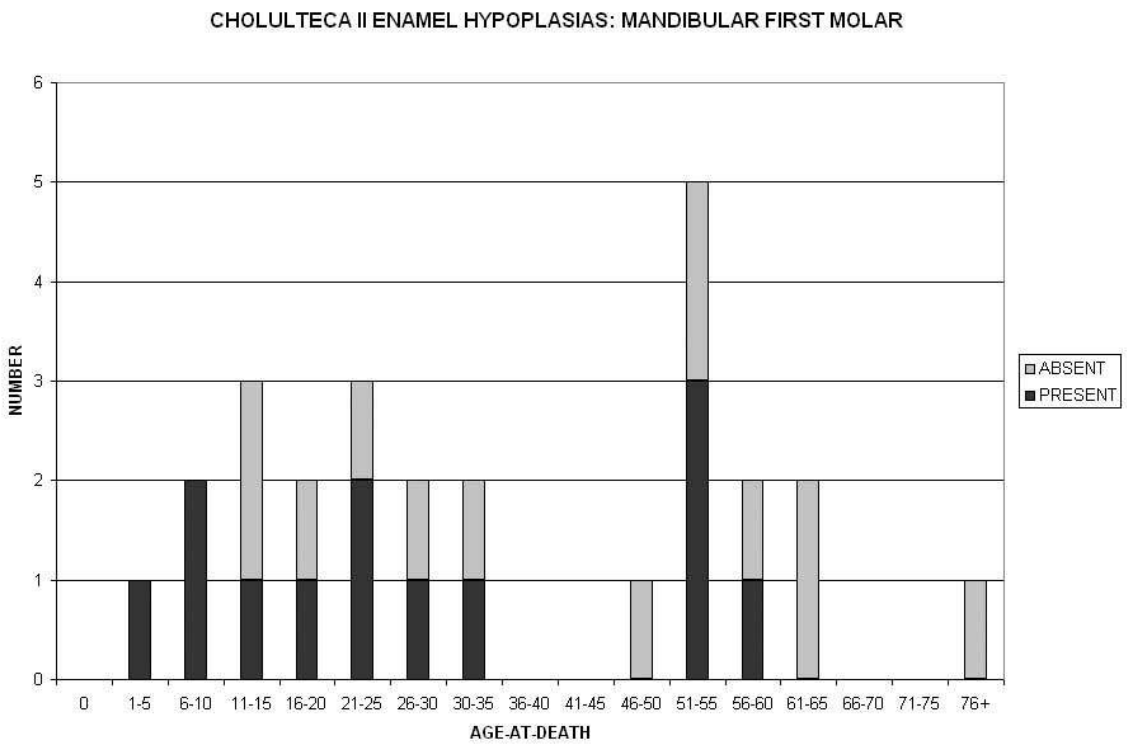

Table 21. Cholulteca II enamel hypoplasias: mandibular first molar.

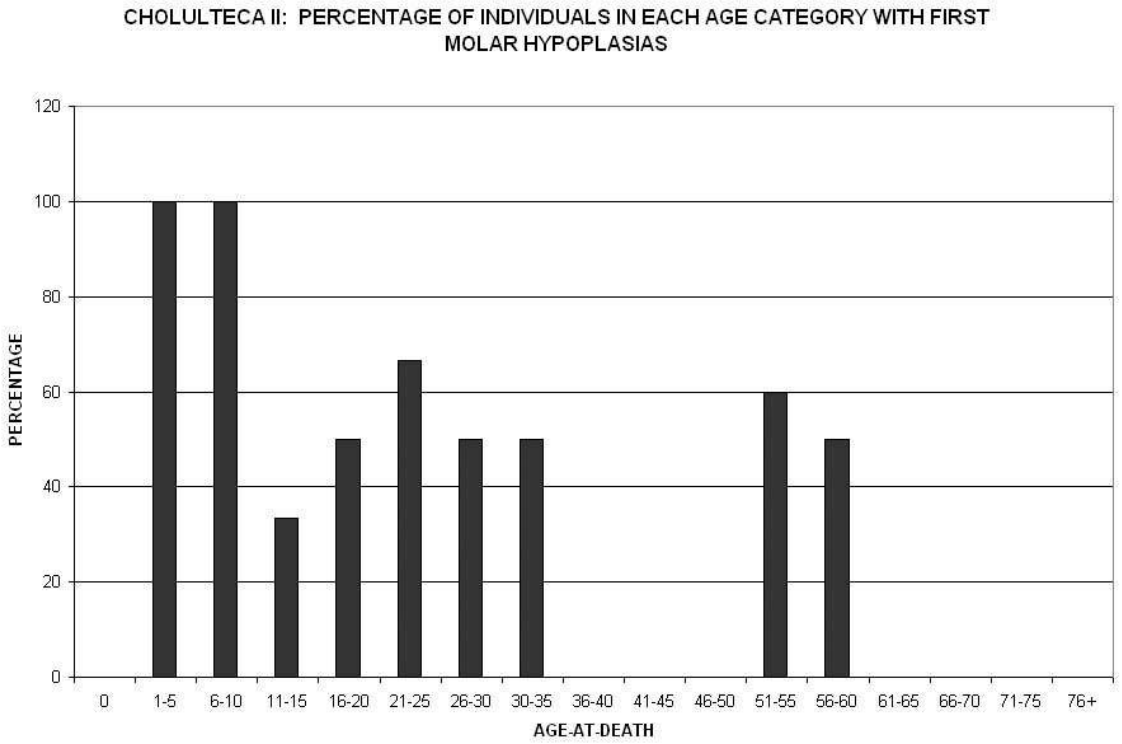

Table 22. Cholulteca II: Percentage of individuals in each age category with first molar hypoplasias.

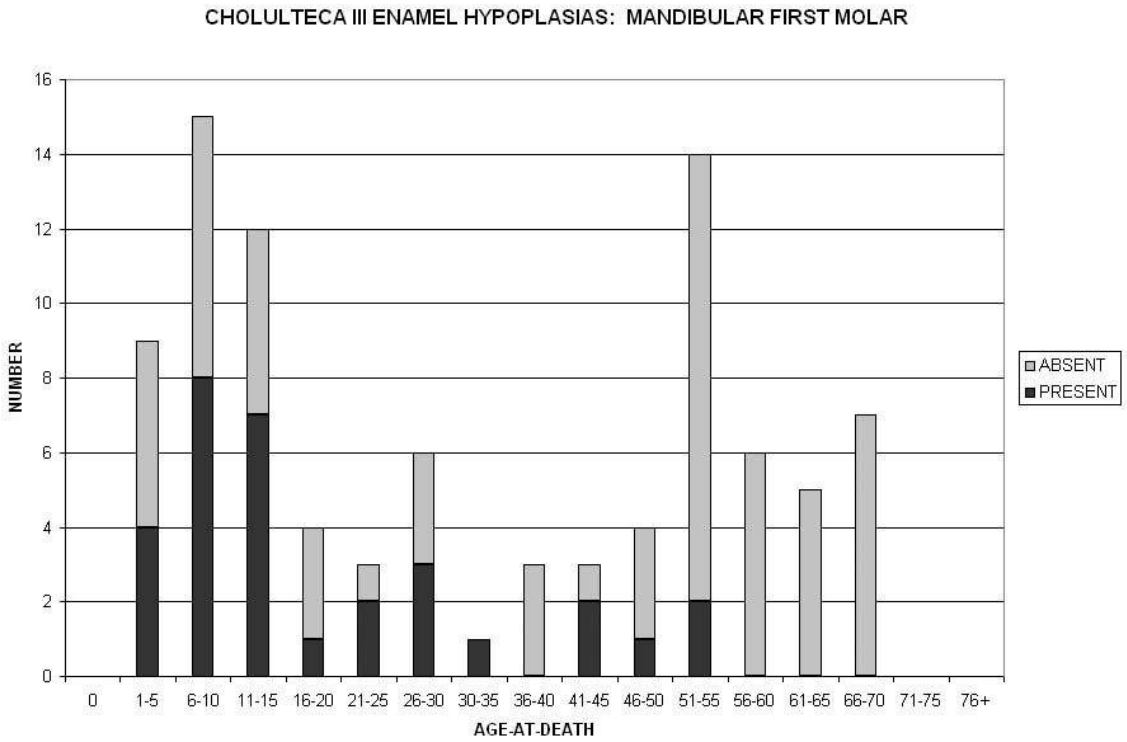

Table 23. Cholulteca III enamel hypoplasias: mandibular first molar.

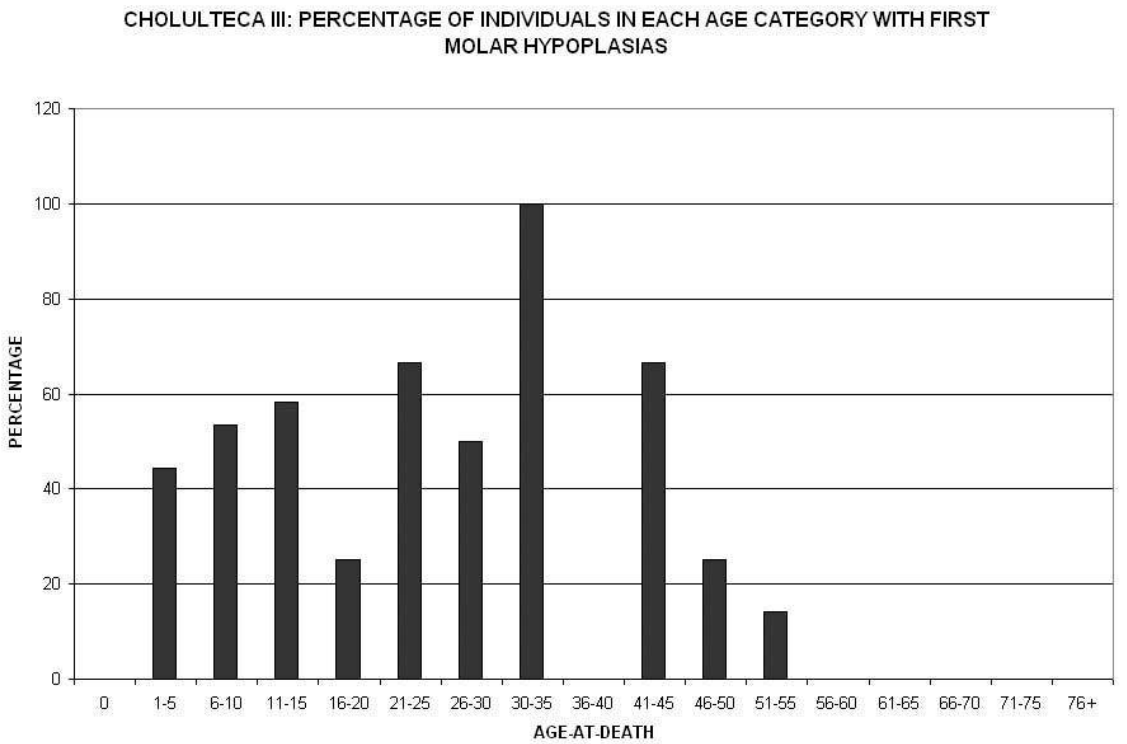

Table 24. Cholulteca III: Percentage of individuals in each age category with first molar hypoplasias.

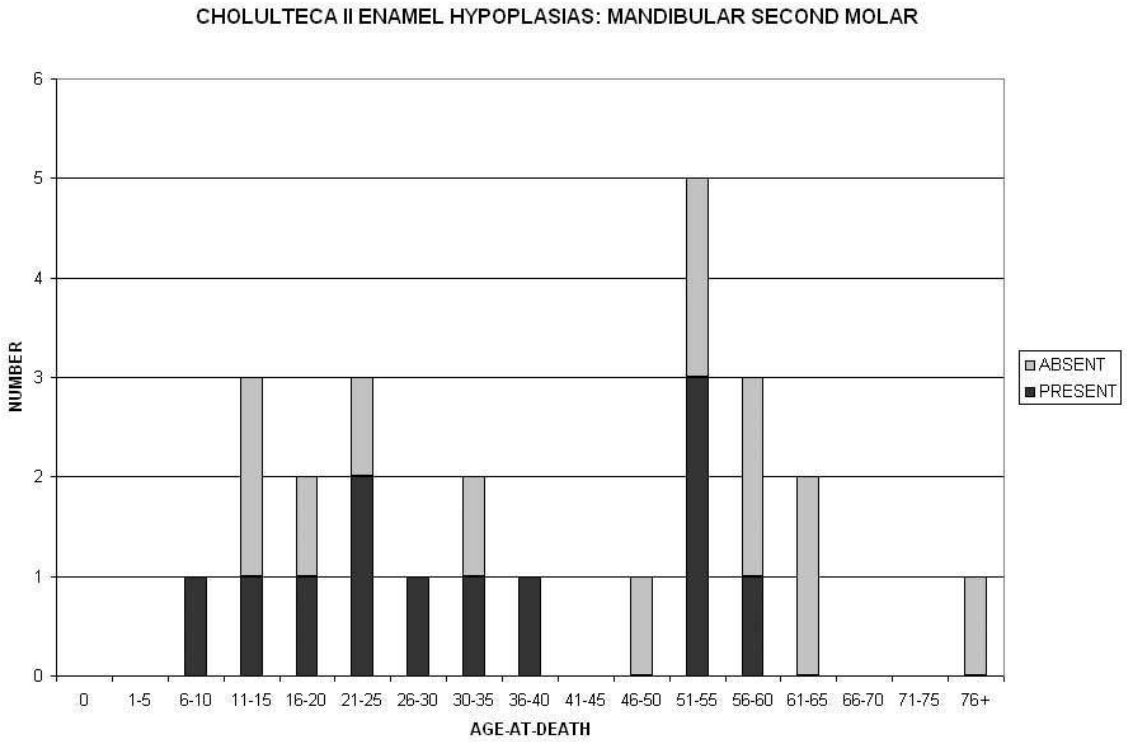

Table 25. Cholulteca II enamel hypoplasias: mandibular second molar.

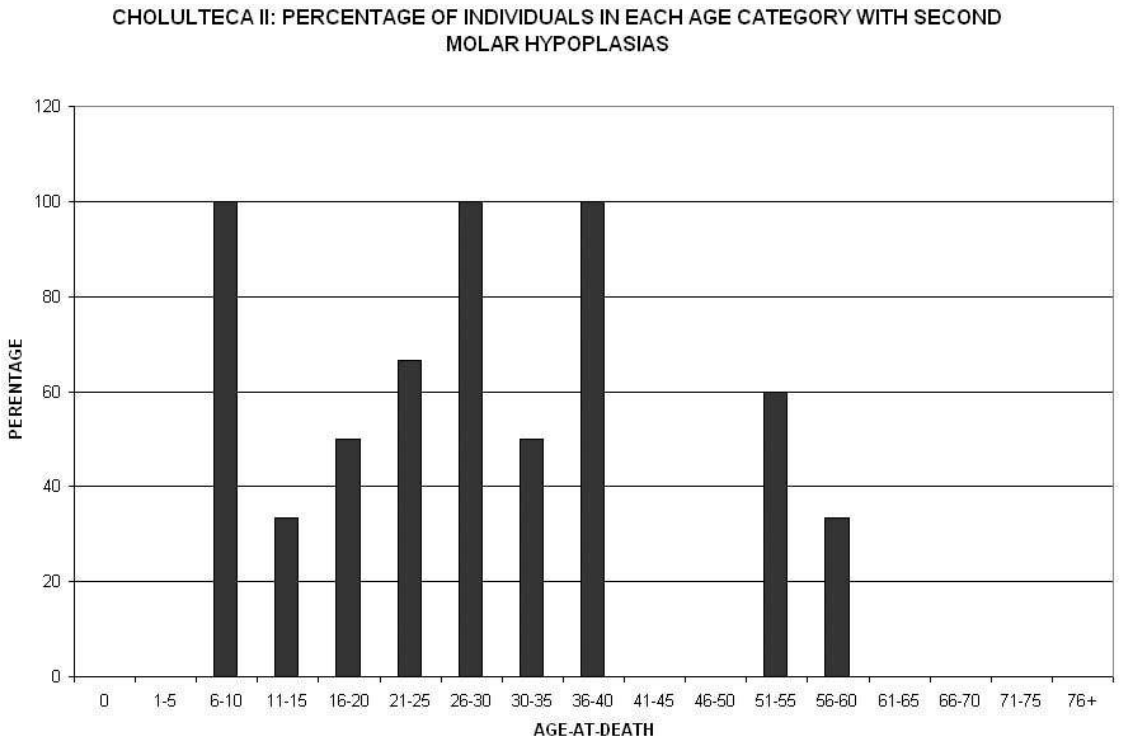

Table 26. Cholulteca II: Percentage of individuals in each age category with second molar hypoplasias.

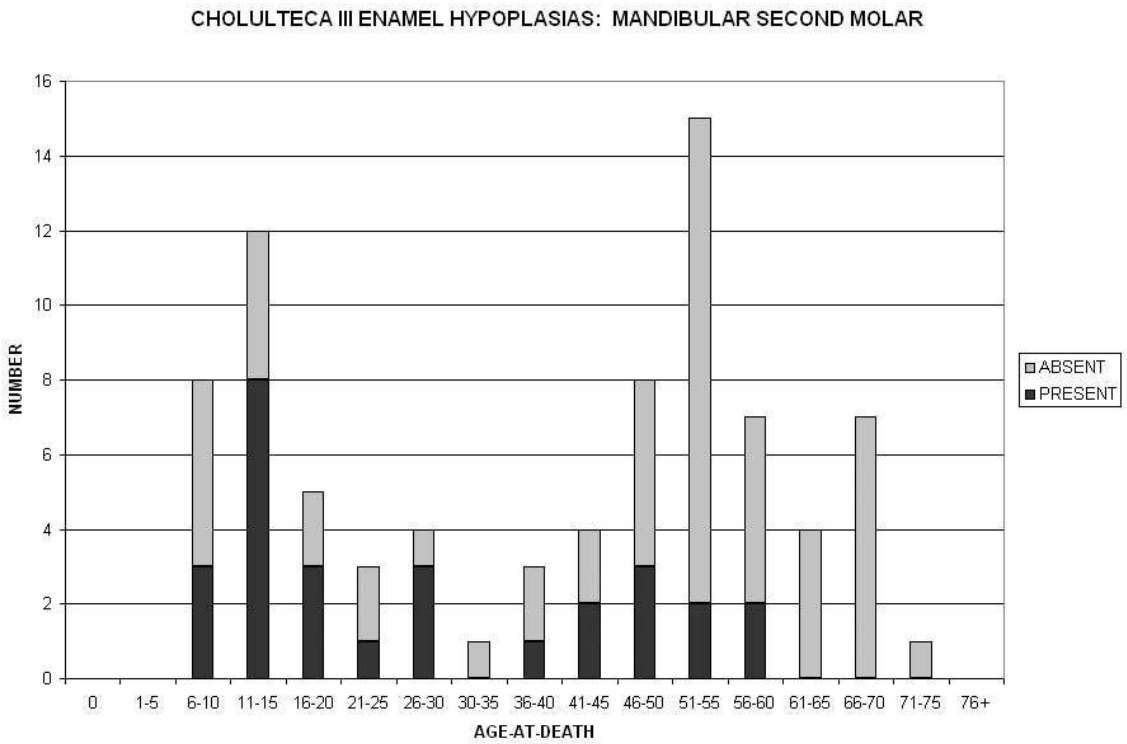

Table 27. Cholulteca III enamel hypoplasias: mandibular second molar.

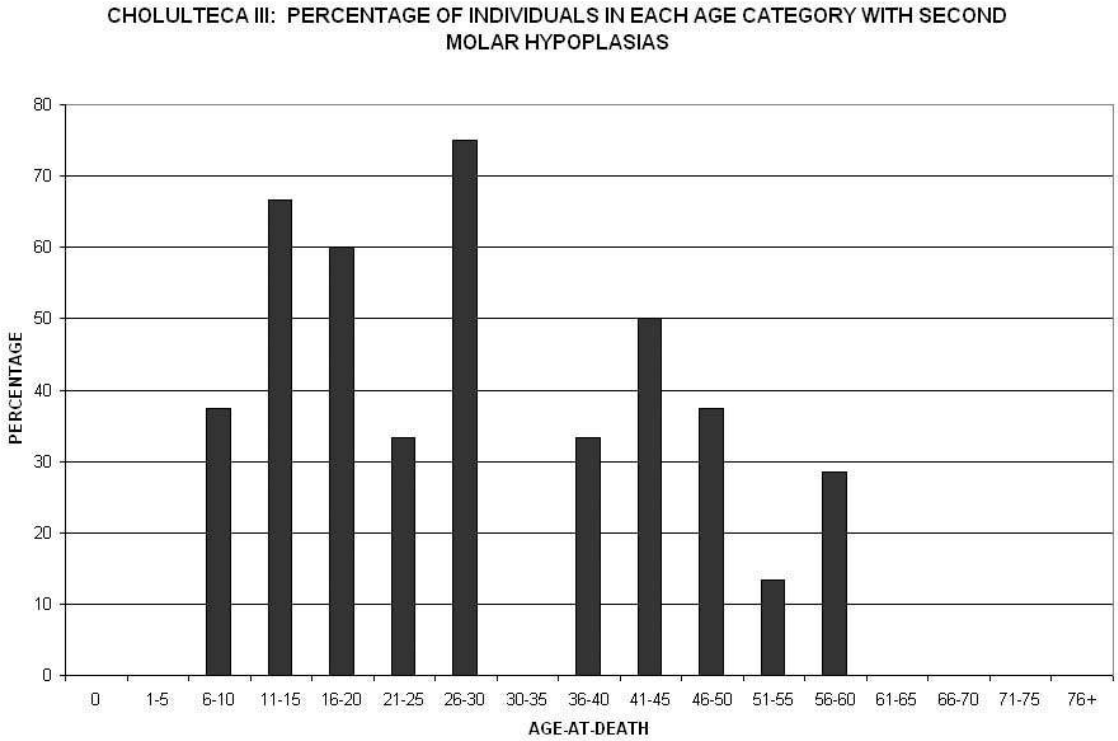

Table 28. Cholulteca III: Percentage of individuals in each age category with second molar hypoplasias.

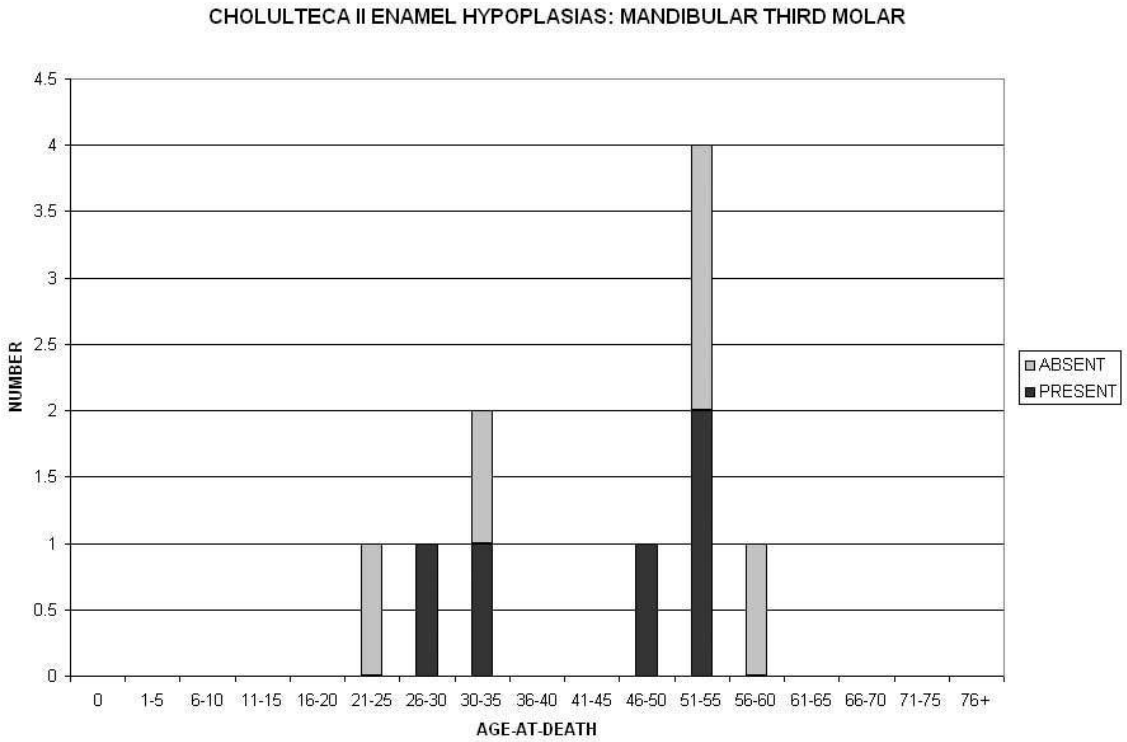

Table 29. Cholulteca II enamel hypoplasias: mandibular third molar.

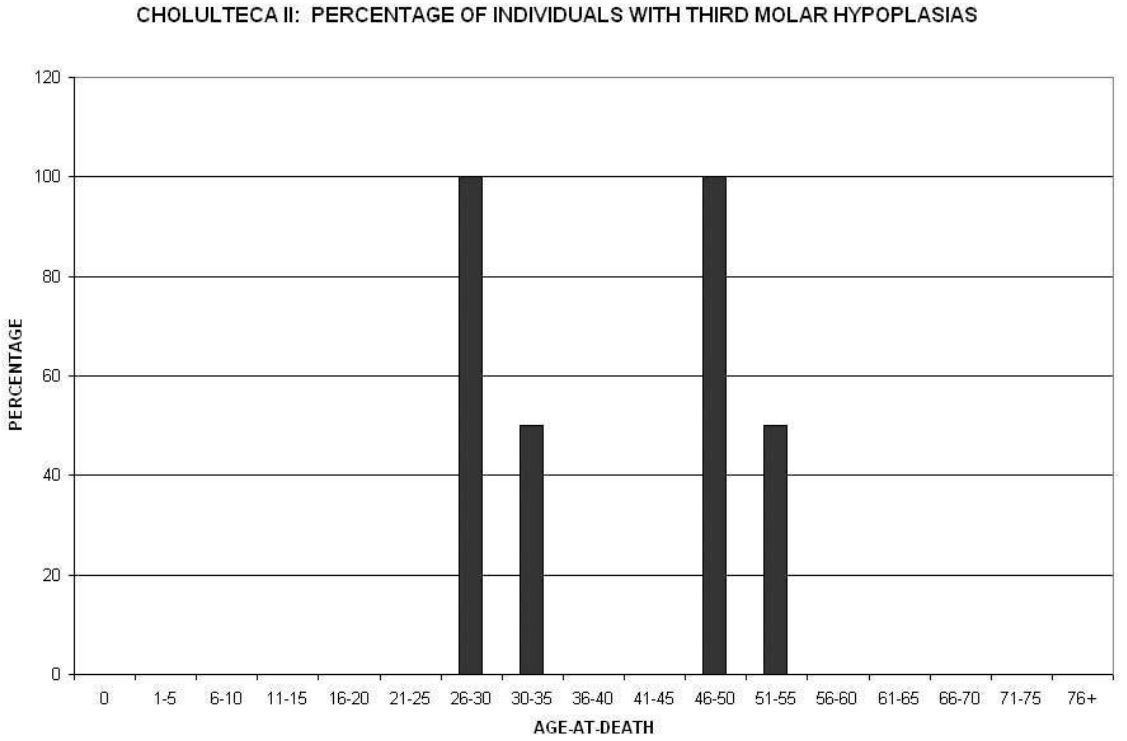

Table 30. Cholulteca II: Percentage of individuals in each age category with third molar hypoplasias.

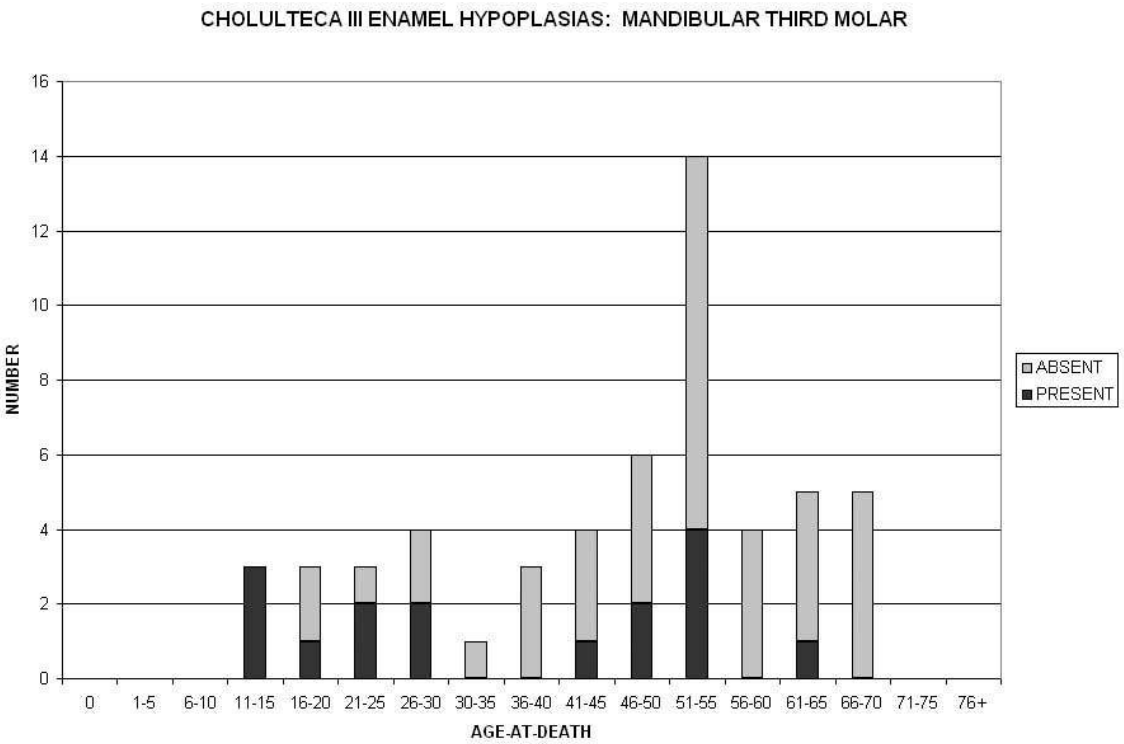

Table 31. Cholulteca III enamel hypoplasias: mandibular third molar.

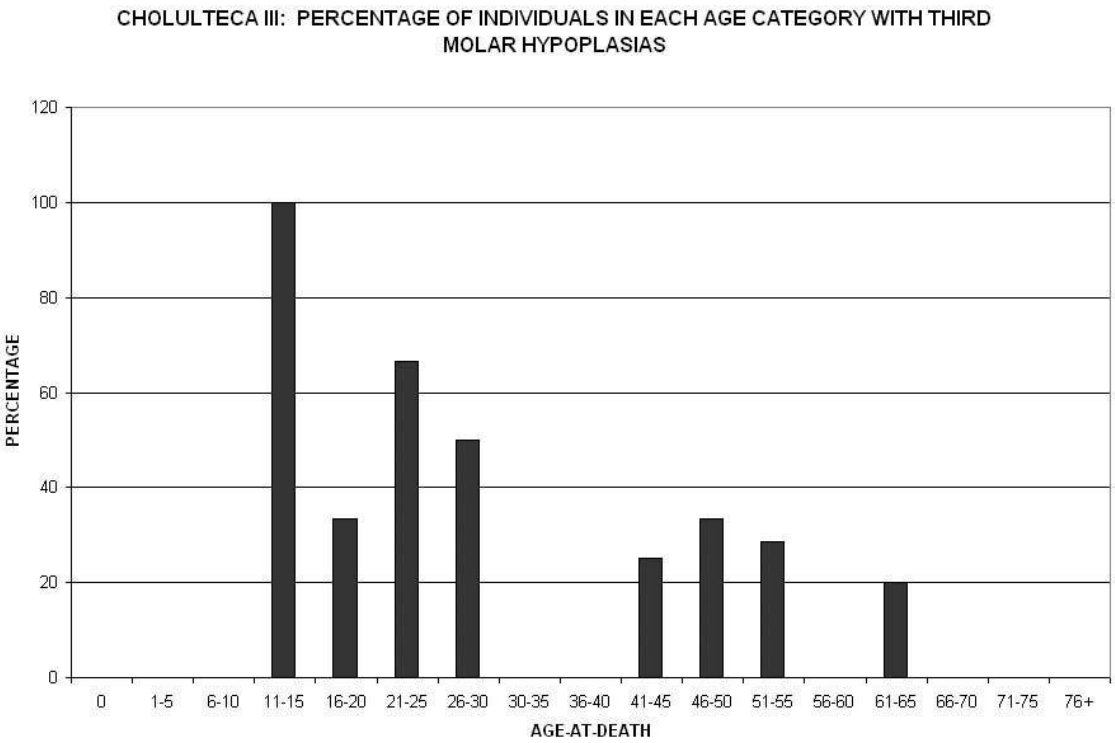

Table 32. Cholulteca III: Percentage of individuals in each age category with third molar hypoplasias.

Conclusions

The data presented in this report are the preliminary results of research involving the Postclassic population of Cholula, and the conclusions drawn are very much tentative and may be altered based on future analyses. Research into the effects of urbanism on this population that will verify these interpretations and provide additional information is still being completed. Based on the initial data, however, it appears that the demography of the New World urban center of Cholula may vary from that of Old World preindustrial centers in regards to the mortality of migrants. Immigrants to New World urban centers may have had different reasons for migrating to the city than their Old World counterparts and may have experienced distinct conditions once they arrived. In addition, they clearly faced a very different epidemiological situation in the urban environment than Old World immigrants. Consequently, New World cities potentially had demographic profiles distinct from those of European preindustrial cities. The completed analysis of the effects of urbanism on the health and demography of the Cholula population will be disseminated as a doctoral dissertation and in future publications.

List of Figures

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