

CHAPTER  
**19** DEMOGRAPHY, HEALTH & REGIONAL BIODISTANCE

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## INTRODUCTION

The remains of 480 individuals were recovered from CCO-18/548. The size and age of the skeletal sample make this a very important site in California prehistory. Extensive population samples from the Early Period of California (9000-1000 BC, Walker 2006) are rare. The paucity of sites from this time period is especially true for the Central Valley. Cultural deposits associated with the early Middle Archaic of the Central Valley (circa 5550-2050 BC) are rare due in part to the dynamic alluvial processes which occurred in this area during this time period (Rosenthal et al. 2007). In other words, sites of this time period in this region are generally either obliterated or buried by ongoing alluvial activity. The preliminary dates also place portions of the site occupation at the intersection of three important cultural patterns: the Windmill (2500-1000 BC), Berkeley (1500 BC – AD 500), and the Meganos Aspect of the Berkeley Pattern (~500-200 BC). This is the largest population sample of this completeness for this crucial period in central California prehistory. This report examines three principle aspects of the skeletal sample from CCO-18/548: (1) the demographic profile of the sample, (2) the general health status of the population, and (3) the population affinity of the sample in a local geographic perspective. In order to provide context for interpretation, the information from the above aspects is compared to similar data collected for other California sites (Table 82).

## DEMOGRAPHIC ANALYSIS

Over the past 40 years there has been a continued interest in what Brothwell (1981) referred to as the “vital statistics of past populations”. The demographic profile for a site or region has the potential of revealing a number of diagnostic features for a population. Namely, sex and age ratios give vital clues in regard to population growth and decline, composition of communities, and the distribution of populations in space and time. It is expected that (1) life expectancy will vary from group to group, (2) males and females will vary in life expectancy and representation from site to site, and (3) diet, disease, culture, and environment will impact overall demographic profiles. While this vital information can be inferred from demographic data, Boddington (1987) points out that demographic reconstruction must take into account post-depositional disturbance and decay, differential burial patterns and damage or loss due to excavation. Demographic profiles in central California can be highly variable with disparate sex ratios and differential representation of age categories. The results from the analyses described below will be compared to other temporally and geographically close samples.

### Sex Assessment

Sexual dimorphism in the human cranium is primarily due to the action of testosterone at puberty (St. Hoyme and İşcan 1989). At birth there is little if any difference between the male and female cranium (St. Hoyme and İşcan 1989). At puberty, differences begin to manifest in the male cranium. Muscle attachments become larger and more pronounced and the overall thickness and size of the cranium increases (Krogman and İşcan 1986). These changes are the result of increased levels of testosterone. Females generally do not undergo such changes and thus the architecture of the female cranium is often referred to as pedomorphic because it retains many juvenile characteristics (St. Hoyme and İşcan 1989).

TABLE 82. Comparative Sites Included in Study.

| SITE     | N   | DATE <sup>1</sup> | ASSOCIATION            | REFERENCE                                    |
|----------|-----|-------------------|------------------------|--|
| SJO-68   | 34  | 2393 BC - 1030 BC | Middle Archaic         | Lillard et al. 1939; Ragir 1972; Heizer 1949 |
| SRI-41   | 79  | 2000-1000 BC      | Middle Archaic         | Orr 1968; Walker and Erlandson 1986          |
| BUT-233  | 34  | 2300 BC – AD 1300 | Middle/Upper Archaic   | Weldon 1990; Donahue 1993                    |
| BUT-294  | 37  | 2000 BC – AD 1450 | Middle/Upper Archaic   | Donahue 1993                                 |
| SAC-66   | 42  | ~2000 BC – AD 500 | Middle/Upper Archaic   | Lillard et al. 1939; Suchey 1975             |
| SAC-127R | 30  | ~2000 BC – AD 500 | Middle/Upper Archaic   | Grady 1967; Suchey 1975                      |
| SAC-151  | 32  | ~2000 BC – AD 500 | Middle/Upper Archaic   | Suchey 1975                                  |
| SJO-91   | 21  | 1035 BC - AD 630  | Middle/Upper Archaic   | Suchey 1975                                  |
| ALA-328  | 39  | 386 BC ± 150      | Upper Archaic          | Suchey 1975                                  |
| ALA-329  | 52  | AD 300 ± 85       | Upper Archaic          | Suchey 1975                                  |
| BUT-496  | 68  | AD 1-500          | Upper Archaic          | Donahue 1993                                 |
| CCO-139  | 26  | ~500 BC – AD 500  | Upper Archaic          | Suchey 1975                                  |
| CCO-696  | 154 | 765 BC – AD 600   | Upper Archaic          | Meyer and Rosenthal 1997                     |
| SAC-29   | 25  | AD 200 ± 500      | Upper Archaic          | Hawkes 1965; Suchey 1975                     |
| SAC-60   | 24  | ~ AD 0 - 1000     | Upper Archaic          | Lillard et al. 1939; Suchey 1975             |
| SAC-99   | 20  | ~AD 1000          | Upper Archaic          | Lillard et al. 1939; Suchey 1975             |
| SCRI-83  | 28  | ~ 500 BC – AD 500 | Upper Archaic          | Suchey 1975                                  |
| SJO-17   | 35  | ~500 BC           | Upper Archaic          | Molnar 1971                                  |
| SJO-154  | 60  | ~AD 500           | Upper Archaic          | Hoffman 1987                                 |
| SOL-2C   | 33  | ~ 500 BC – AD 500 | Upper Archaic          | Suchey 1975                                  |
| SRI- 2A  | 14  | ~AD 180           | Upper Archaic          | Orr 1968; Walker and Erlandson 1986          |
| YOL-13   | 32  | AD 1085 - 1135    | Upper Archaic          | Suchey 1975                                  |
| CCO-138  | 135 | AD 721 - 1450     | Upper Archaic/Emergent | Suchey 1975                                  |
| SAC-43   | 38  | ~ AD 0- 1500      | Upper Archaic/Emergent | Suchey 1975                                  |
| SCL-38   | 252 | AD 500-1769       | Upper Archaic/Emergent | Leventhal et al. 1993; Morley 1997           |
| SCL-294  | 94  | AD 900 - 1700     | Upper Archaic/Emergent | Clewlow et al. 1980; Richards 1988           |
| SOL-2L   | 20  | ~AD 500-1500      | Upper Archaic/Emergent | Suchey 1975                                  |
| COL-1    | 20  | ~AD 1500          | Emergent               | Lillard et al. 1939; Suchey 1975             |
| SRI-2B   | 65  | AD 1100-1500      | Emergent               | Orr 1968; Walker and Erlandson 1986          |
| TEH-10   | 178 | AD 1500-1700      | Emergent               | Dickel 1980                                  |

<sup>1</sup>Dates for burials after Rosenthal et al. (2007)

In the postcranial skeleton, because one of the manifestations of sexual dimorphism in humans is size, elements of the male skeleton tend to be larger than those of females (Bass 1995). There is, however, considerable overlap in the size ranges of males and females. Two exceptions are the humeral and femoral heads which tend to be highly sexually dimorphic (Stewart 1979), making these structures quite useful indicators of sex (Bass 1995).

There is considerable variation between individuals, families, and populations in the expression of sexual dimorphism. Some populations are noted for generally gracile males, while others are characterized by quite robust females (Krogman and İşcan 1986). There is no one single trait that is absolutely diagnostic as male or female. Given these considerations, researchers should evaluate as many traits as possible when trying to ascertain the sex of an individual to obtain the most accurate assessment.

A variety of methods were used to ascertain sex for this study. The primary methods included general observation of relative robusticity (Bass 1995; White and Folkens 1991), nonmetric sex characteristics (Acsádi and Nemeskéri 1970; Buikstra and Ubelaker 1994; Wolfe et al. 1994), humeral and femoral head diameter (Ubelaker 1989), and the Phenice method for visual assessment of the pubis (Phenice 1969). The methods are separated into cranial and postcranial categories. Observations of relative robusticity (Bass 1981; White and Folkens 1991) and nonmetric sex characteristics (Acsádi and Nemeskéri 1970; Buikstra and Ubelaker 1994; Wolfe et al. 1994) were used to assess sex for the cranium. Humeral and femoral head diameter (Ubelaker 1989), the Phenice method for visual assessment of the pubis (Phenice 1969), and graded nonmetric variables of the innominate (Buikstra and Ubelaker 1994) were used for postcranial sex assessment.

Aside from the general size of the cranium, more specific characteristics, such as the shape of the chin, angle of the ascending ramus and roundness of the orbital margins were compared to estimate sex (Wolfe et al. 1994). The cranial traits used for sex assessment of the crania avoid characteristics that derive from functional anatomy and are therefore only minimally influenced by environmental factors. Combining postcranial methods with cranial methods provided a composite estimate of the sex of an individual.

The Phenice method (Phenice 1969) for sex estimation of the innominate is a widely used sex assessment method for archaeological populations (White and Folkens 1991). This method is by far the easiest and most reliable of the methods for assessment of the pelvis because it focuses on key elements of pubic morphology that are highly sexually dimorphic. The Phenice method is ideal for sex assessment of prehistoric populations because (1) even in highly fragmentary remains, the pubis is usually preserved, (2) the elements of morphology are relatively simple, and (3) even in ambiguous cases the pubis tends to exhibit clear sexual dimorphism (Phenice 1969). This method focuses on the visual assessment of the ventral arc, subpubic concavity, and the ischiopubic ramus ridge.

In addition to the Phenice method, Buikstra and Ubelaker (1994) provide sex assessment methods using a graded system utilizing morphology of the innominate based on standards developed by Acsádi and Nemeskéri (1970). The specific aspects of the pelvis include the greater sciatic notch and the preauricular sulcus. The shape of the greater sciatic notch is one of the focal points of this method. The greater sciatic notch tends to be broad in females and narrow in males (Buikstra and Ubelaker 1994). In the Buikstra and Ubelaker system, Stage 1 begins with a broad sciatic notch and progresses with the notch narrowing to male-like morphology at Stage 5 (Buikstra and Ubelaker 1994).

Similar to the graded system for the sciatic notch, Buikstra and Ubelaker (1994) describe a system for the preauricular surface. The method is based on the finding that the preauricular surface appears more commonly in females than in males. Buikstra and Ubelaker describe four possible expressions in the preauricular surface. The preauricular surface is more feminine at Stage 1 and more masculine closer to Stage 4 (Buikstra and Ubelaker 1994). The female expression in preauricular morphology reflects the

effect of parturition on the pelvis. Since the morphology of the greater sciatic notch and preauricular surface are variable, the methods are more reliable when used in conjunction with other sex assessment methods.

Aside from the nonmetric methods for visual sex assessment of the pelvis, metric methods were also utilized for the postcranial skeleton. Stewart (1979) developed a method of metric sex assessment using the vertical (maximum) diameter of the head of the humerus. Using the Terry collection, Stewart (1979) concluded that diameters of less than 43 mm are female, diameters greater than 47 mm are male, and diameters between 44 mm and 46 mm are indeterminate. The femoral head is similarly sexually dimorphic. In general, diameters less than 42.5 mm are female, diameters greater than 47.5 mm are male, and diameters between 43.5 mm and 46.5 mm are indeterminate (Bass 1995).

### **Age-at-Death Assessment**

A variety of methods were employed to estimate age-at-death for this skeletal sample. The different methods concentrated on different parts of the skeleton that undergo separate metamorphoses guided by differing developmental processes. This is an important consideration because the various methods should yield similar results for the same individual despite the differing developmental pathways. The methods used to estimate age-at-death concentrate on five primary areas: (1) subadult cranial development, (2) cranial suture closure, (3) dental calcification and eruption, (4) postcranial epiphyseal closure, and (5) the metamorphosis of three postcranial cartilaginous joints. The comparison of estimates derived from each of these methods allows one to arrive at a composite estimate based on the relative reliability of each method.

Ironically, although the human cranium offers the best evidence for racial affiliation, age-at-death estimates for the cranium are not particularly *precise*. They are however *accurate* within a decade time range. As with the estimation of all demographic parameters, the more methods that can be used, the more accurate the age-at-death estimate. Three separate methods were used to estimate the age-at-death for the subadult crania: (1) development of the temporal bone (Weaver 1979), (2) development of the occipital bone (Redfield 1970), and dental calcification and eruption (Ubelaker 1989). Four separate methods were used to estimate age-at-death for the adult crania: (1) general age related changes of the cranium (Krogman and İşcan 1986), (2) vault and lateral anterior ectocranial suture closure (Buikstra and Ubelaker 1994), (3) regression analysis of cranial suture closure (Nawrocki 1998), and (4) dental attrition (Smith 1984).

The cranium undergoes a number of predictable changes during development. The predictable timing of these events provides some reliable methods by which to estimate age-at-death in an individual. Developmentally, changes in the cranium can be divided into two different stages: (1) the period of active growth or the subadult period and (2) the period after active growth or the adult period. The subadult period is marked by many dramatic changes in the characteristics of the human cranium. There are numerous methods that have been described for predicting the age-at-death of subadult individuals. The two most accurate methods document the chronological changes in the occipital and temporal bones of the cranium. Redfield (1970) provides a means of estimating the age of juveniles from the shape and union of the parts of the occipital bone that are separate at birth. The five distinct parts of the occipital bone are formed by the third fetal month and progressively unite culminating with the fusion of the occipital to the sphenoid (*synchondrosis spheno-occipitalis*) by the mid-twenties. Weaver (1979) devised a system of estimating subadult age from the chronological metamorphosis of the temporal bone. The system divides development of the temporal into six stages and provides the most accurate ages for individuals between birth and 2.5 years of age.

There are two useful methods for estimating age at death for adult crania. One produces general age category estimates and the other provides more specific age ranges. The first method is based on the

chronological change of general features of the cranium. Several researchers (Krogman and İşcan 1986) have noted a series of changes in the adult cranium which are for the most part subjective. They have not been standardized either by definition or by statistical analysis but they do offer a beginning point for assessing adult age-at-death. These subjective features involve observations of the texture of the ectocranial surface, the definition of muscle markings, depth and frequency of arachnoid foveae, and the depth of the middle meningeal grooves. In addition to these observations, careful observation and scoring of the degree of suture closure can give fairly precise estimations of age-at-death. Numerous studies have documented the range of ages for cranial suture closure. Buikstra and Ubelaker (1994) recommend recording the degree of suture closure for 10 ectocranial, 4 palatal, and 3 endocranial locations. Summing the scores for each location yields an age-at-death estimate within a 10-year range. Nawrocki (1998) has refined the method of Buikstra and Ubelaker (1994) by the application of regression equations to the maximum number of cranial sutures available in an individual cranium. The results for this method can be quite accurate.

Because the dentition is less affected than other skeletal tissues by malnutrition, endocrinopathies, and other disturbances (Smith 1991, Pirinen 1995), the most reliable method for age estimation in children up to age 14 is dental calcification and eruption. The standards used here are from Ubelaker (1989). The second dental age-at-death estimator is through observation of dental attrition. Age-at-death estimates based on dental attrition are fairly reliable within a population, after the rate of dental attrition has been calculated for that population. Generally, attrition in a population is calibrated using seriation. In other words, all of the individuals for a sample are placed in a sequence from least worn teeth to most worn and the rate of attrition is calculated based in part on the degree of attrition in the subadults. The dental attrition standards of Smith (1984) are used here. The Smith (1984) system for dental attrition is most accurately used when a dental seriation has been performed for the *population* (see Miles 1963); however a reasonable estimate can be obtained even in the absence of a seriation.

Similar to cranial and dental age-at-death methods, postcranial methods are divided into subadult and adult development phases. The two methods used for subadult postcranial remains are (1) diaphyseal long bone length (Ubelaker 1989) and (2) epiphyseal appearance and fusion (Krogman and İşcan 1986). The age-at-death estimates for adult postcranial remains are derived from observation of the metamorphosis of three major cartilaginous joints: (1) the pubic symphysis (Meindl et al. 1985), (2) the auricular surface of the innominate (Lovejoy et al. 1985a, 1985b), and (3) the sternal end of the rib (İşcan et al. 1984a, 1984b, 1985). According to Krogman and İşcan (1986), the diaphyseal length of long bones can be used to confidently estimate subadult age-at-death for individuals less than 12 years. This method is more accurate for younger ages (<9 years of age) when skeletal growth is appreciably more rapid. The standards used here are based on data from Ubelaker (1989). One of the predictable patterns of preadult development is the linear growth of long bones, which is facilitated by the separation of the long bones during growth into segments separated by growth plates. The ends of the long bones are referred to as epiphyses and it is the appearance and later fusion of these epiphyses to the shaft (diaphysis) of the long bone which allows one to calculate the age-at-death of subadult postcranial remains. Epiphyseal closure is one of the most accurate and straightforward methods for estimating the age-at-death of subadults. The standards developed by Krogman and İşcan (1986) are used here.

Estimation of age-at-death for the adult postcranium involves observation of the metamorphosis of three major cartilaginous joints: the pubic symphysis, the auricular surface of the innominate and the sternal end of the rib. The pubic symphysis undergoes a predictable series of changes as the cartilage gradually ossifies nearing the third decade and then progressively degenerates after the fourth decade. Todd (1920, 1921a, 1921b, 1921c) was the first researcher to formalize a system of age-at-death estimation from these changes in the pubic symphysis. Various revisions and refinements were subsequently suggested for Todd's system by a number of later researchers. Meindl et al. (1985) conducted a blind test of all the pubic symphysis age estimation methods. They concluded that by preserving the simplicity of the original Todd system and condensing Todd's 10 phases into "five major

biological phases" researchers could accurately estimate age-at-death. Similar to the pubic symphysis, the sacroiliac joint undergoes a systematic series of changes throughout life. These changes are reflected in the metamorphosis of morphology of the auricular surfaces of the sacrum and ilium. Lovejoy et al. (1985a) introduced a method of age-at-death estimation based on metamorphosis of the auricular surface of the ilium. The system is based on changes in topography, marginal lipping, and porosity. When mastered, this is an extremely reliable method for age-at-death estimation. The sternal end of the rib also undergoes a predictable metamorphosis throughout life. İşcan and coworkers (1984a, 1984b, 1985) developed a system of standards for age-at-death estimation from the sternal rib end. The system relies on systematic changes in pit shape, depth, rim configurations and overall condition of the bone. Chronological metamorphosis of the sternal rib end has been demonstrated to be an accurate age-at-death estimator with no significant variance in expression between sexes or populations (İşcan et al. 1984b, 1985; Russell et al. 1993; Yoder et al. 2001).

Each individual was evaluated with as many of the above methods as possible to obtain an age-at-death estimate. The most accurate methods were given the heaviest emphasis in the formation of a composite age-at-death estimate. The heaviest emphasis for the juvenile estimations was given to dental development and eruption followed by epiphyseal formation and union. The heaviest emphasis for adult estimations was given to auricular surface estimations, followed by pubic symphysis estimates. Using these estimates alone provided age-at-death estimations for only 234 individuals. An additional 122 individuals were assigned age-at-death estimations based on dental seriation. Because of the general paucity of juvenile individuals, a traditional seriation was not possible (cf., Miles 1969). Instead, individuals with both auricular surface estimations and at least half of the dentition present were seriated correlating auricular surface estimates with Smith (1984) attrition scores. The Smith attrition scores were then assigned five-year age brackets based on the auricular surface estimates. The results for sex and age-at-death estimates are reported in Table 83 and Figures 104 and 105. Examination of these results indicates an unusual distribution for this sample in regard to age cohort distribution. That is, the average age-at-death here is relatively old compared to other populations. More than 48% of the sample is older than 40 years of age. Another important demographic finding can be observed by comparing the data from CCO-18/548 with other large samples (Figures 106 and 107). A notable feature of the demographic data for the CCO-18/548 sample is the relative lack of individuals in the juvenile age cohorts (i.e., less than 18). The above observations may indicate an extraordinarily healthy population with few deaths in the younger age cohorts and the majority of adult individuals living to considerable old age. However, given the mortuary pattern seen at other precontact California Native American sites it may be more likely that the demographic profile observed here is the result of differential burial patterns. That is, the majority of younger individuals were simply buried elsewhere and the sample at CCO-18/548 represents a unique subset of the population rather than a representative cross-section.

TABLE 83. Age and Sex Distribution.

|        | 0-5 | 6-10 | 11-15 | 16-20 | 21-25 | 26-30 | 31-35 | 36-40 | 41-45 | 46-50 | 51-55 | 56-60 | TOTAL |
|--------|-----|------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|
| Female | 0   | 0    | 1     | 2     | 6     | 2     | 8     | 15    | 16    | 25    | 5     | 0     | 80    |
| Male   | 0   | 0    | 0     | 1     | 4     | 10    | 14    | 24    | 27    | 28    | 3     | 0     | 111   |
| Indet. | 12  | 11   | 6     | 5     | 8     | 14    | 14    | 14    | 24    | 26    | 9     | 1     | 144   |
| Total  | 12  | 11   | 7     | 8     | 18    | 26    | 36    | 53    | 67    | 79    | 17    | 1     | 335   |

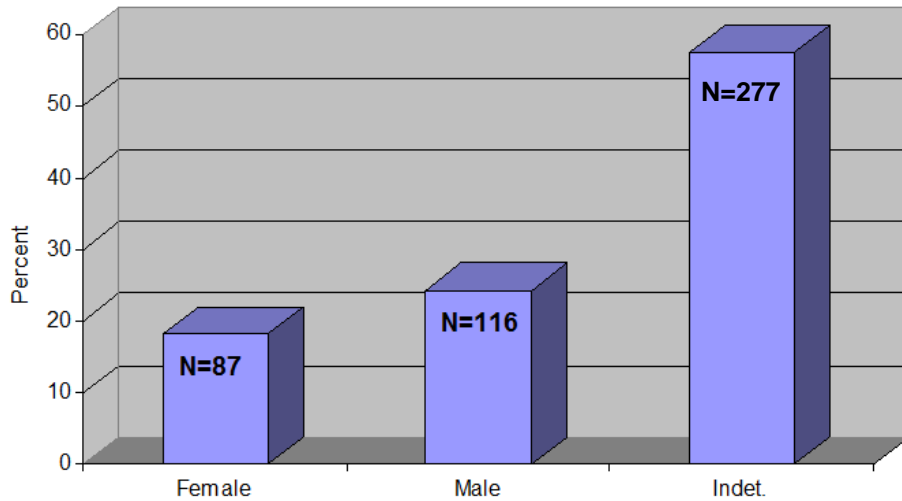


FIGURE 104. Sex Distribution<sup>10</sup>.

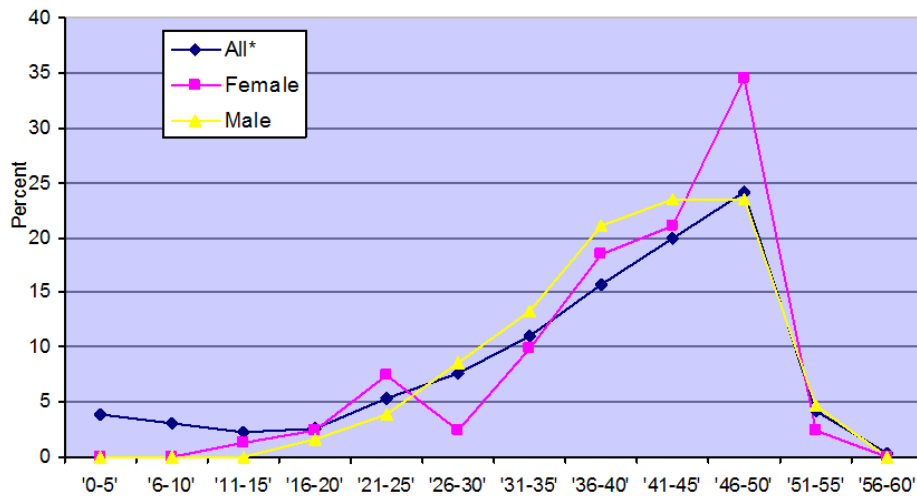


FIGURE 105. Mortality Curves for CA-CCO-18/548 (“All” includes female, male and indeterminate).

<sup>10</sup> Breakdown of female and male burials differs from totals presented in Figure 85 (Chapter 18) because five burials where age was recorded as questionable (four Male? and one Female?) were placed in the Indeterminate category in Figure 85 and included as Female or Male in this distribution table.

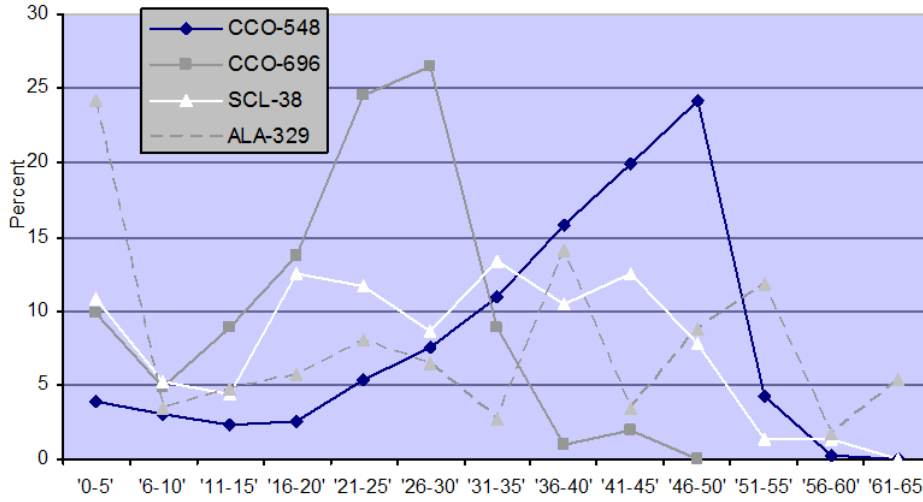


FIGURE 106. Mortality Curves for CA-CCO-18/548 and Three California Samples.

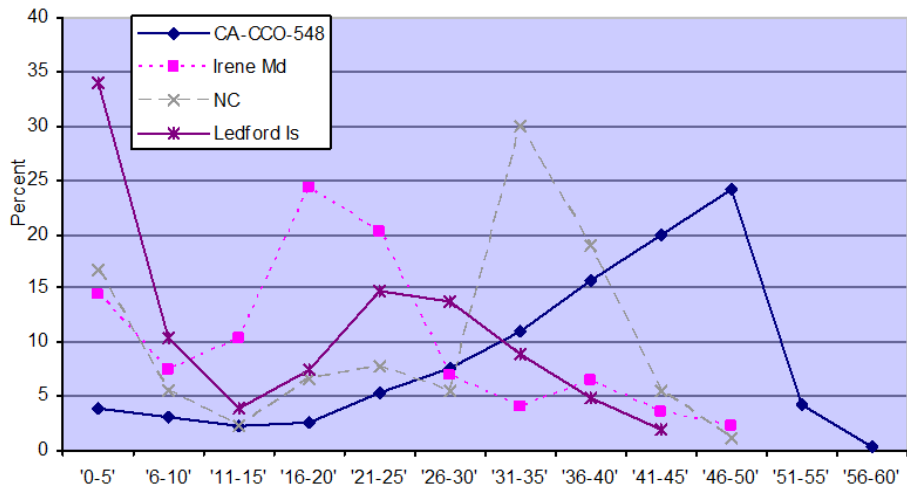


FIGURE 107. Mortality Curves for CA-CCO-18/548 and Three Comparative Population Samples.



## INDICATORS OF HEALTH STATUS

The importance of examining the occurrence and frequency of pathological conditions in skeletal populations is that it can offer insight regarding health status and lifeways of past populations. Bearing this in mind, any paleopathological analysis should evaluate the meaning of the presence of pathological conditions in a population rather than simply cataloguing “interesting” anomalies. In the assessment of the meaning of the presence and frequency of pathological conditions there are a number of important considerations that must be kept in mind. One important consideration is that only a limited number of disease processes leave their mark on the hard tissues. As Ortner (1992:5) points out “only about 15% of all skeletons in a typical archaeological sample from North America will show evidence of significant disease.” Most of these cases will fall into one of three general disease categories: (1) trauma, (2) chronic bacterial infection, and (3) arthritides (degenerative joint disorders). From this observation, one might be led to the erroneous conclusion that the most important pathological conditions in prehistory fall into these three categories. In reality, these three classifications of disease are the ones that leave their indelible mark on the hard tissues. Some of the most important diseases in human history leave no marks on the skeleton either because they are acute (i.e., they have a rapid onset and rapid resolution) or because they primarily affect the soft tissues. Most of the major epidemic diseases, which are predominantly viral rather than bacterial, fall into this category. From the foregoing, it should be understood that most of the pathological conditions that are recorded in skeletal populations record diseases that individuals are living with rather than dying from. Many pathological conditions then actually represent the successful response to disease rather than an indication of poor health. On the other hand, many of these conditions certainly contribute to morbidity although they may not be the proximate cause of death.

There are a number of informative pathological conditions present in this population sample. Most do offer some insight into the general health characteristics of the population; many provide information about activity patterns in this sample; and some provide information about habitual diet. The pathological conditions are discussed below in two organizational regions of the body: masticatory apparatus of the cranium and postcranial. The results from this part of the analysis will be compared to data from other temporally and geographically close samples.

### Dental Pathology

The dentition can often provide an accurate gauge of the overall health and diet of a population. Anthropological investigations of oral health span the history of the discipline and have been important for investigating differing subsistence strategies between and among human populations (Rose et al. 1991). The relationship between subsistence strategy and oral health is complex; however the causal nature of the relationship is rather direct. The reasons for this are that the dentition is the first part of the digestive system and as such the oral cavity is an active entry point for invading organisms. Examination of the dental remains in this sample shows a number of important features: (1) all of the individuals exhibit extensive dental attrition, (2) the frequency of carious lesions is extraordinarily low, (3) considering the level of attrition and the age of the individuals the frequency of periodontal disease is relatively low, and (4) the frequency of enamel hypoplasias while low for the entire dentition is quite high for certain key teeth. Details of these dental features will be examined in relation to other nearby populations of similar antiquity.

#### *Dental Attrition*

Dental attrition is dependent on two major variables, diet and age of the individual. Populations that have a more rigorous diet (e.g., hunter-gatherer subsistence) will experience more rapid dental attrition than those that have a softer more highly processed diet (e.g., agricultural subsistence). Attrition is also age progressive. That is, the older an individual is the more attrition their teeth will exhibit. Not only does

subsistence regime affect the overall level of attrition, it also affects the specific wear pattern on the teeth. Because of the rigorous nature of the hunter-gatherer diet the wear pattern generally tends to be flat across the occlusal surface (Smith 1984, 1991). The softer texture of the agricultural diet tends to produce a “scooped out” appearance to the occlusal surface.

Dental attrition is dependent on two major variables, diet and age of the individual. In general, hunter-gatherers have a more rigorous diet and experience rapid dental attrition. In contrast, agricultural populations consume a softer more highly processed diet and experience much less attrition. Attrition is also age progressive. That is, the older an individual is the more attrition they will have on their teeth. Each tooth was graded for dental wear using the Smith system (Smith 1984). Because a considerable number of teeth exceeded Stage 8 in the system, a grade nine was added for this study. Stage nine was used when the entire crown of the tooth was worn away as well as a considerable portion of the root. In order to facilitate comparison with data from other studies, left and right antimeres were pooled while maintaining separate counts for each tooth classification in the maxillary and mandibular dentitions.

Examination of the average wear across the entire skeletal sample (Figure 108) indicates a population exhibiting considerable dental attrition. The average overall dental attrition for the sample is 6.1 with more than 30% of the sample exhibiting average wear scores of eight or above. Comparison of male and female average attrition scores (Figures 109 and 110) reveals very little difference in attrition between the two sexes. None of the differences are statistically significant. Comparison of average attrition scores for each tooth class of the upper and lower dentition for males and females from three central California precontact sites (Figure 111) reveals that overall the sample from CCO-18/548 has some of the highest attrition scores. All of the sites exhibit similar levels of attrition and only the maxillary dentitions of the individuals from SJO-17 showed significant differences between males and females.

Isotope data provided by Beasley et al. (2009) for CCO-18/548 may reveal one possible explanation for the high attrition rates at this site. The isotope ratios fall in between those of coastal sites and sites located more inland. These isotope ratios may suggest a reliance on non-marine fish as a dietary staple. Traditional methods of fish preparation would contribute a significant amount of abrasives to the diet. Another possibility for the high levels of attrition is revealed by examining the wear patterns of the articulated maxillary and mandibular dentitions. In the majority of intact dentitions from this site, the maxillary and mandibular teeth are not in direct occlusion. Griffin and coworkers (2009) hypothesize that much of this wear has to do with non-alimentary behavior such as basket-making or cordage manufacture. Regardless of the exact source of this wear, the fact that the teeth of the opposing dentitions cannot contact one another suggests that the wear is non-alimentary in nature.

### *Dental Caries*

Dental caries (from the Latin, rottenness) is a multifactorial, multibacterial disease of the dentition. It is characterized by the focal demineralization of the inorganic portion and destruction of the organic component of the teeth (Aufderheide and Rodríguez-Martin 1998). Dental caries has been unequivocally demonstrated to be an infectious and transmissible disease (Marsh 1995). The human oral cavity is inhabited by more than 500 indigenous species of microflora, most of which are commensal (Ruby and Goldner 2007). Bacterial populations attached to tooth surfaces consist of dense biofilm communities that may be among the most complex that exist in nature (Meurman et al. 2004). A biofilm is a three-dimensional structured micro-organism community with a complex web of fluid channels for transport of substrate, waste products, and signal molecules (Costerton 1999). Micro-organisms organized in a biofilm are more resistant to immune defense mechanisms (Scheie and Peterson 2004). The oral microflora is initially introduced through salivary transmission associated with intimate human contact (Carlsson et al.

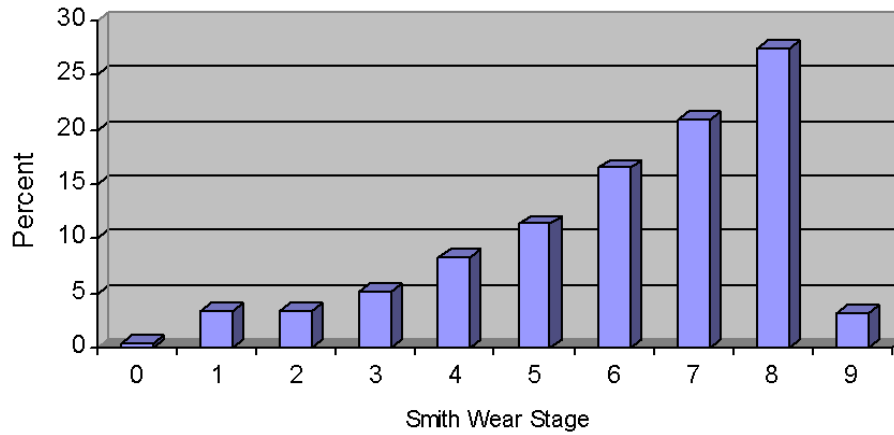


FIGURE 108. Frequencies of Smith (1984) Composite Dental Wear Scores for CA-CCO-18/548.

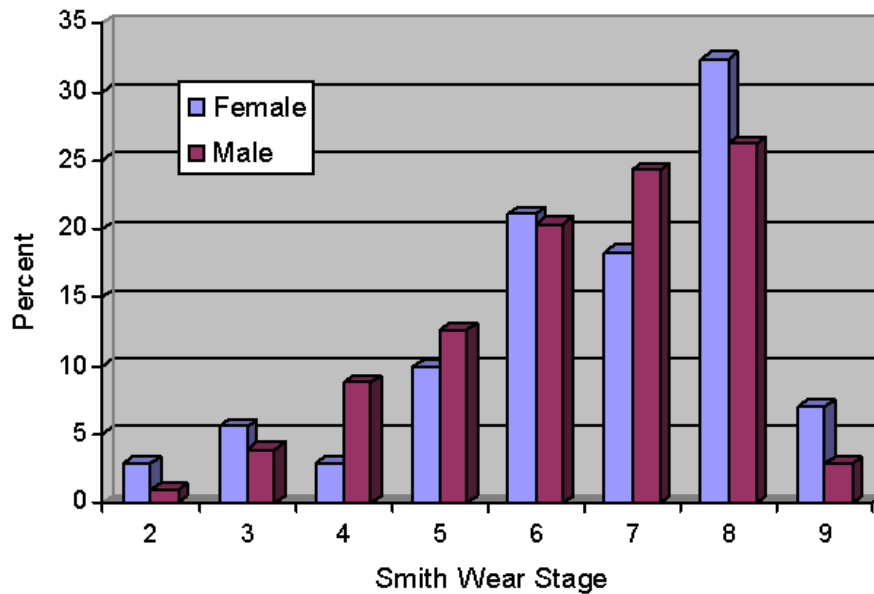


FIGURE 109. Frequencies of Smith (1984) Composite Dental Wear Scores for Males and Females at CA-CCO-18/548.

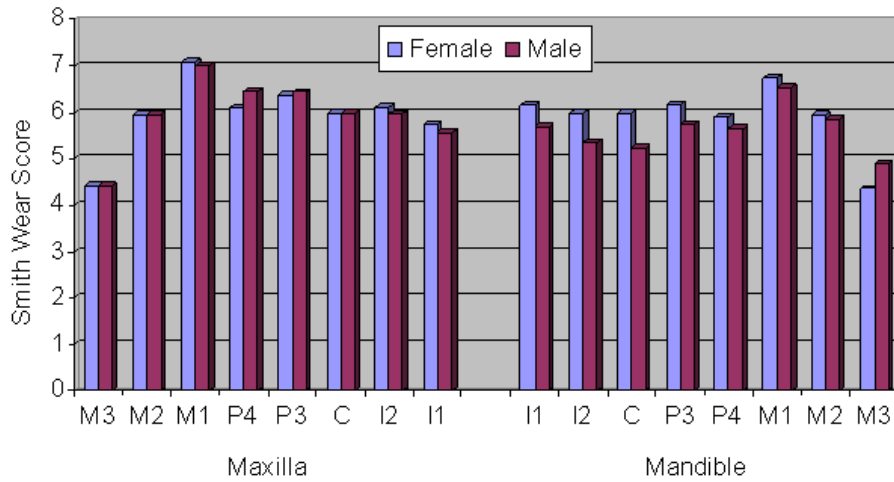


FIGURE 110. Mean Dental Wear by Tooth Class and Sex.

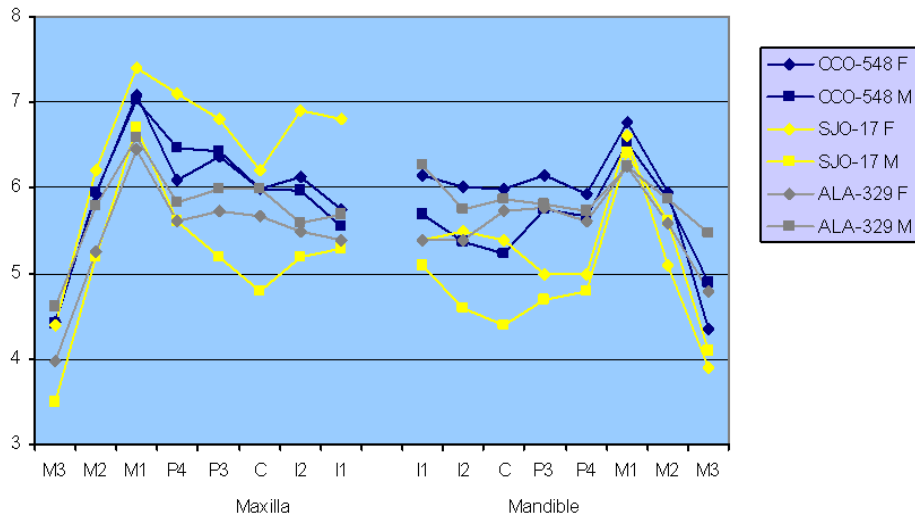


FIGURE 111. Mean Dental Wear by Tooth Class and Sex for CA-CCO-18/548 Compared to Two Other Sites, CA-SJO-17 and CA-ALA-329 (Comparative Data from Molnar 1971 and Jurmain 1990).

1975). Occasionally, these normally symbiotic protean entities can become parasitic and initiate pathological conditions. In the normal individual there is a delicate balance between commensal and pathogenic microflora of the oral cavity. The homeostasis of the indigenous oral microflora can be disrupted by reductions in pH which encourages the overgrowth of pathogenic species (Marsh 1994). Active disease arises from three interrelated factors: (1) host susceptibility mediated by genetic and non-genetic influences, (2) the presence of pathogenic organisms, and (3) the relative absence of beneficial species (Hassell and Harris 1995; Smalley 1994; Livingstone et al. 2002).

Caries is the direct result of two interrelated events: (1) disturbance of the balance of commensal and pathogenic microflora in the oral cavity and (2) frequent consumption of fermentable carbohydrates (Marsh 1995; Lingström et al. 1994). Two primary bacteria (*Streptococcus mutans* and *Streptococcus sobrinus*) consume carbohydrates in the oral cavity and produce metabolic wastes which contribute organic acids and proteases to the oral fluids (Liljemark and Bloomquist 1996). In sufficient concentration, the metabolic products can dissolve the mineral component of dental enamel. Left unchecked carious lesions can completely destroy a tooth and expose the circulatory system to infection (bacteremia and septicemia). Dental caries may also be implicated in the development of cardiovascular disease (Meurman et al. 2004).

Dental caries epidemiology is one of the most important ways in which the diet of past populations can be reconstructed (Hillson 2001). It is one of the few conditions which have been recorded in almost all reports on human remains from archaeological sites (Rose et al. 1991). Intersite comparisons are however hampered because preservation and degree of dental wear are highly variable from site to site. This makes standardized data collection methods for dental caries problematic. Intersite comparisons are further complicated because the methods used for diagnosis and recording dental status generally have lacked consistency, and also have often not provided enough detail to allow proper interpretation (Hillson 2001). Many researchers have moved in the direction of standardizing the data collection methods (Buikstra and Ubelaker 1994), but caries epidemiology is difficult enough even amongst the living and there is a need for continued debate on methods for recording and quantifying caries in archaeology (Hillson 2001).

Both living and archaeological populations have provided information regarding common caries patterns among humans. In all populations, there is an age-related trend line showing a progressive rise in the average number of lesions per person in successive age cohorts from childhood to adolescence and early adulthood (Hillson 2001). Research has also shown that left and right sides are equally affected by coronal caries, and upper cheek teeth about the same as lower cheek teeth, but upper anterior tooth crowns are more frequently involved than lower (Hillson 2001). Different classes of teeth have also been shown to be more susceptible than other teeth in the dentition. Cheek teeth are far more susceptible than anterior teeth, and the occlusal fissures of first molars (the earliest erupting permanent teeth) are generally the teeth affected in the lowest caries rate populations (Hillson 2001). The occlusal sites of the second molars present an even higher caries rate than seen with the first molars. Typically the upper dentition is the first to present carious lesions, followed by the lower dentition. This hierarchy appears to remain the same, regardless of fluorine in toothpaste and water, and level of dental care (Hillson 2001). There is also an inverse relationship between average level of dental attrition and frequency of carious lesions in populations. In other words, populations with high levels of attrition tend to have low frequencies of carious lesions (Hardwick 1960; Moore and Corbet 1975; Burt 1981; Maat and Van der Velde 1987). The operative mechanism in this relationship may be the cleansing effect of abrasive food. It may also be that abrasive food is so coarse that the debris cannot enter the fissures or stick to the teeth (Maat and Van der Velde 1987).

Studies have shown that not only is there a difference in caries rates between tooth class, but also differences in rates between men and women. As is seen in most archaeological studies, women tended to have more teeth affected than men, with a more strongly marked progression with age, although the

difference was not great, especially in the younger age groups (Hillson 2001). While sex differences in caries rates follow a relatively recognizable pattern, other conditions associated with dental caries do not follow a distinguishable pattern. Tooth loss due to periodontal disease is highly variable between differing populations. Studies of children and adults in Kenya (Manji et al. 1988) and West Africa (Matthesen et al. 1990) show that the proportion of teeth with lesions penetrating to the pulp chamber rose gradually with age, as did the proportion of teeth missing because of caries. In contrast, studies of Inuit populations (Hilming and Pedersen 1940) show that in no cases had teeth been lost as a direct consequence of caries, and only rarely did tooth loss appear related to pulp exposure by attrition or alveolar bone loss resulting from periodontal disease (Hillson 2001).

Dental caries can be an accurate indicator of dental health and diet in past populations and has been used extensively to document changes in lifeways (Larsen 1982; Shavit 1988; Dahlberg 1960; Carbonell 1966; Milner 1984; Larsen et al. 1991). Milner (1984) in a survey of literature concerning frequency of carious lesions in prehistoric eastern native North Americans concluded that there is a substantial increase in the frequency of lesions after the adoption of agriculture. This is in part the consequence of the increased consumption of carbohydrates (maize in many parts of the New World) which when converted to sugar by saliva provides ideal conditions for the microflora which precipitate dental caries. Larsen (1980, 1982, 1983a) has also documented a dramatic increase in the frequency of carious lesions with the transition from a hunting and gathering economy to a subsistence based primarily on agriculture on the Georgia coast. Preagricultural populations in this study exhibited only 1.3% of the teeth affected by carious lesions while 11.6% of the agricultural teeth were affected by caries. In the agricultural group, males exhibited a lower incidence of carious lesions than did females. This may suggest differential consumption of maize. Larsen (1987) concludes that there is near unanimity from other regions of the world concerning the positive relationship between a shift from a hunter-gatherer mode of subsistence to an agricultural one and a relative increase in the frequency of carious lesions. This increase is likely related to the increased consumption of starchy foodstuffs with the shift to agricultural production.

Dental caries has been used to document dietary changes in precontact California Native American populations (Walker and Erlandson 1986; Schulz 1977; Bartelink 2006). While maize agriculture was never adopted in California as it was in other parts of the precontact U.S., highly processed acorn meal did provide a carbohydrate-rich diet for many California groups beginning in the Middle Archaic. Some authors (Schulz 1977; Bartelink 2006) have suggested that acorns processed into a sticky gruel may have had significant cariogenic properties.

In order to examine frequencies of carious lesions in this population, each tooth was examined with 3.5x dental loupes and stainless steel dental probes. Carious lesions were only counted with the presence of a definable pit. Tooth discolorations alone were not counted as carious lesions. Lesions were recorded with regard to their location on the tooth (occlusal, cervical, buccal, lingual, mesial, and distal) and their size (measured in millimeters). For the purposes of this study, carious lesions are reported simply as present without regard to number on a particular tooth, size, or location on the tooth. In order to facilitate comparison with other studies, left and right antimeres were pooled while maintaining separate counts for each tooth classification in the maxillary and mandibular dentitions. Frequencies of carious lesions in the CCO-18/548 sample and comparative samples are presented in Tables 84 and 85, and Figures 112-114.

Examination of the frequencies of carious lesions in the teeth from CCO-18/548 reveals that the overall frequencies are quite low (2.5% for the maxillary dentition and 2.5% for the mandibular dentition). Compared to the other sites reported in Tables 84 and 85 these frequencies are extraordinarily low. In fact, they are less than half of the lowest frequencies reported. These frequencies are even more remarkable considering only the molar teeth. Molars usually have the highest frequencies of carious lesions due to their complex fissure patterns (Klein and Palmer 1941; König 1963; Grainger et al. 1966). Similar to the overall comparison, the frequencies of carious lesions in the molar teeth for CCO-18/548 are the lowest of all the comparison sites. In most of the comparisons the frequencies of lesions for the

molars of CCO-18/548 are less than half of that found at other sites and in some of the comparisons are less than one quarter. Comparison of carious lesion frequencies between males and females reveals that as in most populations, females exhibit higher frequencies for most teeth. Despite the obvious differences in frequencies between the sexes, only two teeth (lower M2 and M3) have differences that are statistically significant at  $P < 0.05$ .

Frequencies of carious lesions at CCO-18/548 record a population with remarkably low levels of caries. Previous studies have linked the consumption of high carbohydrate diets with increased frequency of carious lesions. If this conclusion is correct, the interpretation here is that the population at CCO-18/548 was participating in a dietary regime with very low levels of carbohydrates. This is especially notable considering that later California populations exhibit considerably higher frequencies of carious lesions.

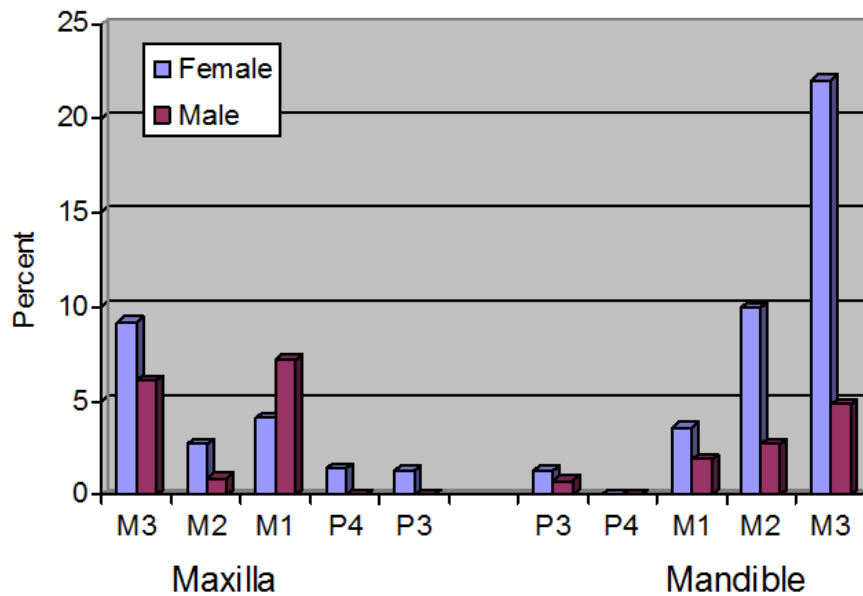


FIGURE 112. Frequencies of Carious Lesions in Maxillary and Mandibular Dentitions of Females and Males.

### *Caries-Attrition Competition*

Researchers have proposed two opposing relationships between caries and level of dental attrition. Some have suggested a synergistic relationship whereby increased attrition exposes the softer dentine to cariogenic bacteria and predisposes individuals to higher frequencies of carious lesions (Hardwick 1960; Miles 1969; Silverstone et al. 1981). Other researchers however have proposed an inverse relationship with high attrition rates and low carious lesion frequencies (Moore and Corbett 1975; Burt 1981). Newman (1999) points out that occlusal wear reduces and eventually eliminates fissures on the posterior dentition. These fissures are the primary repository for the plaque which in turn is one of the essential components of caries.

TABLE 84. Carious Lesion Frequencies in Central California Sites.

| TOOTH      | CCO-18/548 |      | SJO-17 <sup>1</sup> |      | SJO-154 <sup>2</sup> |      | SRI-41 <sup>3</sup> |      | SRI-2A <sup>3</sup> |      | SRI-2B <sup>3</sup> |      |
|------------|------------|------|---------------------|------|----------------------|------|---------------------|------|---------------------|------|---------------------|------|
|            | N          | %    | N                   | %    | N                    | %    | N                   | %    | N                   | %    | N                   | %    |
| Maxilla    |            |      |                     |      |                      |      |                     |      |                     |      |                     |      |
| I1         | 153        | 0.6  | 36                  | 2.8  | 26                   | 7.7  | 82                  | 1.2  | 16                  | 0.0  | 36                  | 0.0  |
| I2         | 190        | 0    | 35                  | 2.8  | 32                   | 3.1  | 107                 | 6.5  | 20                  | 0.0  | 54                  | 0.0  |
| C          | 236        | 0.8  | 52                  | 7.7  | 35                   | 14.3 | 124                 | 16.9 | 22                  | 0.0  | 77                  | 2.6  |
| P3         | 248        | 0.4  | 59                  | 7.7  | 32                   | 9.4  | 111                 | 16.2 | 16                  | 0.0  | 73                  | 0.0  |
| P4         | 237        | 0.4  | 71                  | 1.4  | 32                   | 12.5 | 108                 | 6.5  | 19                  | 5.3  | 70                  | 11.4 |
| M1         | 253        | 6.7  | 59                  | 23.7 | 29                   | 17.2 | 103                 | 28.2 | 17                  | 35.3 | 93                  | 8.6  |
| M2         | 263        | 2.3  | 62                  | 14.5 | 24                   | 8.3  | 101                 | 29.7 | 19                  | 31.6 | 85                  | 17.6 |
| M3         | 202        | 7.9  | 43                  | 14.0 | 21                   | 33.3 | 70                  | 34.3 | 14                  | 28.6 | 43                  | 18.6 |
| Total      | 1782       | 2.5  | 417                 | 9.3  | 231                  | 12.5 | 806                 | 17.0 | 143                 | 11.9 | 531                 | 7.7  |
| Mandible   |            |      |                     |      |                      |      |                     |      |                     |      |                     |      |
| I1         | 186        | 0.5  | 33                  | 0.0  | 30                   | 0.5  | 105                 | 0.0  | 8                   | 0.0  | 39                  | 0.0  |
| I2         | 231        | 0.4  | 34                  | 0.0  | 33                   | 0.4  | 126                 | 1.6  | 12                  | 0.0  | 46                  | 0.0  |
| C          | 258        | 0.0  | 38                  | 7.9  | 33                   | 0.0  | 129                 | 3.9  | 18                  | 5.6  | 52                  | 0.0  |
| P3         | 276        | 0.7  | 39                  | 2.6  | 39                   | 0.7  | 126                 | 7.9  | 15                  | 6.7  | 53                  | 0.0  |
| P4         | 296        | 0.0  | 40                  | 0.0  | 40                   | 0.0  | 120                 | 10.8 | 16                  | 0.0  | 52                  | 0.0  |
| M1         | 340        | 1.8  | 45                  | 4.4  | 35                   | 1.8  | 107                 | 22.4 | 14                  | 14.3 | 63                  | 12.7 |
| M2         | 339        | 5.3  | 41                  | 4.9  | 32                   | 5.3  | 118                 | 19.5 | 14                  | 21.4 | 53                  | 7.5  |
| M3         | 249        | 11.2 | 41                  | 17.1 | 33                   | 11.2 | 81                  | 17.3 | 11                  | 27.3 | 45                  | 13.3 |
| Total      | 2175       | 2.6  | 311                 | 4.8  | 275                  | 12.7 | 912                 | 10.0 | 108                 | 9.3  | 403                 | 4.5  |
| Site Total | 3957       | 2.5  | 728                 | 7.4  | 506                  | 12.6 | 1718                | 13.3 | 251                 | 10.8 | 934                 | 6.3  |

<sup>1</sup>Molnar 1971

<sup>2</sup>Hoffman 1987

<sup>3</sup>Walker and Erlandson 1986



TABLE 85. Carious Lesion Frequencies in Females and Males for Central California Sites.

| TOOTH      | CCO-18/548 |      |      |     |     |      | SJO-154 <sup>2</sup> |      |      |      |     |      | SRI-41 <sup>3</sup> |      |      |      |     |      | SRI-2A <sup>3</sup> |      |      |      |     |      | SRI-2B <sup>3</sup> |      |      |      |     |      |     |      |
|------------|------------|------|------|-----|-----|------|----------------------|------|------|------|-----|------|---------------------|------|------|------|-----|------|---------------------|------|------|------|-----|------|---------------------|------|------|------|-----|------|-----|------|
|            | FEMALE     |      | MALE |     | N % |      | FEMALE               |      | MALE |      | N % |      | FEMALE              |      | MALE |      | N % |      | FEMALE              |      | MALE |      | N % |      | FEMALE              |      | MALE |      | N % |      |     |      |
|            | N          | %    | N    | %   | N   | %    | N                    | %    | N    | %    | N   | %    | N                   | %    | N    | %    | N   | %    | N                   | %    | N    | %    | N   | %    | N                   | %    | N    | %    | N   | %    |     |      |
| Maxilla    |            |      |      |     |     |      |                      |      |      |      |     |      |                     |      |      |      |     |      |                     |      |      |      |     |      |                     |      |      |      |     |      |     |      |
| I1         | 40         | 2.5  | 69   | 0.0 | 14  | 0.0  | 22                   | 4.5  | 11   | 18.2 | 9   | 0    | 42                  | 2.4  | 40   | 0.0  | 11  | 0.0  | 5                   | 0.0  | 22   | 0.0  | 14  | 0.0  | 22                  | 0.0  | 14   | 0.0  | 22  | 0.0  | 14  | 0.0  |
| I2         | 60         | 0.0  | 87   | 0.0 | 13  | 7.7  | 22                   | 0.0  | 14   | 7.1  | 12  | 0    | 51                  | 9.8  | 56   | 3.6  | 12  | 0.0  | 8                   | 0.0  | 31   | 0.0  | 31  | 0.0  | 31                  | 0.0  | 23   | 0.0  | 31  | 0.0  | 23  | 0.0  |
| C          | 73         | 0.0  | 108  | 1.9 | 23  | 13.0 | 29                   | 3.4  | 17   | 23.5 | 15  | 6.7  | 59                  | 20.3 | 65   | 13.8 | 14  | 0.0  | 8                   | 0.0  | 44   | 0.0  | 44  | 0.0  | 44                  | 0.0  | 33   | 6.1  | 44  | 0.0  | 33  | 6.1  |
| P3         | 76         | 1.3  | 112  | 0.0 | 20  | 10.0 | 32                   | 9.4  | 17   | 11.8 | 12  | 8.3  | 55                  | 20.0 | 56   | 12.5 | 11  | 0.0  | 5                   | 0.0  | 37   | 0.0  | 37  | 0.0  | 37                  | 0.0  | 36   | 0.0  | 37  | 0.0  | 36  | 0.0  |
| P4         | 72         | 1.4  | 101  | 0.0 | 14  | 14.3 | 44                   | 2.3  | 15   | 20   | 13  | 7.7  | 54                  | 9.3  | 54   | 3.7  | 10  | 10.0 | 9                   | 0.0  | 38   | 15.8 | 38  | 15.8 | 38                  | 15.8 | 32   | 6.3  | 38  | 15.8 | 32  | 6.3  |
| M1         | 73         | 4.1  | 97   | 7.2 | 13  | 38.5 | 46                   | 19.6 | 14   | 21.4 | 14  | 14.3 | 52                  | 23.1 | 51   | 33.3 | 9   | 44.4 | 8                   | 25.0 | 51   | 7.8  | 51  | 7.8  | 43                  | 16.3 | 42   | 19.0 | 51  | 7.8  | 42  | 19.0 |
| M2         | 75         | 2.7  | 111  | 0.9 | 14  | 28.6 | 48                   | 10.4 | 12   | 16.7 | 12  | 0    | 48                  | 39.6 | 53   | 20.8 | 11  | 45.5 | 8                   | 12.5 | 43   | 16.3 | 43  | 16.3 | 24                  | 25.0 | 19   | 10.5 | 43  | 16.3 | 19  | 10.5 |
| M3         | 54         | 9.3  | 98   | 6.1 | 15  | 26.7 | 28                   | 7.1  | 9    | 44.4 | 12  | 25   | 28                  | 42.9 | 42   | 28.6 | 7   | 42.9 | 7                   | 14.3 | 24   | 25.0 | 24  | 25.0 | 19                  | 10.5 | 24   | 25.0 | 19  | 10.5 | 24  | 25.0 |
| Total      | 523        | 2.5  | 783  | 2.0 | 126 | 16.7 | 271                  | 8.1  | 109  | 19.3 | 99  | 8.0  | 389                 | 19.8 | 417  | 14.4 | 85  | 15.3 | 58                  | 6.9  | 290  | 7.9  | 290 | 7.9  | 241                 | 7.5  | 241  | 7.5  | 241 | 7.5  | 241 | 7.5  |
| Mandible   |            |      |      |     |     |      |                      |      |      |      |     |      |                     |      |      |      |     |      |                     |      |      |      |     |      |                     |      |      |      |     |      |     |      |
| I1         | 52         | 0.0  | 90   | 1.1 | 11  | 0.0  | 22                   | 0.0  | 13   | 0    | 11  | 0    | 45                  | 0.0  | 60   | 0.0  | 5   | 0.0  | 3                   | 0.0  | 27   | 0.0  | 27  | 0.0  | 27                  | 0.0  | 12   | 0.0  | 27  | 0.0  | 12  | 0.0  |
| I2         | 67         | 0.0  | 111  | 0.9 | 12  | 0.0  | 22                   | 0.0  | 15   | 0    | 14  | 14.3 | 54                  | 1.9  | 72   | 1.4  | 7   | 0.0  | 5                   | 0.0  | 32   | 0.0  | 32  | 0.0  | 32                  | 0.0  | 14   | 0.0  | 32  | 0.0  | 14  | 0.0  |
| C          | 68         | 0.0  | 120  | 0.0 | 12  | 0.0  | 26                   | 7.7  | 15   | 0    | 16  | 12.5 | 58                  | 5.2  | 71   | 2.8  | 11  | 9.1  | 7                   | 0.0  | 34   | 0.0  | 34  | 0.0  | 34                  | 0.0  | 18   | 0.0  | 34  | 0.0  | 18  | 0.0  |
| P3         | 77         | 1.3  | 123  | 0.8 | 12  | 8.3  | 27                   | 0.0  | 17   | 11.8 | 16  | 18.6 | 56                  | 7.1  | 70   | 8.6  | 10  | 10.0 | 5                   | 0.0  | 33   | 0.0  | 33  | 0.0  | 33                  | 0.0  | 20   | 0.0  | 33  | 0.0  | 20  | 0.0  |
| P4         | 82         | 0.0  | 137  | 0.0 | 13  | 0.0  | 27                   | 0.0  | 16   | 6.3  | 17  | 5.9  | 51                  | 15.7 | 69   | 7.2  | 11  | 0.0  | 5                   | 0.0  | 32   | 0.0  | 32  | 0.0  | 32                  | 0.0  | 20   | 0.0  | 32  | 0.0  | 20  | 0.0  |
| M1         | 83         | 3.6  | 154  | 1.9 | 13  | 7.7  | 32                   | 3.2  | 16   | 0    | 18  | 27.8 | 45                  | 28.9 | 62   | 17.7 | 9   | 22.2 | 5                   | 0.0  | 35   | 11.4 | 35  | 11.4 | 28                  | 14.3 | 28   | 14.3 | 28  | 14.3 | 28  | 14.3 |
| M2         | 91         | 9.9  | 149  | 2.7 | 13  | 15.4 | 28                   | 0.0  | 15   | 0    | 15  | 20   | 54                  | 16.7 | 64   | 21.9 | 7   | 28.6 | 7                   | 14.3 | 26   | 11.5 | 26  | 11.5 | 27                  | 3.7  | 27   | 3.7  | 27  | 3.7  | 27  | 3.7  |
| M3         | 59         | 22.0 | 124  | 4.8 | 14  | 28.6 | 27                   | 11.1 | 17   | 23.5 | 13  | 38.5 | 32                  | 18.8 | 49   | 16.3 | 5   | 40.0 | 6                   | 16.7 | 21   | 14.3 | 21  | 14.3 | 24                  | 12.5 | 24   | 12.5 | 24  | 12.5 | 24  | 12.5 |
| Total      | 579        | 4.5  | 1008 | 1.6 | 100 | 8.0  | 211                  | 2.8  | 124  | 5.6  | 120 | 17.5 | 395                 | 11.1 | 517  | 9.1  | 65  | 12.3 | 43                  | 4.7  | 240  | 4.2  | 240 | 4.2  | 163                 | 4.9  | 163  | 4.9  | 163 | 4.9  | 163 | 4.9  |
| Site Total | 1102       | 3.5  | 1791 | 1.8 | 226 | 12.8 | 482                  | 5.8  | 233  | 12.0 | 219 | 13.2 | 784                 | 15.4 | 934  | 11.5 | 150 | 14.0 | 101                 | 5.9  | 530  | 6.2  | 530 | 6.2  | 404                 | 6.4  | 404  | 6.4  | 404 | 6.4  | 404 | 6.4  |

<sup>1</sup>Molnar 1971

<sup>2</sup>Hoffman 1987

<sup>3</sup>Walker and Erlandson 1986

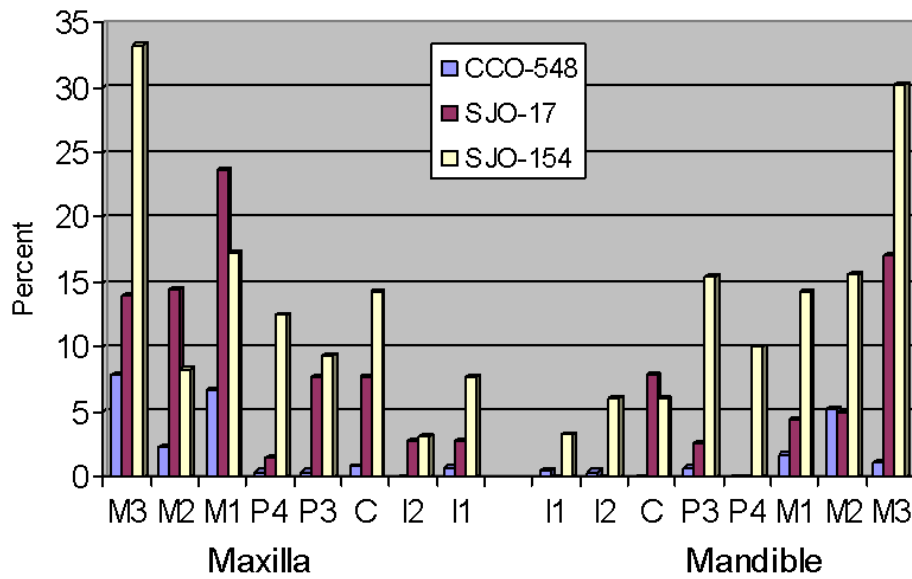


FIGURE 113. Frequencies of Carious Lesions in Maxillary and Mandibular Dentitions of Three Central California Populations (comparative data from Molnar 1971 and Hoffman 1987).

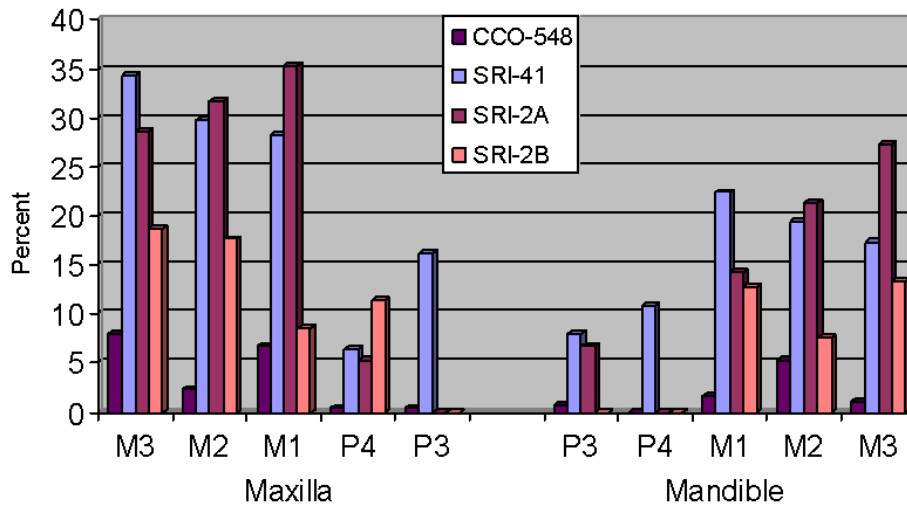


FIGURE 114. Frequencies of Carious Lesions in Maxillary and Mandibular Dentitions of CA-CCO-18/548 and Three Channel Islands Populations (comparative data from Walker and Erlandson 1986).

In the normal individual there is a delicate balance between commensal and pathogenic microflora of the oral cavity. The homeostasis of the indigenous oral microflora can be disrupted by reductions in pH which encourages the overgrowth of pathogenic species (Marsh 1994). Active disease arises from three interrelated factors: (1) host susceptibility mediated by genetic and non-genetic influences, (2) the presence of pathogenic organisms, and (3) the relative absence of benign species (Hassell and Harris 1995; Smalley 1994; Livingstone et al. 2002). Caries is the direct result of two interrelated events: (1) disturbance of the balance of commensal and pathogenic microflora in the oral cavity and (2) frequent consumption of fermentable carbohydrates (Marsh 1995; Lingström et al. 1994). Two primary bacteria (*Streptococcus mutans* and *Streptococcus sobrinus*) consume carbohydrates in the oral cavity and produce metabolic wastes which contribute organic acids and proteases to the oral fluids (Liljemark and Blooquist 1996). In sufficient concentration, the metabolic products can dissolve the mineral component of dental enamel. Left unchecked carious lesions can completely destroy a tooth and expose the circulatory system to infections such as bacteremia and septicemia. Dental caries may also be implicated in the development of cardiovascular disease (Meurman et al. 2004).

Examination of the frequencies of carious lesions in the teeth from CCO-18/548 reveals that the overall frequencies are quite low (2.5% for the maxillary dentition and 2.5% for the mandibular dentition). Comparison of carious lesion frequencies between males and females reveals that similar to other populations, females exhibit higher frequencies for most teeth. Despite the obvious differences in frequencies between the sexes, only two teeth (lower M2 and M3) have differences that are statistically significant at the point 05 level. Compared to other sites from the region, these frequencies are quite low. In fact, they are less than fifty percent of the lowest frequencies reported. These frequencies are even more remarkable considering only the molar teeth. Molars usually have the highest frequencies of carious lesions due to their complex fissure patterns (Klein and Palmer 1941; König 1963; Grainger et al. 1966). Similar to the overall comparison, the frequencies of carious lesions in the molar teeth for CCO-18/548 are the lowest of all the comparison sites. In most of the comparisons the frequencies of lesions for the molars of CCO-18/548 are less than half of that found at other sites and in some of the comparisons are less than one quarter. Frequencies of carious lesions at CCO-18/548 record a population with remarkably low levels of caries. Previous studies have linked the consumption of high carbohydrate diets with increased frequency of carious lesions. If this conclusion is correct, the interpretation here is that the population at CCO-18/548 was participating in a dietary regime with very low levels of carbohydrates. This is especially notable considering that later California populations exhibit considerably higher frequencies of carious lesions.

While the frequencies of carious lesions are quite low in the skeletal sample from the Marsh Creek Site dental attrition is notably high. Dental attrition is dependent on two major variables, diet and age of the individual. In general, hunter-gatherer populations have a more rigorous diet and will experience rapid dental attrition. In contrast, agricultural populations consume a softer more highly processed diet and experience much less attrition. Attrition is also age progressive. That is, the older an individual is the more attrition they will have on their teeth.

Researchers have long noted the remarkable degree of wear observed on the teeth of prehistoric California populations (Leigh 1928; Molnar 1971; Walker 1978; Reinhardt 1983; Jurmain 1990). Individuals frequently exceed the highest score for the various wear stage systems (Reinhardt 1983; Jurmain 1990). Interpretations vary as to the causative factors involved in this extreme wear. Jurmain (1990:341) suggests that the “quartz grit from sandstone mortars and pestles along with spalled fragments of cooking stones, all used in the preparation of acorns, as well as other abrasives introduced with shellfish processing” contributed in large part to the excessive attrition observed in the teeth of individuals from Late Horizon site ALA-329. He also observes that some of the wear may be due to fiber processing for baskets and cordage (cf. Molnar 1972; Schulz 1977; Grant 2007). Molnar (1971) also suggests a non-alimentary activity related cause for the excessive dental attrition seen in Early Horizon populations of central California. He points to a possible connection with the “occupations of basketry,

sandal making, and skin preparation” (Molnar 1971:187). Walker (1978) attributes the remarkable dental attrition seen in early prehistoric inhabitants of the Channel Islands to the consumption of marine resources, most especially shellfish and other resources obtained from the littoral zone. These resources would unintentionally introduce appreciable amounts of sand and grit to the oral cavity during their consumption.

In a study of Dutch whalers from an arctic whaling station, Maat and Van de Velde (1987) observed a difference in caries incidence between different wear stages. They concluded that the reason for the difference was due to the cleansing effects of an abrasive diet. However, for their study they compared individuals who all participated in the same diet. Therefore all of the individuals should have experienced the same level of dental cleansing from the abrasives in the diet. Using individuals from different populations and with differing diets might yield different results. Griffin and coworkers (2009) examined the hypothesis that dental attrition and frequencies of dental caries are inversely related using data from CCO-18/548 and a Late Mississippian site in southeastern Tennessee. The sample from the Marsh Creek Site was compared to a sample from the Davis Farm Site in southeastern Tennessee. The Davis Farm Site is a Late Mississippian village site dating to around AD 1550. As is typical for many Late Mississippian samples, the individuals from the Davis Farm Site exhibit moderate to high frequencies of carious lesions. Examination of the comparative frequencies between the two sites in regard to caries and attrition reveals a near perfect inverse relationship between the two sets of frequencies. Griffin and coworkers (2009) found that when compared between sites differing in diet and attrition levels there is indeed an inverse relationship between attrition and caries. The average overall attrition level for the CCO-18/548 population is 6.1 using the Smith wear system. Conversely, the frequency of carious lesions is extraordinarily low at 2.5%. In comparison with the Davis Farm Site, the frequencies are reversed. That is, for the Davis Farm Site the average level of attrition is 2.7 and the frequency of carious lesions is 10.8%. In addition to the high level of attrition in the CCO-18/548 population, the individuals exhibit an unusual wear pattern that the authors interpreted to be the result of non-dietary abrasion.

The results reported here indicate that there is an inverse relationship between attrition and frequencies of carious lesions. Due to the complex relationship between the oral environment and caries, the inverse correlation could be due to a number of factors. Heavy abrasion eventually removes the fissures from the molar teeth which are the primary formation area for carious lesions. Heavy abrasion also acts to consistently remove biofilm from the tooth surface. Once the biofilm is removed it is no longer a threat from the over-production of acid. Constant wear of the teeth by various abrasives therefore acts as an inadvertent tooth cleaning. With the consistent removal of biofilm initiation of caries is prevented even in the presence of high levels of carbohydrates. Dietary items and non-alimentary material consistently in contact with the oral cavity may also change the pH of the oral cavity thus disrupting the ability of pathogenic bacteria to displace the commensal bacteria. Further research should help shed light on the most likely cause for the reduction in cariogenic activity with increased attrition.

### *Periodontal Disease*

Periodontal disease or periodontitis is considered to be a ubiquitous pathological process of preindustrial societies (Gold 1985). Periodontitis has been defined as a chronic slowly progressive and destructive inflammatory process affecting one or more of the four components of the periodontium: gingival, periodontal ligament, cementum, and alveolar bone (Aufderheide and Rodríguez-Martin 1998). Gingivitis is an initial occurrence subsequent to an initiation of an imbalance of the indigenous oral microflora (Genco et al. 1988). The composition of microflora associated with gingivitis suggests that it is a transitional phase between health and periodontitis (Moore et al. 1987). Generalized periodontitis has been characterized as a horizontal reduction in alveolar bone height exposing the root surface to the oral fluids. The reduction in crestal height is thought to be the result of host immune and inflammatory activity which is in response to commensal bacteria proliferating in the oral cavity. Over time the bacterial colonies are thought to invade the deep periodontium to become periodontitis (Clarke et al.

1986). According to Aufderheide and Rodríguez-Martin (1998) periodontitis in modern populations is estimated by the World Health Organization to affect 75% of adults worldwide to some degree. Shafer and coworkers (1983) assert that periodontitis is responsible for more antemortem tooth loss than is dental caries in modern humans.

*Porphyromonas gingivalis* is one of the prominent members of the pathogenic microbiota associated with periodontitis (Loesche et al. 1985; Dzink et al. 1988). Ninety-three percent of patients with periodontitis harbor *P. gingivalis*, however it is unclear whether it occurs as part of the indigenous oral microflora or if it is an exogenous pathogen (Genco et al. 1988). *P. gingivalis* is associated with destruction of both connective tissues and bone in periodontal disease (Slots and Genco 1984). However, similar to the case with the etiology of dental caries, it is very likely that periodontal disease arises from a disruption of the microflora balance rather than caused by one specific organism.

According to Aufderheide and Rodríguez-Martin (1998) the single most important event leading to gingivitis and periodontitis is the loss of interproximal tooth contact. A number of events can lead to this loss of contact: traumatic enamel fracture, exfoliation of adjacent teeth, and advanced attrition. By far the most important of these in prehistoric populations is severe attrition. Once wear of the occlusal surface has eroded a tooth below the interproximal contact areas with the adjoining teeth, a gap or sulcus is created. During mastication food is forced into this sulcus, progressively expanding it with each successive meal. The progressive accumulation of food debris provides an ideal environment for bacterial colonization. As bacteria multiply and invade the alveoli (tooth sockets) the periodontal ligament is progressively compromised. The body reacts by resorbing the alveolar bone away from the site of infection. Left untreated, this continual retreat of the alveolus will result in the eventual exfoliation of the tooth.

Clarke and coworkers (1986) challenge the conventional assumption that generalized alveolar resorption and discrete alveolar bone lesions (“abscesses”) are part of the same chronic process. They point to the work of other researchers that indicates that gingivitis does not usually extend into the underlying bone (Listgarten et al. 1985) and that periodontitis does not progress continuously (Socransky et al. 1984). In their examination of modern and premodern dentitions Clarke and coworkers (1986) found very low incidence of periodontal disease which could be attributed to gingival inflammation. They also found that superficial lesions of the alveolar crest were usually not aggressive and tissue loss was minimal. Clarke and coworkers point to earlier research which implicates severe attrition as the mechanism for the formation of severe suppurative apical lesions. Ruffer (1920) is one of the earliest researchers to make this association. Later researchers have confirmed the association between pronounced dental attrition and severe isolated apical lesions (Larato 1970; Clarke and Hirsch 1991). The progression of pathological processes leading to apical lesions begins with injury of the dental pulp through excessive dental attrition. Inflammation of the periodontal ligament ensues from the diffusion of inflammatory mediators from the damaged pulp through the dentinal tubules and cementum (Torobinejad and Bakland 1980). Lesions resulting from this process are generally deep and seriously destructive of the alveolar bone ultimately compromising tooth support (Simring and Goldberg 1964).

Clarke and Hirsch (1991) extend this model by pointing to a connection between heavy functional force, pulp perforation, apical abscess formation, pronounced tooth tilting, and ultimately tooth exfoliation. They suggest that typically with chronic heavy masticatory stress there is inevitably significant dental attrition and physiological continuous tooth eruption. That is, the body compensates for progressive loss of tooth occlusal structure by vertical tooth movement in the supporting bone structure (Murphy 1959; Barker 1975; Newman 1999). This has the effect over time of reducing the amount of bony support structure surrounding the tooth. Continued attrition eventually results in pulp exposure which allows oral bacteria to invade the endodontic space. This results in a dental abscess. Dental abscesses typically have a number of associated complications including destruction of supporting bone at the cemento-enamel junction (Kelly and Ellinger 1988) and eventual invasion of the periodontal ligament

resulting in a periapical lesion. The later results in considerable destruction of the surrounding tooth supporting bone. The compromise of the thin buccal bone of the alveolus combined with the destruction of the majority of tooth support from the processes described above and continued heavy masticatory force inevitably results in lingual tilting of the tooth and eventual exfoliation (Reinhardt 1983). Clarke and Hirsch also note that the use of teeth as tools has been implicated in excessive attrition and lingual tooth tilting (c.f. Osborn 1982; Reinhardt 1983).

Dias and Tayles (1997) point out that most periapical cavities found in the archaeological record are likely not abscesses but rather the result of perapical granuloma and cysts. Both form as part of a chronic low-grade infection and both result in a cavity in the bone. Granulomas are a soft tissue mass composed of fibroblasts, endothelial cells, vascular channels, lymphocytes, plasma, and collagen fibers. Expansion of the mass results in osteoclastic excavation of a bony cavity. Cysts frequently form secondarily to a granuloma as epithelial cells migrate over the surface of the mass and the granulation tissue is replaced by fluid. Granuloma and cysts normally result in smooth walled cavities with circumscribed margins and measure less than 3 mm. These can be contrasted with abscesses which are the result of an acute pyogenic infection with tissue necrosis and ebulent pus formation. Periapical abscesses are generally greater than 3 mm, are extremely painful, and usually result in slight extrusion of the tooth from the alveolus. The walls of an abscess are characterized by slightly roughened appearance in dry bone.

The distinction between abscesses and granuloma and cysts is important in that the patient with the later two conditions is not necessarily visibly ill. In fact it can be argued that granuloma and cysts are the response of a healthy immune system to a bacterial invasion (Dias and Tayles 1997). They however point out that all three pathological processes have the potential to contribute to an individual's death through systemic infection. All three also certainly contribute to tooth exfoliation.

Each tooth position was examined macroscopically for the presence of alveolar defects. Defects were recorded by location and size. For purposes of intersite comparison, all locations and sizes of defects were pooled together. Left and right maxillary and mandibular tooth positions were also pooled for comparison. The frequencies are reported in Table 86 and Figures 115-117. Examination of the frequencies reported in Table 86 reveals a relatively high number of alveolar defects across the entire sample, especially in the region of the maxillary and mandibular first and second molars. Considering the likely etiology for apical lesions (the majority of defects observed here) proposed by Clarke and coworkers (1986), the most likely explanation for the high frequencies of apical lesions in these regions is excessive attrition. The frequencies of alveolar defects at each tooth position correspond nearly perfectly with the attrition levels (reported above) observed for the teeth at those positions.

### *Dental Hypoplasias*

The hard tissues of the human body (bone and teeth) provide a durable record of the growth and development of the individual and also of changes over time among and between populations. This record is particularly valuable in assessing the biological changes accompanying a subsistence shift (e.g. from hunting and gathering to agriculture) or changes in local resource base. A number of different aspects of the human skeleton have been examined to delineate changes with the adoption of agriculture. Among these are growth and development of the hard tissues, pathological indicators of deprivation, dental and skeletal infection, pathological articular modifications, and alterations in bone form and function.

Measurement of rate and timing of growth in humans provides an index of differential response to specific environmental circumstances or conditions (Larsen 1987). Examination of growth rates of femoral diaphyses (Cook 1972, 1984) and tibial diaphyses (Goodman et al. 1984) has demonstrated that growth rates are slower among agriculturalists than among their hunter-gatherer predecessors. These studies have suggested that slower growth rates in these populations reflect an increase in stress.

TABLE 86. Frequencies of Periapical Abscesses in Maxillary and Mandibular Dentitions.

| TOOTH      | TOTAL SAMPLE |       | FEMALE |       | MALE |       |
|------------|--------------|-------|--------|-------|------|-------|
|            | N            | %     | N      | %     | N    | %     |
| Maxilla    |              |       |        |       |      |       |
| I1         | 185          | 9.73  | 52     | 11.54 | 101  | 9.90  |
| I2         | 193          | 6.73  | 59     | 8.47  | 102  | 5.88  |
| C          | 224          | 8.93  | 69     | 10.14 | 110  | 8.18  |
| P3         | 227          | 8.37  | 67     | 8.95  | 106  | 8.49  |
| P4         | 226          | 9.29  | 67     | 16.42 | 106  | 8.49  |
| M1         | 236          | 34.74 | 71     | 40.84 | 109  | 38.53 |
| M2         | 213          | 12.21 | 60     | 18.33 | 105  | 13.33 |
| M3         | 167          | 5.39  | 43     | 2.32  | 89   | 8.99  |
| Total      | 1671         | 12.4  | 488    | 15.6  | 828  | 12.9  |
| Mandible   |              |       |        |       |      |       |
| I1         | 201          | 1.49  | 61     | 3.28  | 96   | 1.04  |
| I2         | 228          | 3.51  | 73     | 9.59  | 106  | 0.94  |
| C          | 254          | 5.51  | 77     | 12.99 | 115  | 0.87  |
| P3         | 275          | 6.18  | 88     | 12.50 | 121  | 1.65  |
| P4         | 295          | 5.08  | 88     | 6.82  | 135  | 5.18  |
| M1         | 323          | 23.84 | 91     | 30.77 | 150  | 24.67 |
| M2         | 320          | 15.31 | 91     | 15.38 | 148  | 18.92 |
| M3         | 258          | 7.36  | 71     | 5.63  | 126  | 10.32 |
| Total      | 2154         | 9.4   | 640    | 12.8  | 997  | 9.0   |
| Site Total | 3825         | 10.7  | 1128   | 14.0  | 1825 | 10.8  |

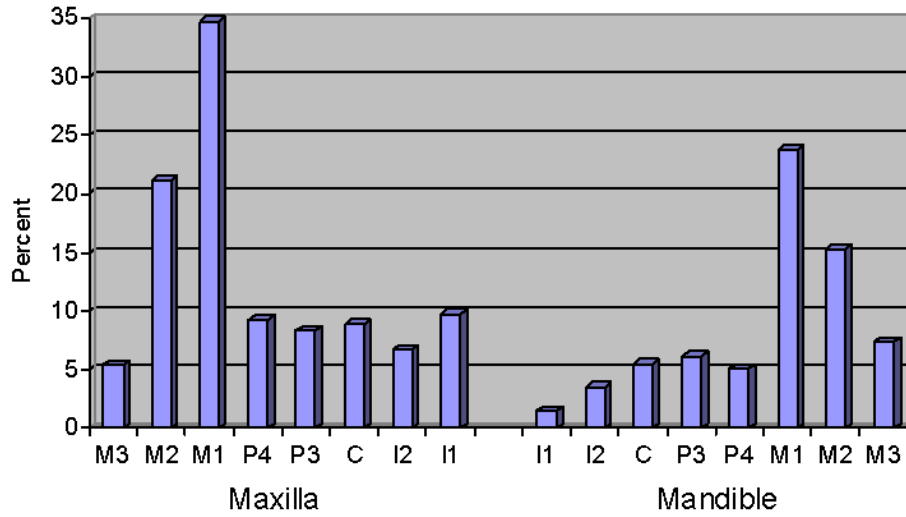


FIGURE 115. Frequencies of Periapical Abscesses in Maxillary and Mandibular Dentitions for CA-CCO-18/548.

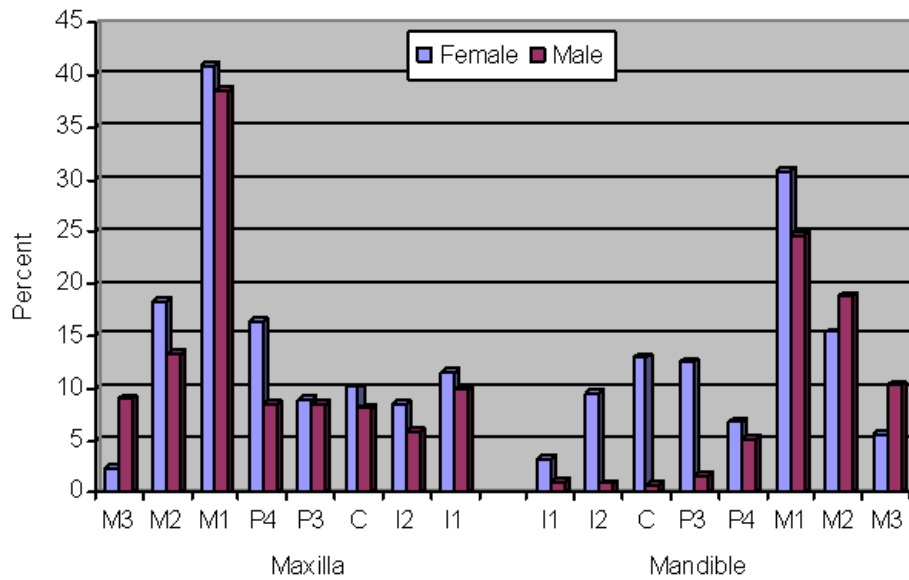


FIGURE 116. Frequencies of Periapical Abscesses in Female and Male Maxillary and Mandibular Dentitions for CA-CCO-18/548.



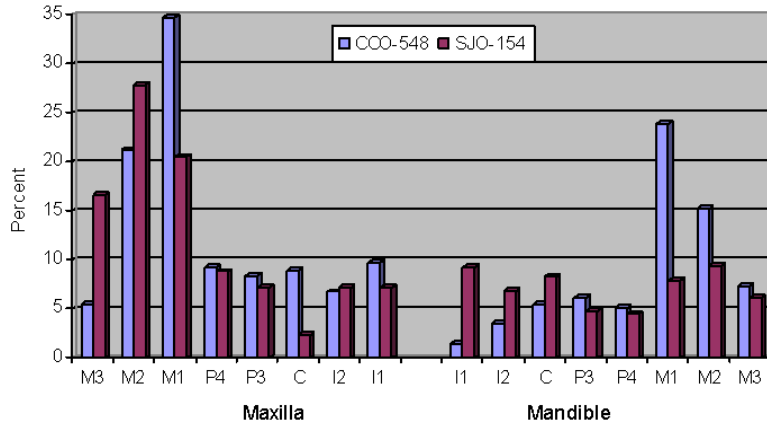


FIGURE 117. Frequencies of Periapical Abscesses in Maxillary and Mandibular Dentitions of CA-CCO-18/548 and CA-SJO-154 (Hoffman 1987).

Depressed growth rate is directly reflected in adult body size. A number of researchers have presented evidence for reduction in body size and stature in prehistoric populations (Cohen and Armelagos 1984; Nickens 1976; Haviland 1967; Larsen 1982). For all of these studies nutritional deficits related to the adoption or intensification of agriculture, specifically maize agriculture, are implicated. Although maize can provide adequate caloric intake, it contains only small amounts of protein (<4 gms per lb) and lacks two essential amino acids, lysine and typtophan, and a vitamin, niacin. The lack of even one amino acid in the diet will preclude the utilization of the rest (Stini 1971). Consequently, an increased reliance on maize to the exclusion of other complimentary food resources necessarily leads to poorer nutrition and depressed growth rates.

Dietary deficiencies can also be reflected in tooth size. Garn et al. (1979) have suggested that dental development may be retarded *in utero* by maternal dietary insufficiencies. The result of this slowing of development is a reduction in size of both deciduous and permanent dentitions. Larsen (1983b) demonstrated that deciduous tooth size was reduced in agricultural period populations in comparison with earlier hunter-gatherer populations from the Georgia coast. It was suggested that this change was the result of poorer maternal health during the agricultural period, possibly as a result of poorer nutrition.

Growth disruption, like growth retardation, can be the result of dietary insufficiencies. The connection between nutritional stress and growth disruption is well documented in prehistoric skeletal populations. The connection between specific stressors (e.g., rickets and scurvy) and growth disruption has also been implicated in the skeletal record (Huss-Asmore et al. 1982; Martin et al. 1985). However, specific nutritional deficiencies are quite rare in the skeletal record (Goodman et al. 1988). Growth disruption can result in the formation of linear enamel hypoplasias on the developing teeth of young individuals. A dental enamel hypoplasia is a deficiency in enamel thickness which manifests as a band around the circumference of a tooth's crown. They are roughly parallel to the cement-enamel junction and perpendicular to the tooth's long axis (Hillson 1996; Goodman and Song 1999). Normal amelogenesis (enamel formation) proceeds in two stages, secretory and maturation (Nanci 2007). During the secretory stage ameloblasts release proteins into the area surrounding the already formed dentine portion of the tooth. The enzyme alkaline phosphatase initiates the partial mineralization of the enamel during this stage. The maturation stage of amelogenesis involves the transport of proteins to the enamel formation area. Most of the proteins are gradually removed leaving behind a striated mineralized enamel matrix. Interruption of this process results in linear bands of enamel insufficiency (linear enamel hypoplasias).

Linear enamel hypoplasias (LEH) are accepted to be related to periodic physiological disruptions to enamel matrix secretion during development (Ritzman et al. 2008). According to Larsen (1997) tooth enamel formation is especially sensitive to metabolic insults that result from nutritional deficiencies or disease. He observes that ameloblasts “are especially sensitive to even minor physiological disruptions” (Larsen 1997:44). Clinical and epidemiological studies have demonstrated the association of LEH formation with systemic diseases, neonatal disturbances, and nutritional deprivation (reviewed in Hillson 1996). These defects are therefore considered to be general indicators of metabolic stress (Goodman and Rose 1990, 1991; Hillson 1996; Hillson and Bond 1997; Larsen 1997; Goodman and Song 1999; Guatelli-Steinberg 2001). LEH’s are especially prevalent on the anterior dentition (incisors, canines, premolars) and are normally found on the cervical and middle thirds of the tooth crown (Condon and Rose 1992; Goodman and Armelagos 1985a, 1985b; Hutchinson and Larsen 1988, 2001; Li et al. 1995; Zhou 1995). These teeth therefore provide the most representative record of stress for the dentition.

Dental enamel differs from bone in that it cannot be remodeled after tooth formation. LEH’s then form a permanent chronological record of a stressful event during the first seven years of life (Goodman and Rose 1990). Enamel is laid down in a series of layers beginning at the cusp of the tooth and proceeding downward to the cement-enamel junction (Ritzman et al. 2008). Thus a measurement taken from the LEH to the cement-enamel junction can be used to calculate the age at which the stress event occurred. Researchers have developed standards of dental development to ascertain age of LEH formation from these measurements (Swärdstedt 1966; Goodman et al. 1980; Ritzman et al. 2008). Lewis and Roberts (1997) advise that since tooth crown heights differ from population to population then corrective equations must be used to calculate times of LEH formation for each unique population. While this method would certainly make age-at-occurrence estimates more accurate, it is of little practical value in populations that exhibit considerable attrition from an early age. This is because the corrective equations require a sample of unworn teeth from the population, a precondition that is frequently not available in central California prehistoric populations.

All anterior teeth for the dentitions of CCO-18/548 were examined with 3.5x dental loupes for the presence of linear enamel defects. Teeth included in the data reported below had at least 2 mm of continuous enamel measured from the cement-enamel junction to the occlusal surface. The distance of each defect was measured from the cement-enamel junction. The method of Goodman and Rose (1990) was used here to make estimates of age of occurrence for the enamel defects in this population. Corrective equations suggested by Ritzman et al. (2008) are not used because of the absence of unworn teeth needed to calibrate the equations. The frequencies are reported in Table 87.

Examination of frequencies of enamel hypoplasias in this population sample reveals moderate to low levels of hypoplastic defects. Compared with other population samples from the region the frequencies of hypoplastic defects at CCO-18/548 fall in the middle of the range (Figure 118). In general, precontact California Native American populations exhibit very low levels of hypoplastic defects when compared to other North American regional populations. The low to moderate level observed here indicates a relatively healthy population, at least in regard to the early developmental years. The high concentration of defects in the 3-5 year age range (Figures 119 and 120) is typical and likely corresponds to ages at which individuals are at highest risk for childhood illnesses.

TABLE 87. Frequencies of Anterior Teeth with One or more Linear Enamel Hypoplasias.

| TOOTH | MAXILLA |      | MANDIBLE |      |
|-------|---------|------|----------|------|
|       | N       | %    | N        | %    |
| I1    | 82      | 15.8 | 91       | 0.0  |
| I2    | 93      | 14.0 | 115      | 1.7  |
| C     | 115     | 32.2 | 139      | 45.3 |
| P3    | 115     | 1.7  | 121      | 5.0  |
| P4    | 116     | 3.4  | 130      | 4.6  |

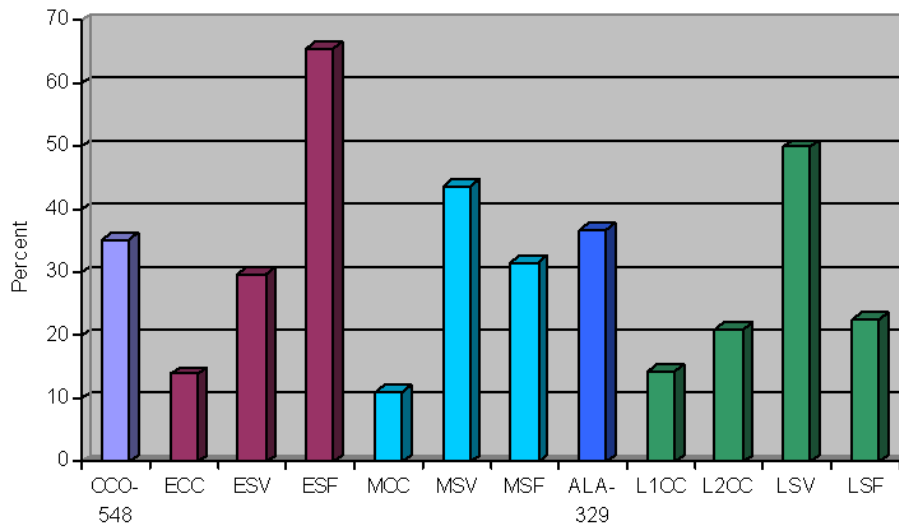


FIGURE 118. Frequencies of Enamel Hypoplasias in Canines of CA-CCO-18/548 Compared with other Central California Populations: CA-ALA-329 (Nechayev 2007); Early (ECC), Middle (MCC), Late Period 1 (L1CC), Late Period 2 (L2CC) from Central California (Schulz 1981); Early (ESV, ESF), Middle (MSV, MSF), and Late (LSV, LSF) Period samples from Sacramento Valley and San Francisco Bay (Bartelink 2006).

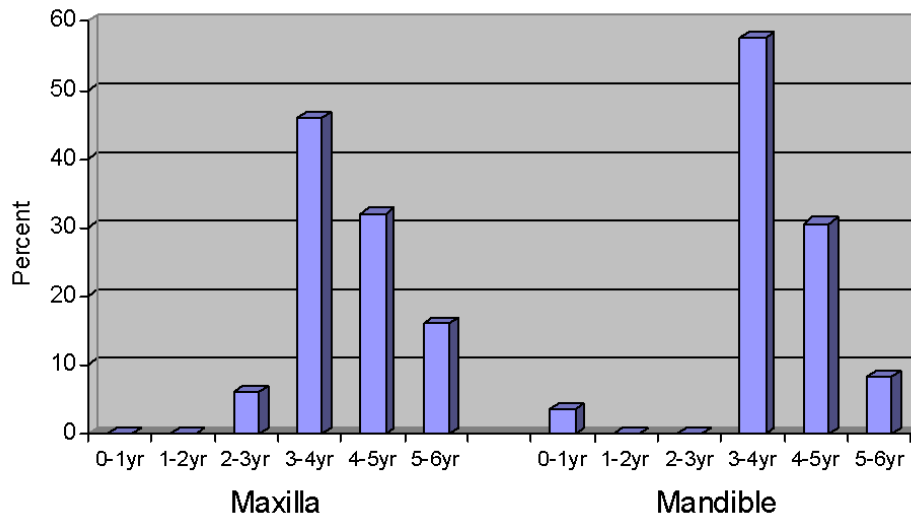


FIGURE 119. Age Distribution of Enamel Hypoplasias in Maxillary and Mandibular Canines; calculated using Goodman and Rose (1990).

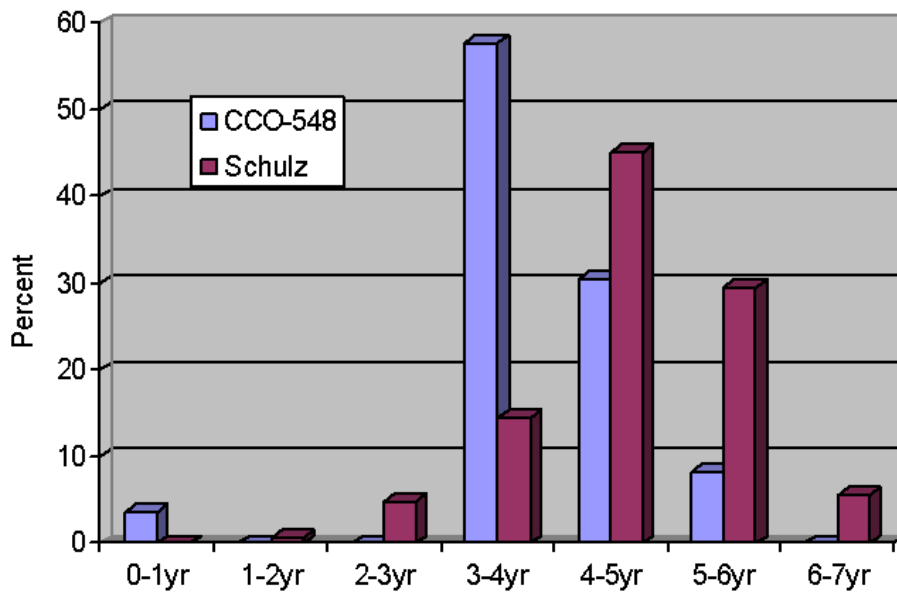


FIGURE 120. Age Distribution of Enamel Hypoplasias in Mandibular Canines of CA-CCO-18/548 and Other Central California Populations; comparative data from Schulz (1981).

## Postcranial Pathology

The postcranial skeleton frequently exhibits the highest number of pathologic conditions. This is likely due to a number of factors. Most important among these would be the fact that the postcranial skeleton contains the highest number of individual elements, the largest number of freely mobile (diarthrotic) joints, and is subject to modifications and injury due to various activities. The pathologic conditions observed on the postcranial skeletons of this sample include three separate arthropathies (diseases of the joints): degenerative joint disease, rheumatoid arthritis, and spondyloarthropathy. In addition there were many cases of periosteal reactions (nonspecific infection) and numerous fractures. The conditions specifically reported on here are degenerative joint disease and periosteal reactions.

### *Degenerative Joint Disease*

Osteological analysis of degenerative diseases has been of particular importance in the examination of past human lifeways (Jurmain 1977, 1980; Merbs 1983; Kilgore 1984; Kelley and Angel 1987). Certainly one of the oldest diseases afflicting humans, degenerative joint disease is one of the most commonly encountered in archaeological populations (Jurmain 1977). The term “degenerative joint disease” is used here rather than the frequently used term “arthritis.” The reason for this is that the clinical term “arthritis” refers to inflammation of the joints, a soft tissue condition, which cannot usually be demonstrated in skeletal tissue.

Osteoarthritis is a clinically diagnosed, age progressive joint disorder. It ultimately results in the deterioration of articular cartilage, reduction of the joint space, formation of osteophytes, eburnation, and incongruity at the joint surfaces and margins (Terrono and Horner 2000; Lozada and Altman 2001; Boissonnault and Goodman 2003). In its clinical presentation, osteoarthritis causes a reduced range of motion in the affected joint(s) which is a response to the pain and dysfunction associated with the condition (Felson et al. 2000). It is however notable that many patients with pathologic and radiographic indications of osteoarthritis are asymptomatic (Lawrence et al. 1966). Osteoarthritis is a multifactorial condition which has been linked to biochemical, biomechanical, and genetic triggers (Hoderbaum et al. 1999). Among the specific factors cited are age, systemic issues, acute trauma, weight, genetic predisposition, and mechanical stress (Felson et al. 2000).

In the absence of clinical records documenting a patient’s symptomology it is impossible to diagnose osteoarthritis in the skeletal record. Archaeologists frequently use the presence of osteophytes or articular surface remodeling to infer the presence of osteoarthritis and also frequently attempt to relate these to levels of pain or dysfunction (Jurmain and Kilgore 1995). Bioarchaeologists also frequently attempt to link frequencies of arthropathies to specific activities. As Jurmain and Kilgore (1995) point out, on the basis of currently available data, these conclusions are virtually untestable. Based on the uniformity of expression of degenerative joint disease of the spine across prehistoric populations, Jurmain and Kilgore (1995) conclude that degenerative changes in the vertebral column are the ubiquitous manifestation of a lifetime in a bipedal posture. They however interpret the variation observed in frequencies of degenerative joint disease in the peripheral joints (e.g., elbow and knee) as reflecting differences in culturally based activities. These activities place mechanical loads of varying degrees on the synovial joints, thus possibly predisposing them to damage. Brown et al. (2008) however have demonstrated in cadaveric spines that a combination of age and load-bearing above a 50% threshold of compressive force is associated with severe degenerative changes in cartilage and bone. This finding suggests that there may be a connection between activity patterns and degenerative changes in the spine.

Degenerative joint disease has frequently been attributed to high chronic demand on the affected joints (Larsen 1982, 1987; Merbs 1983; Pickering 1984; Walker and Hollimon 1989; Bridges 1991; Larsen et al. 1995, 1996; Hemphill 1999; Larsen and Hutchinson 1999). While the clinical research

clearly indicates that degenerative changes in the joints is multifactorial, activities that chronically overwork the joints and fatigue the associated muscles are implicated in an appreciable percentage of osteoarthritis diagnoses (Felson et al. 2000). Activity patterns are therefore a likely cause of an appreciable amount of degenerative joint changes in the skeletal record. Weiss and Jurmain (2007) caution that much of the variation observed in degenerative disease frequencies is attributable to age. They also caution that the variation of hormones may play a far greater role in sex disparities in populations than differences in activity levels between the sexes.

Degenerative joint disease is identified in skeletal tissues by the presence of joint hypertrophy through osteophytic development (lipping) and erosion (porosity) and eburnation (polishing) of subchondral bone (Aufderheide and Rodríguez-Martin 1998). A variety of schemes have been proposed for the systematic scoring of degenerative changes in skeletal joints (e.g., Jurmain 1975; Brothwell 1981; Buikstra and Ubelaker 1994; Rojas-Sepúlveda 2008). Joints for this skeletal sample were recorded using Brothwell’s system. Because of the very low frequencies for this site, the categories were collapsed to only presence or absence.

Examination of the frequencies reported in Table 88 and Figure 121 reveals a population with very low frequencies of degenerative joint disease especially considering the age-at-death profile for this sample. The frequencies reported in Figures 122 and 123 show that the overwhelming majority of individuals affected are nearly evenly divided between the age cohorts of 40-49 and 50-59. This reflects the age-progressive nature of degenerative joint disease but also shows a remarkably healthy population in regard to arthropathies. The joints affected most frequently here are the spine and knee. This is consistent with overall general trends for populations exhibiting degenerative joint disease (Figures 124 and 125). Examination of frequency differences between females and males (Figure 126) reveals very few differences. The joints that do exhibit statistically significant differences are the sacrum, knee, and ankle. This may indicate a differing activity pattern impacting the lower joint more heavily for females than males.

TABLE 88. Frequencies of Degenerative Joint Disease.

| JOINT    | TOTAL SAMPLE |       | FEMALE |       | MALE |       |
|----------|--------------|-------|--------|-------|------|-------|
|          | N            | %     | N      | %     | N    | %     |
| Cervical | 121          | 36.36 | 37     | 43.24 | 52   | 40.39 |
| Thoracic | 145          | 27.59 | 46     | 26.09 | 62   | 29.03 |
| Lumbar   | 114          | 50.88 | 35     | 57.14 | 51   | 52.94 |
| Sacrum   | 101          | 16.83 | 36     | 25.00 | 46   | 8.70  |
| Shoulder | 179          | 13.41 | 58     | 13.79 | 77   | 14.29 |
| Elbow    | 174          | 21.84 | 55     | 23.64 | 72   | 25.00 |
| Wrist    | 53           | 11.32 | 12     | 8.33  | 32   | 15.63 |
| Hand     | 41           | 19.51 | 5      | 20.00 | 29   | 20.69 |
| Hip      | 153          | 6.54  | 46     | 8.70  | 69   | 7.25  |
| Knee     | 71           | 28.17 | 20     | 35.00 | 41   | 24.39 |
| Ankle    | 107          | 14.02 | 34     | 20.59 | 52   | 11.54 |
| Foot     | 40           | 15.00 | 7      | 14.29 | 23   | 13.04 |

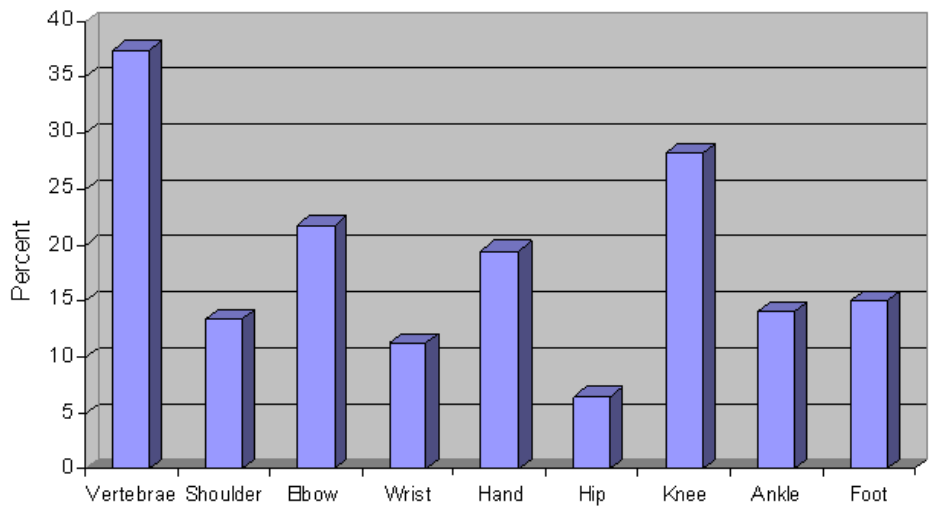


FIGURE 121. Frequencies of Degenerative Joint Disease in CA-CCO-18/548.

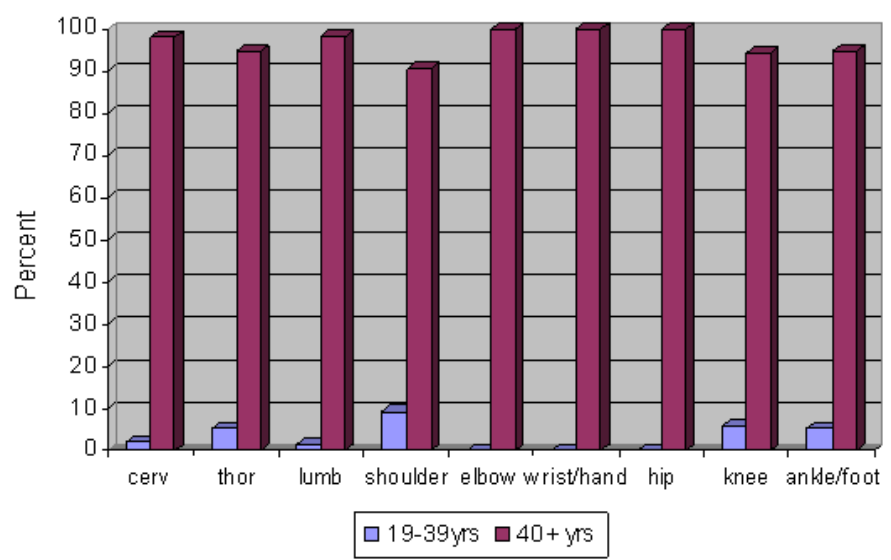


FIGURE 122. Frequencies of Degenerative Joint Disease in CA-CCO-18/548, Two Age Categories.

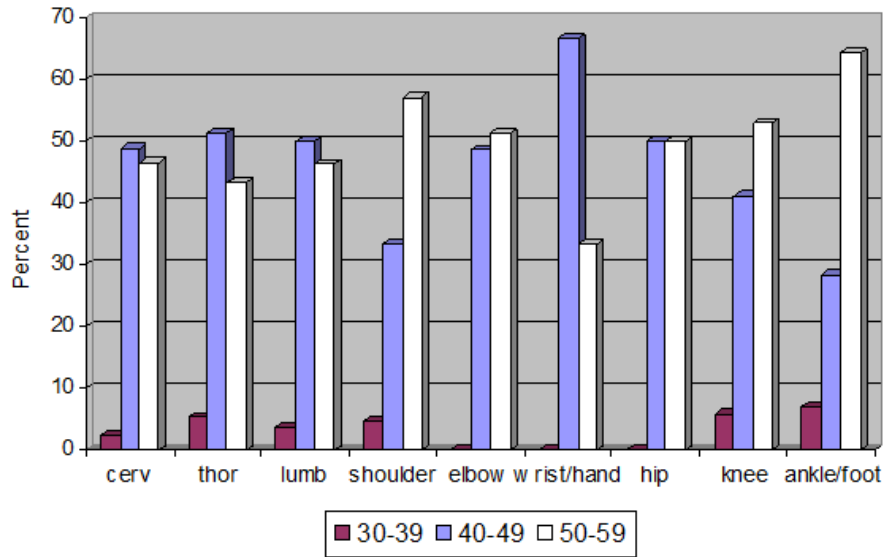


FIGURE 123. Frequencies of Degenerative Joint Disease in CA-CCO-18/548 by Decade.

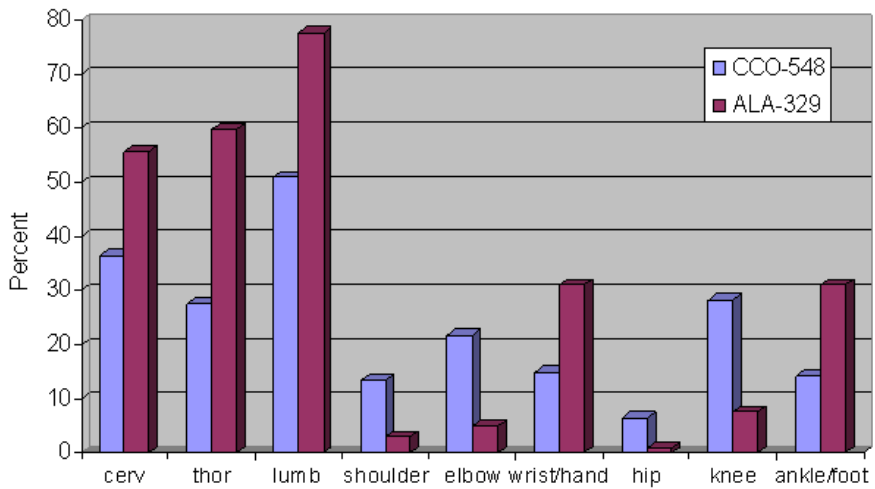


FIGURE 124. Frequencies of Degenerative Joint Disease in CA-CCO-18/548 and CA-ALA-329; comparative data from Jurmain (1990).



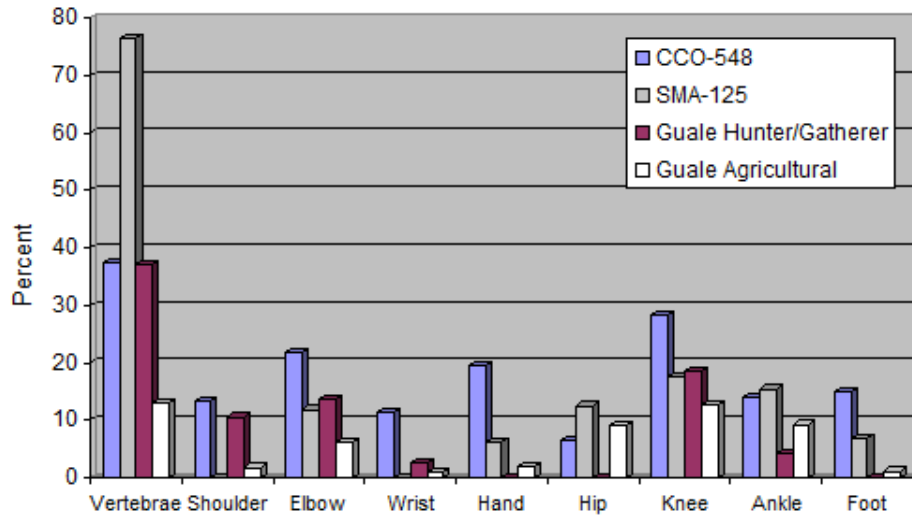


FIGURE 125. Frequencies of Degenerative Joint Disease in CCO-18/548 and Three other Prehistoric Populations; comparative data from Griffin et al. (2006) and Larsen et al. (1996).

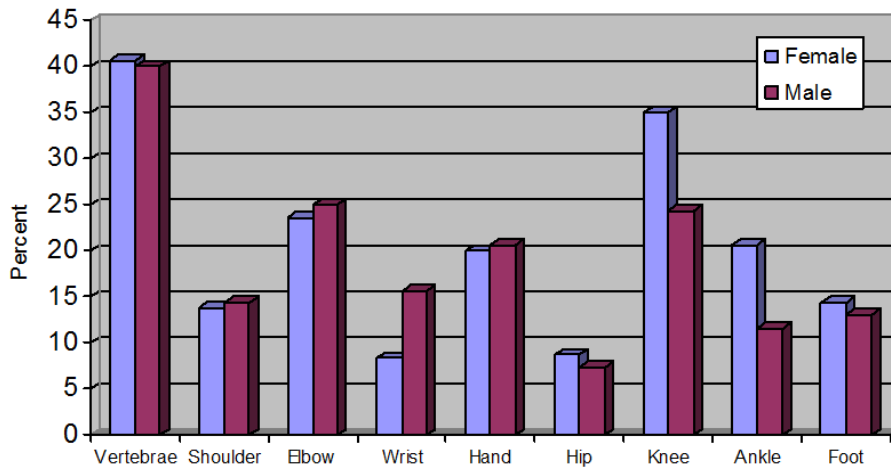


FIGURE 126. Female and Male Frequencies of Degenerative Joint Disease in CA-CCO-18/548.

### *Nonspecific Inflammation*

The examination of nonspecific bone inflammation may indicate the relative health of populations. As such, the study of skeletal lesions may give indications of the health risks associated with a particular dietary regime (although other factors certainly contribute to the incidence of disease). Examination of infectious disease in archaeologically derived populations is generally limited to the identification of nonspecific skeletal lesions. These lesions are usually referred to as periosteal reactions (Cohen and Armelagos 1984). The identification of specific infectious diseases like treponema, tuberculosis, and leprosy are more difficult to document from the characteristics of skeletal lesions. Recent research with documented historical skeletal collections indicates that there are no qualitative or quantitative characteristics of periosteal reactions which will allow the identification of specific disease states (Weston 2008). The frequency and severity of nonspecific skeletal lesions may give a general indication of the overall health of a population and can be used to infer the relative amount of stress resulting from lifeway transition.

The periosteum is a dense, white, fibrous membrane that forms a tight covering around the non-articular surfaces of bone (Tortora and Grabowski 2003). The periosteum is responsible for (1) holding in place the blood vessels, lymphatic vessels, and nerves that pass into the bone and (2) new bone formation during growth and repair. The second function is of primary interest here. When the periosteum or the underlying bone is injured or becomes infected, the bone forming cells of the periosteum (osteoblasts) increase their activity and can produce massive amounts of new bone (Ortner and Putschar 1985). This new bone may later become incorporated into the underlying cortex and be remodeled into lamellar bone. The result of this new bone accumulation is the formation of an osseous plaque on the external surface of the bone (Eyre-Brook 1984; Simpson 1985). This process of increased bone formation is referred to variously as periosteal reactions or periostitis. While periostitis as a distinct disease process is rare (Ortner and Putschar 1985), periosteal reactions frequently occur secondarily to trauma and infection. Putschar (1966) asserts that it is often impossible to determine in dry bone the exact etiology of a periosteal reaction. Thus it may be impossible to determine whether a periosteal reaction is the result of trauma, secondary infection, or a general disease process (e.g., syphilis). Virtually anything that breaks, tears, or even touches the periosteum can stimulate the formation of new bone (Richardson 2007). Weston (2008) points out that many researchers have mistakenly made a direct connection between periosteal reactions and infectious processes. Periosteal reactions are frequently associated with inflammation. However inflammation is the body's vascular response to tissue damage and infection is only one cause of this response. Therefore frequencies of periosteal reactions can only be used as general indicators of overall health and not as indicators of levels of infection. Periosteal reactions can occur on any bone, but are particularly common on the tibia. One possible explanation for this is that bones near the skin are exposed to more direct trauma than bones protected by overlying muscles (Ortner and Putschar 1985).

Periosteal reactions are characterized by the formation of areas of rough, uneven cortical bone that have a "woven" surface appearance. Often the additional bone is not of uniform thickness over the entire cortical surface. Highly localized reactions are common especially as a result of penetrating wounds or overlying skin ulcers. Marked, uneven hypervascularity typically appears on dry bone as areas of variable sized pores.

The examination of frequencies of periosteal reactions has been used to infer the relative health of populations (Cohen and Armelagos 1984). The study of periosteal lesions has been used to evaluate the general health risks associated with a particular dietary regime (Lallo et al. 1978; Larsen 1982; Cohen and Armelagos 1984; Detweiler-Blakely 1988). In this way the frequency and severity of nonspecific skeletal lesions give an accurate indication of the general health of a population and can be used to infer the relative amount of stress resulting from lifeway transition. Larsen (1982) in an examination of infectious lesions in population samples from the Georgia coast found an increase in frequency of lesions in comparisons between hunter-gatherers and agriculturalists. The author suggests that these findings

support the general epidemiological model that an increase in population size and density during later prehistoric times was conducive to the maintenance and spread of infectious disease. Lambert (1993) also observed an increase in frequency over time of periosteal reactions for prehistoric populations. Lambert's study examined a time-successive series of populations in the Channel Islands of California. Lambert (1993) attributes the increase in frequencies of periosteal reactions in the populations from the Channel Islands to two possible causes. One possibility is a transition from a hunter-gatherer economy concentrating on carbohydrate-rich roots and tubers in the late Early Period (2400-1400 BC) to a fish based economy in the early Middle Period (1400 BC – AD 300). Lambert (1993) suggests that while the later fish-based diet provided adequate protein, the diet may have been deficient in other important nutrients due to inadequate supplies of plant foods. Lambert also observes that another possibility for the increase in periosteal reactions over time in the Channel Islands are episodes of sea-water warming and extended periods of drought during the late Middle Period indicated by sea-core and tree-ring data. These would have diminished the availability of marine resources, terrestrial plant resources and potable water.

Examination of the frequencies of periosteal lesions reported in Table 89 and Figure 127 reveals a population with extraordinarily low incidence of nonspecific infections. The comparative frequencies from other California precontact sites reported in Figure 128 show a revealing pattern. Early and Middle Period sites show very low levels of nonspecific infection as reflected by frequencies of periosteal lesion while late period sites show markedly elevated levels. Many authors have noted the adverse impact of sedentism on levels of nonspecific infection (Lallo et al. 1978; Larsen 1982; Cohen and Armelagos 1984; Deweiler-Blakely 1988). The frequencies reported in Figure 128 likely also reflect increased sedentism with a shift to acorn harvesting.

It is equally informative that the pattern of pathological periosteal involvement indicated by the frequencies reported in Table 89 is localized. That is, the bones most frequently affected in this population sample are those of the distal lower limb (tibia and fibula). Larsen (1982) and Detweiler-Blakely (1988) have concluded that this type of localized pattern is likely an indication of trauma rather than generalized stress. Considering the frequencies reported in Figure 129 it is apparent that this pattern is especially prevalent in females compared to males. Comparison of male and female frequencies of periosteal lesions in the tibia and fibula reveals statistically significant differences between the sexes ( $p < 0.05$ ). This pattern suggests a differing activity pattern in males and females predisposing the latter to injuries of the lower limb. Differential activity patterns more heavily impacting the lower limb in females is also supported by the frequencies of degenerative joint disease reported above.

TABLE 89. Frequencies of Periosteal Reactions.

| ELEMENT  | TOTAL SAMPLE |      | FEMALE |       | MALE |      |
|----------|--------------|------|--------|-------|------|------|
|          | N            | %    | N      | %     | N    | %    |
| Clavicle | 153          | 0.00 | 50     | 0.00  | 66   | 0.00 |
| Humerus  | 311          | 1.60 | 76     | 1.32  | 90   | 2.22 |
| Radius   | 219          | 0.40 | 62     | 1.61  | 81   | 0.00 |
| Ulna     | 245          | 0.80 | 65     | 0.00  | 83   | 0.00 |
| Femur    | 348          | 4.00 | 76     | 5.26  | 103  | 4.84 |
| Tibia    | 322          | 7.40 | 71     | 14.09 | 96   | 5.21 |
| Fibula   | 267          | 6.70 | 62     | 11.29 | 84   | 4.76 |

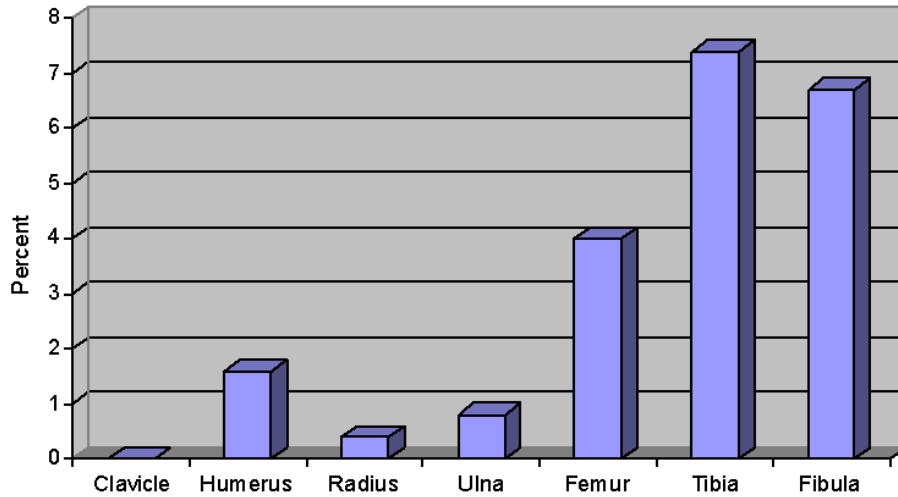


FIGURE 127. Frequencies of Periosteal Lesions in CA-CCO-18/548.

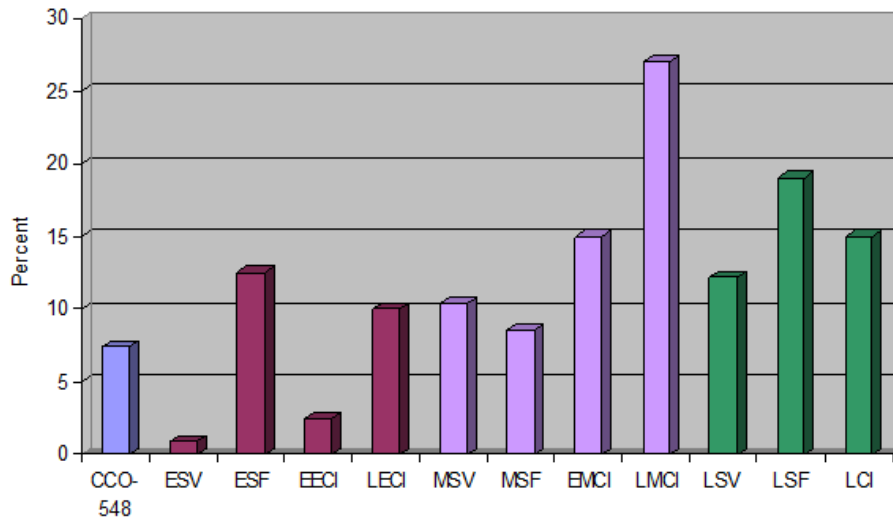


FIGURE 128. Frequencies of Tibial Periosteal Lesions in CA-CCO-18/548 Compared with other Central California Populations (Early, Middle, and Late Period samples from Sacramento Valley and San Francisco Bay: Bartelink 2006; five time periods for the Channel Islands, EE 6000-3500 BC, LE 3500-1400 BC, EM 1400 BC–AD 300, LM AD 300-1150, L AD 1150-1782; Lambert 1993).

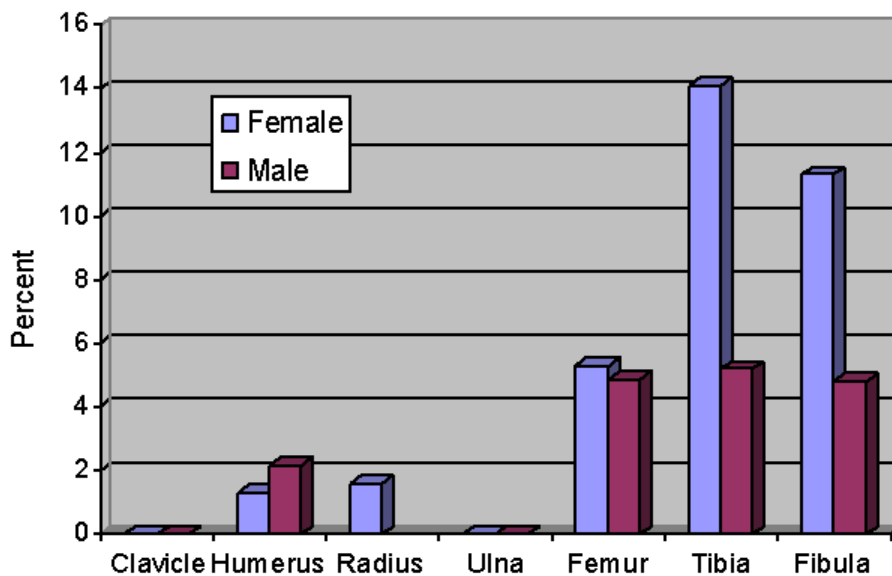


FIGURE 129. Frequencies of Periosteal Lesions in Females and Males at CA-CCO-18/548.

## BIOLOGICAL DISTANCE ANALYSIS

“Bioarchaeology is the contextual study of the biology, culture, and evolution of human populations using skeletal remains interpreted within archaeological, historical, and contemporary problem orientations” (Stojanowski and Schillaci 2006:49). An important component of bioarchaeological studies is the estimation of biological distance between populations or among individuals of a single population. Biodistance analyses use genetically mediated phenotypic traits of the skeleton or dentition to assess similarity between aggregates of individuals within a population and between populations (Buikstra et al. 1990; Larsen 1997). The theoretical model underlying biodistance analysis is that populations that exchange mates (and therefore genes) will become phenotypically more similar over time (gene flow). Those populations that do not exchange mates with one another will become more dissimilar over time through the process of genetic drift. The efficacy of this model depends on four assumptions: (1) that the populations observed are represented by unbiased, statistically valid samples, (2) changes in gene frequencies will result in measurable phenotypic changes, (3) environmental influences on traits are minimal or randomly distributed among the samples being studied, and (4) inheritance of the traits is due to the action of multiple genes, each with a small contribution to the phenotype and therefore similarity among relatives is strong. Researchers must take care in choosing their population samples to satisfy the first assumption. The remaining three assumptions concern questions of heritability and have been addressed by numerous studies of nonmetric traits (summarized in Sjøvold 1984; Hauser and DeStefano 1989; Scott and Turner 1997). The majority of studies indicate a strong genetic component for cranial and dental nonmetric traits.

Nonmetric traits have been recognized and cited in anthropological literature as descriptive features in human populations since the turn of the century (Chambellan 1883; Dorsey 1897; Gregory 1916; Le Double 1903, 1906, 1912; Hrdlicka 1920, 1921, 1924; Russell 1900). Nonmetric traits offer a useful tool in determining population distance (e.g., Balakrishnan and Sanghvi 1968; Ossenber 1976; Sjøvold 1973, 1984; Kellock and Parsons 1970a, 1970b; Konigsberg 1987; Suchey 1975). Population distance, as used here, is an expression of morphological similarity between two or more populations, based on a statistical treatment of selected variables. In choosing these variables, one assumes that they are (1) largely under

genetic control and (2) minimally affected by environmental or nutritional conditions. Also, the variables should be (1) reliably scored to ensure that population differences in trait frequency reflect biological not observational variance, (2) sufficiently variable to provide information to measure population distance, and (3) independent of one another in order to be useful in a multivariate analysis (Molto 1983). Furthermore, the sample must represent the population and be of adequate size (Rösing 1982, 1984). Distance measures between populations are useful in anthropology for inferring residence patterns, patterns of diffusion and migration, and microevolutionary changes through time (Ubelaker 1989).

Much time has been devoted to debating the relative usefulness of nonmetric traits as opposed to so called "metric" traits for the estimation of biological distance (e.g., Berry and Berry 1967; Carpenter 1976; Cheverud et al. 1979; Pietrusewsky 1981; Rightmire 1972). "Metric" traits, as used here, refer to linear measurements of skeletal elements or indices derived from linear measurements. Essentially, the debate has focused on the degree of environmental influence on trait expression in each of these classes of traits. As Kennedy (1981) has pointed out, existing information suggests that neither nonmetric traits nor metric data are of superior value in studies of biological relationships. Both are likely influenced by environmental factors such as climate, nutrition, maternal physiology, or biomechanical stress. More importantly, considering the probable polygenic nature of many of both classes of traits, the effect of environmental factors is probably minimal (A.C. Berry 1972; Corruccini et al. 1986; DeVilliers 1968; Kolakowski et al. 1980; Moller 1967; Nichol 1989). Interestingly, researchers have found that both sets of traits seem to vary systematically and have proposed a common developmental pathway (Benfer 1970; Cheverud et al. 1979; Corruccini 1975; Richtsmeier et al. 1984).

A key difference in the usefulness of nonmetric traits versus metric traits lies in the context of the population samples under examination. For skeletal populations derived from an archaeological context, nonmetric traits offer a distinct advantage over metric data. Nonmetric data can be collected from fragmentary or incomplete skeletons. This condition most typically represents the condition of skeletal remains from archaeological contexts. In such a situation nonmetric data represent a distinct advantage over metric measurements which often require a high degree of skeletal integrity and thereby markedly reduce sample sizes. Statistical analyses commonly used for biological distance studies typically require large skeletal samples (Rösing 1982, 1984). Owing to the generally fragmentary nature of much of the skeletal material considered here, nonmetric traits offer a crucial advantage over metric variables.

Dental and cranial morphological traits have been extensively used to assess population affinity and patterns of microevolution (e.g., Greene 1982; Haeussler et al. 1989; Lukacs and Hemphill 1991; Nichol 1989, 1990; Sofaer et al. 1986; Townsend et al. 1990; Turner 1986a, 1986b, 1987a, 1987b, 1990; Dodo 1987; Dodo et al. 1992; Ishida 1990; Ishida and Dodo 1993; Katayama 1988; Pietrusewsky 1981, 1984; Saunders and Popovich 1978; Sciulli 1990). Recent research using both types of morphological traits has focused on population microdifferentiation (Adler 2005; Coppa et al. 2007; Hallgrímsson et al. 2004; Haeussler 1996; LeBlanc et al. 2008; Lee 2007; Sutter and Mertz 2004; Weets 2004; Wroebel 2003). That is, morphological traits in recent research have been used to differentiate between local populations rather than between large, aggregate, geographically defined populations (e.g., between groups of Native Americans rather than between Native Americans and Europeans). The present study is also focused on population microdifferentiation.

In order to assess population affinity, biological distances are calculated using dental and cranial morphological data. Previous studies have demonstrated that this combined approach of including more than one source of data provides complimentary rather than redundant results (Corruccini 1974; Griffin 1993; Griffin et al. 2001; Hanihara 1992a, 1992b; Kennedy 1981; Molto 1983; Trinkaus 1978). The two sets of traits were chosen because of their demonstrated usefulness in describing population relationships. Dental morphology, or more specifically, the number, configuration, and size of cusps and other surface features of teeth, has been shown to be highly correlated with genetic ancestry below the level of reproductive population and often to the level of family group. Cranial morphology, specifically the

presence, number, and placement of ossicles, foramina, and other features of the skull, has similarly been shown to be highly correlated with genetic ancestry. Biological distance, in the sense used here, refers to a statistical expression of morphological similarity between populations that is derived from genetically controlled traits.

## **Bilateral Asymmetry**

Many nonmetric variables can occur bilaterally. That is, a trait occurring on the left side of the skeleton is usually mirrored on the right reflecting the general bilaterally symmetrical nature of vertebrates. Occasionally a trait will occur on one side only, making the expression bilaterally asymmetrical. The implications of asymmetrical trait expression are unclear. Studies with human subjects have come to two disparate conclusions regarding the cause of bilateral asymmetry. Sellevold (1980) and Axelsson and Hedegard (1981) studied mandibular tori in prehistoric Norse and historic Eskimo, and Icelandic school children, respectively. Both studies concluded that unilateral expression was primarily the result of environmental stress. On the other side of the issue, Noss et al. (1983) and Mayhall and Saunders (1986), in studies among Pima Indians and Eskimos, found that there was no conclusive evidence for environmental stress as the causal agent in bilateral trait expression in molar teeth. The differences in these studies may simply reflect the relative genetic stability of the human dentition (see Sofaer et al. 1986).

Each tooth in the normal human dentition exists in two copies. That is, each tooth on the left side is duplicated on the right. Scott (1980) outlines three commonly employed counting procedures used for dental morphologic traits. The first procedure, total tooth count, scores all antimeres for each trait (Hellman 1929; Dahlberg 1963; Sofaer et al. 1972; Harris 1977). This method tends to inflate sample size unnecessarily. For the type of statistical analyses typically used for nonmetric dental traits, it is desirable not to artificially inflate the sample by counting both antimeres. Some researchers have chosen to overcome this problem by scoring only one side of the dentition in each individual. This procedure, referred to as the unilateral count method, restricts scoring entirely to right or left antimeres (Smith 1977; Goose 1977; Kirveskari 1977; Axelsson and Kirveskari 1977). As some teeth will not be observable for various reasons (e.g., tooth loss, attrition, carious lesions), this approach tends to reduce sample size. This method seems to unnecessarily restrict the sample size, although some authors (e.g., Zubov and Kahldeyeva 1979) have surmounted this difficulty by observing the antimeres if the tooth on the selected side is missing. Turner and Scott (1977) have recommended observation of both antimeres, and the recording of the higher of the two scores as the observation for that individual. This approach, referred to as the individual count method, assumes that the member of a pair of asymmetric antimeres that exhibits the greatest degree of trait expression best represents the genetic potential for that character in that individual (Turner 1967; Turner and Scott 1977; Scott 1977, 1980). Turner and Scott's procedure eliminates any artificial inflation of the sample while at the same time maximizing the available information.

For the present investigation, 20 of the cranial nonmetric traits are bilateral (occurring on left and right sides). For bilateral traits, presence on either side is considered "present". This method was chosen instead of "total side frequency" (Ossenberg 1976). Researchers that favor the individual scoring method used here rather than "total side frequency" (e.g., Korey 1970; Buikstra 1972; Birkby 1973; Suchey 1975; Saunders 1978) point out that (1) it is more realistic to treat individuals, rather than sides, as members of a population (2) because of the age-dependency of the proportion of bilateral occurrence, the side method exaggerates the effect of age-regression in variant incidence, and (3) the side method artificially inflates sample size and introduces redundant information deriving from the strong positive correlation in trait frequency between sides.

## Dental Traits

Morphological observations were made on 100 dental crown and root variables using the standardized Arizona State University (ASU) dental anthropology system (Turner et al. 1991). This system is comprised of a wide range of dental morphological variables, including multitooth expression of a single trait and traits involving a single pair of antimeres. This information facilitates the identification of the most variable tooth or site for trait expression. The ASU system has proved particularly useful for distinguishing between local populations as well as larger regional series (Turner 1985; Scott and Dahlberg 1982). Most of the traits in this study are observed using ordinal scales with several grades. The dental morphological traits used in the present study are summarized in Table 90. Because of the lack of adequate comparative data, the dental traits were not used in the calculation of biological distance. Dental morphological traits were scored along a continuum of expression in each population sample using the methodology provided by Turner et al. (1991). Dental morphological trait frequencies are presented in Table 91.

## Cranial Traits

Morphological observations were made on 25 nonmetric cranial traits described by Hauser and De Stefano (1989). Numerous nonmetric cranial traits have been described in the literature, but these descriptions generally lack information on the reliability of specific traits in describing and comparing populations. This creates a situation where the researcher must rely to a large degree on precedents set by other workers or on personal preference. The traits selected from this study were chosen on the basis of three criteria: (1) reliability of scoring and observation, (2) demonstrated ability to discriminate populations, and (3) low intercorrelation with sex and age. The 25 traits included in this study are listed in Table 92. The traits used here were observed recording presence or absence of the trait. No ordinal scales are used because these tend to be highly subjective (see Kennedy 1981). Cranial morphological traits were recorded as present or absent, with present representing any degree of trait expression. Cranial morphological trait frequencies are presented in Table 93.

## Trait Intercorrelation

With the large number of genetic and non-genetic factors influencing the expression of nonmetric traits, the number of traits which are statistically correlated is expected to be low. Research has shown this presumption to be generally false (Buikstra 1972; Corruccini 1974; DeVilliers 1968; Molto 1983; Ossenberg 1976; Suzuki and Sakai 1960). Molto (1983) attributes the higher than expected frequencies of correlations to four major factors: nonmetric traits (1) are often alternative expressions of a single underlying variable, (2) often have a common regional or embryological origin, (3) can be affected by similar developmental phenomenon, and (4) may be affected by the shared interaction of some combination of the above. Given these factors, frequencies of nonmetric traits are expected to show a considerable number of positive correlations. Arguably, nonmetric traits offer redundant information in the real population.

Many authors have recommended the use of the phi coefficient rather than other coefficients to detect the correlations between nonmetric traits in place (Benfer 1970; Molto 1983; Sjøvold 1977). Another related statistic, Tau-b (Goodman and Kruskal 1954, 1959, 1963), is used here because many of the traits are not exclusively dichotomous, as required for phi correlation analysis. Tau-b, like phi, gives a close approximation to the chi-square distribution, and therefore is more sensitive to this task than other coefficients of association (Thomas 1986). Correlation coefficients were calculated for both classes of traits in this study.



TABLE 90. Dental Morphological Traits.

| TRAIT                 | TEETH OBSERVED           | REFERENCES   |
|-----------------------|--------------------------|--|
| winging               | upper central incisors   | Enoki and Dahlberg 1958; Dahlberg 1963; Scott 1973                   |
| shoveling             | upper incisors           | Hrdlicka 1920; Dahlberg 1956, 1963; Moorrees 1957; Scott 1973        |
| curvature             | upper central incisors   | Nichol et al. 1984   |
| double-shoveling      | upper central incisors   | Dahlberg 1956; Turner 1967   |
| interruption groove   | upper lateral incisors   | Turner 1967  |
| metacone              | upper 3rd molars         | Turner et al. 1991   |
| hypocone              | upper 1st and 2nd molars | Larson 1970, 1978; Scott 1973; Turner et al. 1991                    |
| metaconule            | upper 1st and 2nd molars | Harris 1977; Harris and Bailit 1980                                  |
| Carabelli's trait     | upper 1st and 2nd molars | Dahlberg 1956, 1963; Scott 1973, 1980                                |
| parastyle             | upper 3rd molar          | Katich 1975; Turner et al. 1991                                      |
| peg-shaped incisor    | upper lateral incisor    | Turner et al. 1991   |
| peg-shaped molar      | upper 3rd molar          | Turner et al. 1991   |
| anterior fovea        | lower 1st molar          | Hrdlicka 1924; Turner et al. 1991                                    |
| groove pattern        | lower 1st and 2nd molars | Gregory 1916; Hellman 1929; Jorgensen 1955                           |
| cuspid number         | lower 1st and 2nd molars | Gregory 1916; Hellman 1929; Turner 1967                              |
| deflecting wrinkle    | lower 1st molars         | Weidenreich 1937; Scott and Dahlberg 1982; Turner et al. 1991        |
| distal trigonid crest | lower 1st molars         | Hrdlicka 1924  |
| protostylid           | lower 1st and 2nd molars | Dahlberg 1956, 1963; Turner 1967; Scott 1973                         |
| cuspid 5              | lower 1st and 2nd molars | Turner 1970; Scott and Dahlberg 1982; Turner et al. 1991             |
| cuspid 6              | lower 1st and 2nd molars | Turner 1970; Scott 1973; Scott and Dahlberg 1982; Turner et al. 1991 |
| cuspid 7              | lower 1st and 2nd molars | Turner 1970; Turner et al. 1991                                      |

TABLE 91. Dental Nonmetric Trait Frequencies.

| TRAIT                 | GRADE (N, %PRESENT) |           |           |           |           |           |           |          |         |
|-----------------------|---------------------|-----------|-----------|-----------|-----------|-----------|-----------|----------|---------|
|                       | 0                   | 1         | 2         | 3         | 3.5       | 4         | 5         | 6        | 7       |
| shoveling I1          | 0 (0.0)             | 2 (3.8)   | 7 (13.2)  | 27 (50.9) | —         | 9 (17.0)  | 3 (5.7)   | 5 (9.4)  | —       |
| shoveling I2          | 0 (0.0)             | 2 (3.7)   | 3 (5.6)   | 6 (11.1)  | —         | 23 (42.6) | 15 (27.8) | 2 (3.7)  | 3 (5.6) |
| double shoveling      | 26 (47.3)           | 3 (5.5)   | 9 (13.4)  | 1 (1.8)   | —         | 14 (25.5) | 2 (3.6)   | 0 (0.0)  | —       |
| interruption groove   | 23 (52.3)           | 9 (20.5)  | 12 (27.3) | 0 (0.0)   | —         | 0 (0.0)   | —         | —        | —       |
| metacone M3           | 0 (0.0)             | 0 (0.0)   | 1 (1.3)   | 5 (6.4)   | 50 (64.1) | 22 (28.2) | 0 (0.0)   | —        | —       |
| hypocone M1           | 0 (0.0)             | 0 (0.0)   | 0 (0.0)   | 1 (2.4)   | 3 (7.3)   | 30 (73.2) | 7 (17.1)  | —        | —       |
| hypocone M2           | 2 (4.1)             | 1 (2.0)   | 8 (16.3)  | 18 (36.7) | 14 (28.6) | 6 (12.3)  | 0 (0.0)   | —        | —       |
| metaconule M1         | 23 (85.2)           | 2 (7.4)   | 2 (7.4)   | 0 (0.0)   | —         | 0 (0.0)   | —         | —        | —       |
| metaconule M2         | 30 (96.8)           | 0 (0.0)   | 1 (3.3)   | 0 (0.0)   | —         | 0 (0.0)   | —         | —        | —       |
| Carabelli's trait M1  | 25 (80.6)           | 0 (0.0)   | 2 (6.4)   | 2 (6.4)   | —         | 1 (3.2)   | 1 (3.2)   | 0 (0.0)  | 0 (0.0) |
| Carabelli's trait M2  | 31 (93.9)           | 0 (0.0)   | 1 (3.0)   | 1 (3.0)   | —         | 0 (0.0)   | 0 (0.0)   | 0 (0.0)  | 0 (0.0) |
| parastyle             | 47 (88.7)           | 2 (3.8)   | 2 (3.8)   | 2 (3.8)   | —         | 0 (0.0)   | 0 (0.0)   | 0 (0.0)  | —       |
| peg-shaped incisor    | 58 (98.3)           | 1 (1.7)   | 0 (0.0)   | —         | —         | —         | —         | —        | —       |
| peg-shaped molar      | 80 (100.0)          | —         | 0 (0.0)   | —         | —         | —         | —         | —        | —       |
| anterior fovea        | 1 (7.7)             | 1 (7.7)   | 1 (7.7)   | 7 (53.8)  | —         | 3 (23.1)  | —         | —        | —       |
| groove pattern M1     | —                   | 24 (85.7) | 1 (3.6)   | 3 (10.7)  | —         | —         | —         | —        | —       |
| groove pattern M2     | —                   | 4 (11.4)  | 5 (14.3)  | 26 (74.3) | —         | —         | —         | —        | —       |
| cusp number M1        | —                   | —         | —         | —         | —         | —         | 24 (82.8) | 5 (17.2) | —       |
| cusp number M2        | —                   | —         | —         | —         | —         | —         | 20 (69.0) | 7 (24.1) | —       |
| deflecting wrinkle    | 2 (28.6)            | 2 (28.6)  | 2 (28.6)  | 1 (14.3)  | —         | —         | —         | —        | —       |
| distal trigonid crest | 7 (100.0)           | 0 (0.0)   | —         | —         | —         | —         | —         | —        | —       |
| protostylid M1        | 5 (17.9)            | 7 (25.0)  | 14 (50.0) | 1 (3.6)   | —         | 1 (3.6)   | 0 (0.0)   | 0 (0.0)  | —       |
| protostylid M2        | 6 (25.0)            | 5 (20.8)  | 10 (41.7) | 1 (4.2)   | —         | 0 (0.0)   | 1 (4.2)   | 1 (4.2)  | —       |
| cusp 5 M1             | 0 (0.0)             | 0 (0.0)   | 3 (10.3)  | 1 (3.4)   | —         | 19 (65.5) | 6 (20.7)  | —        | —       |
| cusp 5 M2             | 3 (10.0)            | 1 (3.3)   | 6 (20.0)  | 9 (30.0)  | —         | 11 (36.7) | 0 (0.0)   | —        | —       |
| cusp 6 M1             | 23 (79.3)           | 0 (0.0)   | 3 (10.3)  | 1 (3.5)   | —         | 1 (3.5)   | 1 (3.5)   | —        | —       |
| cusp 6 M2             | 24 (80.0)           | 1 (3.3)   | 1 (3.3)   | 2 (6.7)   | —         | 1 (3.3)   | 1 (3.3)   | —        | —       |
| cusp 7 M1             | 29 (100.0)          | 0 (0.0)   | 0 (0.0)   | 0 (0.0)   | —         | 0 (0.0)   | 0 (0.0)   | —        | —       |
| cusp 7 M2             | 27 (100.0)          | 0 (0.0)   | 0 (0.0)   | 0 (0.0)   | —         | 0 (0.0)   | 0 (0.0)   | —        | —       |

TABLE 92. Cranial Morphological Traits.

| TRAIT                             | REFERENCES  |
|-----------------------------------|---|
| ossicle at lambda                 | Bennett 1965; Berry and Berry 1967; Molto 1983                  |
| lambdoid ossicles                 | Bennett 1965; Berry and Berry 1967; Hertzog 1968                |
| ossicle at asterion               | Berry and Berry 1967; Suchey 1975; Molto 1983                   |
| parietal notch bone               | Oetteking 1930; Berry and Berry 1967; Ossenberg 1969            |
| epipteric bone                    | Wood-Jones 1930; Berry and Berry 1967; Molto 1983               |
| bregmatic bone                    | Wood-Jones 1930; Berry and Berry 1967                           |
| coronal ossicle                   | Wood-Jones 1930; Sublett 1966; Berry and Berry 1967             |
| metopism                          | Limson 1924; Bolk 1931; Torgersen 1952                          |
| fronto-temporal articulation      | Collins 1926, 1930; Ossenberg 1969                              |
| supraorbital foramen complete     | LeDouble 1903; Berry and Berry 1967; Ossenberg 1969; Korey 1970 |
| frontal notch                     | Wood-Jones 1930; Berry and Berry 1967                           |
| auditory torus                    | Wood-Jones 1930; Berry and Berry 1967                           |
| foramen of Huschke                | Anderson 1962; Berry and Berry 1967; Molto 1983                 |
| condylar facet double             | Anderson 1962; Berry and Berry 1967; Kennedy 1981               |
| precondylar tubercle              | Inglemark 1947; Berry and Berry 1967                            |
| foramen ovale incomplete          | Wood-Jones 1930; Berry and Berry 1967                           |
| foramen spinosum open             | Berry and Berry 1967; Korey 1970; Suchey 1975                   |
| accessory lessor palatine foramen | Berry and Berry 1967  |
| palatine torus                    | Suzuki and Sakai 1960; Turner et al. 1991                       |
| maxillary torus                   | Berry and Berry 1967  |
| parietal foramen                  | Berry and Berry 1967; Ossenberg 1969; Molto 1983                |
| posterior condylar                | Boyd 1930; Berry and Berry 1967; Ossenberg 1969;                |
| canal patent                      | Korey 1970  |
| mastoid foramen exsutural         | Berry and Berry 1967  |
| anterior condylar canal double    | Berry and Berry 1967; Korey 1970; Ossenberg 1969; Molto 1983    |
| zygomatico-facial foramen absent  | Berry and Berry 1967; Molto 1983                                |

TABLE 93. Cranial Nonmetric Trait Frequencies.

| CRANIAL TRAIT                | N (% PRESENT)       |                     |                     |                       |            |                      |                     |                     |                      |                      |                     |                     |                     |                     |
|------------------------------|---------------------|---------------------|---------------------|-----------------------|------------|----------------------|---------------------|---------------------|----------------------|----------------------|---------------------|---------------------|---------------------|---------------------|
|                              | SAC-66 <sup>1</sup> | SIO-68 <sup>1</sup> | SAC-99 <sup>1</sup> | SAC-127R <sup>1</sup> | SAC-151    | SCRI-83 <sup>1</sup> | SIO-17 <sup>1</sup> | SIO-91 <sup>1</sup> | SIO-154 <sup>2</sup> | SMA-125 <sup>3</sup> | SOL-2C <sup>1</sup> | SOL-2L <sup>1</sup> | TEH-10 <sup>4</sup> | YOL-13 <sup>1</sup> |
| highest nuchal line          | —                   | —                   | —                   | —                     | —          | —                    | —                   | —                   | 9 (64.3)             | —                    | —                   | —                   | —                   | —                   |
| ossicle at lambda            | —                   | —                   | —                   | —                     | —          | —                    | —                   | —                   | 4 (28.6)             | —                    | —                   | —                   | 42 (19.04)          | —                   |
| lambdoid ossicle             | 39 (30.77)          | 27 (48.15)          | 17 (41.18)          | 24 (33.33)            | 27 (70.37) | 24 (25.00)           | 29 (31.03)          | 19 (47.37)          | 8 (57.1)             | 15 (73.33)           | 31 (38.71)          | 20 (35.00)          | 78 (51.28)          | 25 (40.00)          |
| ossicle at asterion          | 42 (23.81)          | 34 (14.70)          | 20 (35.00)          | 30 (20.00)            | 32 (18.75) | 28 (7.14)            | 35 (11.43)          | 21 (19.05)          | 2 (16.7)             | 14 (14.29)           | 33 (21.21)          | 20 (25.00)          | 61 (22.03)          | 30 (20.00)          |
| parietal notch bone          | 42 (23.81)          | 34 (17.65)          | 20 (40.00)          | 30 (23.33)            | 32 (31.25) | 28 (10.71)           | 35 (11.43)          | 21 (14.28)          | 0 (0)                | 14 (28.57)           | 33 (21.21)          | 20 (35.00)          | 61 (19.67)          | 30 (36.67)          |
| epipteric bone               | 40 (12.50)          | 29 (0)              | 20 (10.00)          | 28 (14.28)            | 28 (10.71) | 24 (0)               | 28 (3.57)           | 21 (9.52)           | 1 (12.5)             | 14 (7.14)            | 33 (15.15)          | 20 (15.00)          | 42 (4.76)           | 29 (3.45)           |
| bregmatic bone               | 30 (0)              | 27 (0)              | 20 (0)              | 26 (0)                | 30 (0)     | 28 (0)               | 34 (0)              | 21 (0)              | 0 (0)                | 14 (0)               | 33 (0)              | 20 (0)              | 47 (0)              | 29 (0)              |
| coronal ossicle              | 31 (0)              | 25 (0)              | 19 (0)              | 24 (0)                | 26 (0)     | 26 (0)               | 35 (0)              | 21 (0)              | 2 (20.0)             | 15 (6.67)            | 33 (0)              | 20 (0)              | 66 (1.51)           | 24 (8.33)           |
| metopism                     | 42 (0)              | 34 (0)              | 20 (0)              | 30 (0)                | 32 (0)     | 28 (0)               | 35 (2.86)           | 21 (0)              | 0 (0)                | 16 (0)               | 33 (0)              | 20 (0)              | 62 (0)              | 32 (0)              |
| fronto-temporal articulation | 40 (0)              | 26 (7.69)           | 20 (0)              | 28 (0)                | 28 (0)     | 24 (0)               | 28 (0)              | 21 (4.76)           | 0 (0)                | 14 (0)               | 33 (0)              | 20 (0)              | 41 (2.43)           | 28 (0)              |
| supraorbital foramen         | 42 (66.66)          | 34 (88.24)          | 20 (75.00)          | 30 (66.66)            | 32 (68.75) | 28 (57.14)           | 35 (77.14)          | 21 (85.71)          | 12 (75.0)            | 16 (37.5)            | 33 (69.70)          | 20 (60.0)           | —                   | 32 (81.25)          |
| frontal foramen/ notch       | 42 (47.62)          | 34 (47.06)          | 20 (55.00)          | 30 (10.00)            | 32 (25.00) | 28 (89.28)           | 35 (28.57)          | 21 (47.62)          | 11 (78.6)            | 16 (50.00)           | 33 (48.48)          | 20 (50.00)          | —                   | 32 (28.12)          |
| auditory torus               | 42 (11.90)          | 33 (21.21)          | 20 (5.00)           | 30 (10.00)            | 32 (21.87) | 28 (0)               | 35 (34.28)          | 21 (0)              | 4 (26.7)             | 16 (0)               | 33 (15.15)          | 20 (10.00)          | 135 (3.70)          | 32 (9.38)           |
| foramen of Husehke           | 42 (33.33)          | 34 (14.71)          | 20 (25.00)          | 30 (20.00)            | 32 (18.75) | 27 (55.56)           | 35 (20.00)          | 21 (14.29)          | 6 (50.0)             | 16 (25.00)           | 33 (21.21)          | 20 (10.00)          | 130 (52.30)         | 32 (25.00)          |
| condylar facet double        | 40 (2.50)           | 31 (0)              | 18 (0)              | 30 (0)                | 31 (3.22)  | 24 (0)               | 35 (0)              | 20 (0)              | 0 (0)                | 14 (0)               | 33 (0)              | 20 (0)              | —                   | 32 (0)              |
| precondylar tubercle         | 40 (10.00)          | 32 (0)              | 19 (0)              | 30 (6.67)             | 31 (16.13) | 24 (0)               | 34 (0)              | 20 (0)              | 3 (25.0)             | 15 (26.67)           | 33 (9.09)           | 20 (5.00)           | 71 (0)              | 32 (6.25)           |
| foramen ovale                | 41 (4.88)           | 31 (6.45)           | 18 (22.22)          | 29 (13.79)            | 32 (6.25)  | 24 (29.17)           | 35 (0)              | 21 (9.52)           | 0 (0)                | 14 (14.29)           | 33 (0)              | 20 (0)              | —                   | 32 (63.12)          |
| foramen spinosum             | —                   | —                   | —                   | —                     | —          | —                    | —                   | —                   | 3 (33.3)             | —                    | —                   | —                   | —                   | —                   |
| accessory palatine foramen   | 36 (50.00)          | 33 (57.58)          | 18 (55.55)          | 27 (51.85)            | 30 (46.67) | 28 (78.57)           | 32 (65.62)          | 20 (40.00)          | 10 (83.3)            | 15 (33.33)           | 33 (54.54)          | 20 (45.00)          | 66 (45.45)          | 32 (62.50)          |
| palatine torus               | 40 (0)              | 33 (33.33)          | 19 (10.53)          | 27 (0)                | 32 (3.13)  | 27 (51.85)           | 32 (12.50)          | 20 (0)              | 0 (0)                | 15 (46.67)           | 33 (21.21)          | 20 (15.00)          | 58 (20.68)          | 32 (3.12)           |
| maxillary torus              | —                   | —                   | —                   | —                     | —          | —                    | —                   | —                   | —                    | —                    | —                   | —                   | 110 (0)             | —                   |
| parietal foramen             | 42 (47.62)          | 28 (64.28)          | 18 (66.67)          | 22 (22.73)            | 31 (48.39) | 27 (85.18)           | 33 (39.39)          | 21 (57.14)          | 7 (53.8)             | 16 (68.75)           | 32 (50.00)          | 20 (50.00)          | 105 (51.42)         | 32 (62.50)          |
| posterior condylar canal     | —                   | —                   | —                   | —                     | —          | —                    | —                   | —                   | 11 (100.00)          | —                    | —                   | —                   | —                   | —                   |
| mastoid foramen exsural      | 41 (65.85)          | 30 (56.67)          | 19 (68.42)          | 30 (60.00)            | 32 (65.62) | 26 (76.92)           | 35 (65.71)          | 21 (66.67)          | 10 (71.4)            | 14 (71.43)           | 33 (65.64)          | 20 (55.00)          | —                   | 29 (55.17)          |
| mastoid foramen absent       | —                   | —                   | —                   | —                     | —          | —                    | —                   | —                   | 8 (61.5)             | —                    | —                   | —                   | —                   | —                   |
| ant. condylar canal double   | 42 (16.67)          | 31 (41.94)          | 20 (15.00)          | 30 (33.33)            | 31 (32.26) | 24 (29.17)           | 34 (32.35)          | 21 (28.57)          | 4 (33.3)             | 16 (12.5)            | 33 (36.36)          | 20 (40.00)          | 81 (23.45)          | 32 (31.25)          |
| zygomatoco-facial foramen    | 41 (87.81)          | 34 (94.12)          | 20 (65.00)          | 29 (20.69)            | 32 (75.00) | 28 (89.29)           | 33 (51.52)          | 21 (76.19)          | 14 (93.3)            | 15 (100.00)          | 33 (78.79)          | 20 (75.00)          | —                   | 32 (21.88)          |
| acc. infraorbital foramen    | 39 (25.64)          | 33 (54.54)          | 19 (47.37)          | 29 (51.72)            | 31 (32.26) | 28 (28.57)           | 32 (21.88)          | 19 (52.63)          | 5 (38.5)             | 15 (6.67)            | 33 (45.45)          | 20 (35.00)          | 55 (7.27)           | 32 (37.50)          |

<sup>1</sup>Suchey 1975

<sup>2</sup>Hoffman 1987

<sup>3</sup>Griffin *et al.* 2004

<sup>4</sup>Dickel 1980

TABLE 93. Cranial nonmetric trait frequencies

| CRANIAL TRAIT                | N (% present)     |                   |                 |                      |                      |                      |                      |                      |                      |                      |            |                      |                     |                     |                     |   |
|------------------------------|-------------------|-------------------|-----------------|----------------------|----------------------|----------------------|----------------------|----------------------|----------------------|----------------------|------------|----------------------|---------------------|---------------------|---------------------|---|
|                              | CCO-18/548 Pooled | CCO-18/548 Female | CCO-18/548 Male | ALA-328 <sup>1</sup> | ALA-329 <sup>1</sup> | BUT-233 <sup>3</sup> | BUT-294 <sup>2</sup> | BUT-496 <sup>2</sup> | CCO-138 <sup>1</sup> | CCO-139 <sup>1</sup> | COL-1      | LAN-264 <sup>1</sup> | SAC-29 <sup>1</sup> | SAC-43 <sup>1</sup> | SAC-60 <sup>1</sup> |   |
| highest nuchal line          | —                 | 9 (22.22)         | 14 (28.57)      | —                    | —                    | —                    | —                    | —                    | —                    | —                    | —          | —                    | —                   | —                   | —                   | — |
| ossicle at lambda            | —                 | 7 (57.14)         | 7 (14.29)       | —                    | —                    | 13 (16.67)           | 17 (11.76)           | 5 (20.00)            | —                    | —                    | —          | —                    | —                   | —                   | —                   | — |
| lambdoid ossicle             | 17 (76.47)        | 6 (66.67)         | 7 (85.71)       | 39 (43.59)           | 41 (39.02)           | 12 (33.33)           | 17 (23.53)           | 8 (0)                | 122 (43.44)          | 23 (26.09)           | 17 (52.94) | 27 (25.92)           | 23 (52.17)          | 33 (30.30)          | 21 (23.81)          | — |
| ossicle at asterion          | 25 (24.0)         | 8 (0)             | 13 (23.08)      | 39 (20.51)           | 51 (15.69)           | 13 (7.69)            | 16 (50.00)           | 8 (25.00)            | 135 (24.44)          | 25 (16.00)           | 20 (10.00) | 31 (16.13)           | 25 (32.00)          | 37 (13.51)          | 24 (8.33)           | — |
| parietal notch bone          | 23 (30.43)        | 4 (50.00)         | 13 (30.77)      | 38 (31.58)           | 51 (35.29)           | 13 (7.69)            | 17 (29.41)           | 8 (12.50)            | 135 (20.00)          | 25 (32.00)           | 20 (20.00) | 31 (22.58)           | 25 (32.00)          | 38 (18.42)          | 24 (16.67)          | — |
| epipteric bone               | 4 (0)             | 1 (0)             | 2 (0)           | 36 (5.56)            | 51 (13.72)           | 12 (0)               | 14 (14.29)           | 2 (0)                | 126 (9.52)           | 19 (10.53)           | 19 (15.79) | 26 (11.54)           | 23 (13.04)          | 32 (18.75)          | 19 (21.05)          | — |
| bregmatic bone               | 19 (0)            | 6 (0)             | 11 (0)          | 39 (0)               | 49 (0)               | 13 (0)               | 17 (0)               | 9 (0)                | 126 (0.79)           | 25 (0)               | 19 (0)     | 31 (0)               | 22 (0)              | 35 (0)              | 23 (0)              | — |
| coronal ossicle              | 18 (27.78)        | 6 (0)             | 10 (50.00)      | 39 (0)               | 46 (4.35)            | 13 (0)               | 17 (0)               | 9 (0)                | 120 (0.83)           | 18 (0)               | 17 (5.88)  | 27 (3.70)            | 21 (9.52)           | 27 (0)              | 20 (0)              | — |
| metopism                     | 79 (0)            | 27 (0)            | 35 (0)          | 39 (2.56)            | 52 (0)               | 13 (7.69)            | 17 (0)               | 9 (0)                | 135 (1.48)           | 26 (3.85)            | 20 (0)     | 31 (6.45)            | 25 (0)              | 27 (0)              | 24 (0)              | — |
| fronto-temporal articulation | 3 (0)             | 1 (0)             | 2 (0)           | 37 (0)               | 51 (3.92)            | 12 (0)               | 15 (0)               | 5 (0)                | 125 (0)              | 20 (0)               | 19 (5.26)  | 26 (0)               | 23 (0)              | 32 (0)              | 19 (0)              | — |
| supraorbital foramen         | 117 (35.04)       | 37 (45.95)        | 52 (36.54)      | 39 (64.10)           | 52 (73.08)           | 13 (76.92)           | 15 (80.00)           | 9 (22.22)            | 134 (78.36)          | 26 (88.46)           | 20 (85.00) | 30 (40.00)           | 24 (83.33)          | 38 (60.53)          | 24 (75.00)          | — |
| frontal foramen/ notch       | 105 (61.90)       | 31 (64.52)        | 51 (54.90)      | 39 (61.54)           | 52 (44.23)           | 13 (38.46)           | 15 (20.00)           | 8 (50.00)            | 134 (58.21)          | 26 (46.15)           | 20 (55.00) | 31 (64.52)           | 25 (64.00)          | 38 (42.10)          | 24 (4.17)           | — |
| auditory torus               | 183 (0.55)        | 59 (0)            | 79 (0)          | 39 (10.26)           | 52 (7.69)            | 13 (15.38)           | 17 (35.29)           | 10 (30.00)           | 135 (5.18)           | 26 (15.38)           | 20 (35.00) | 31 (29.03)           | 25 (8.00)           | 38 (18.42)          | 24 (16.67)          | — |
| foramen of Huschke           | 158 (10.13)       | 53 (9.43)         | 67 (4.48)       | 39 (25.64)           | 52 (38.46)           | 13 (53.85)           | 17 (35.29)           | 10 (70.00)           | 134 (35.07)          | 26 (38.46)           | 20 (30.00) | 31 (22.58)           | 25 (36.00)          | 38 (21.05)          | 24 (37.5)           | — |
| condylar facet double        | 70 (4.29)         | 18 (5.56)         | 40 (5.00)       | 35 (0)               | 49 (0)               | 10 (0)               | 10 (0)               | 3 (33.33)            | 130 (0.77)           | 25 (0)               | 20 (0)     | 28 (0)               | 23 (0)              | 38 (0)              | 24 (0)              | — |
| precondylar tubercle         | 37 (5.41)         | 9 (11.11)         | 21 (4.76)       | 34 (2.94)            | 47 (4.26)            | 8 (0)                | 9 (0)                | 1 (0)                | 130 (8.46)           | 25 (8.00)            | 20 (5.00)  | 27 (7.41)            | 23 (8.70)           | 38 (5.26)           | 24 (8.33)           | — |
| foramen ovale                | 43 (4.65)         | 13 (0)            | 22 (9.09)       | 35 (2.86)            | 48 (4.17)            | 11 (0)               | 14 (0)               | 3 (0)                | 134 (2.98)           | 26 (3.85)            | 20 (5.00)  | 27 (3.70)            | 24 (4.17)           | 38 (5.26)           | 24 (4.17)           | — |
| foramen spinosum             | 35 (17.14)        | 8 (37.5)          | 22 (4.55)       | —                    | —                    | 10 (60.00)           | 14 (21.43)           | 3 (66.67)            | —                    | —                    | —          | —                    | —                   | —                   | —                   | — |
| accessory palatine foramen   | 16 (18.75)        | 2 (0)             | 10 (20.00)      | 35 (74.28)           | 51 (66.67)           | 10 (100.00)          | 9 (66.67)            | 6 (33.33)            | 133 (69.17)          | 23 (60.87)           | 20 (75.00) | 23 (60.87)           | 23 (73.91)          | 36 (58.33)          | 21 (66.67)          | — |
| palatine torus               | 59 (6.78)         | 20 (5.00)         | 27 (22.22)      | 34 (11.76)           | 50 (24.00)           | 9 (0)                | 10 (0)               | 7 (0)                | 134 (4.48)           | 26 (7.69)            | 15 (6.67)  | 27 (7.41)            | 23 (21.74)          | 37 (5.40)           | 23 (0)              | — |
| maxillary torus              | —                 | 24 (4.17)         | 38 (0)          | —                    | —                    | 11 (0)               | 11 (0)               | 13 (0)               | —                    | —                    | —          | —                    | —                   | —                   | —                   | — |
| parietal foramen             | 25 (68.0)         | 6 (83.33)         | 13 (46.14)      | 38 (57.89)           | 48 (66.67)           | 13 (76.92)           | 17 (94.12)           | 7 (57.14)            | 133 (57.89)          | 26 (53.85)           | 18 (61.11) | 27 (66.67)           | 24 (45.83)          | 36 (50.00)          | 24 (37.50)          | — |
| posterior condylar canal     | —                 | 15 (73.33)        | 20 (75.00)      | —                    | —                    | —                    | —                    | —                    | —                    | —                    | —          | —                    | —                   | —                   | —                   | — |
| mastoid foramen exsutural    | 60 (80.0)         | 17 (70.58)        | 34 (85.29)      | 37 (62.16)           | 50 (62.00)           | 13 (84.62)           | 15 (93.33)           | 7 (100.00)           | 133 (54.14)          | 26 (73.08)           | 20 (75.00) | 30 (53.33)           | 24 (83.33)          | 37 (54.05)          | 24 (54.17)          | — |
| mastoid foramen absent       | 60 (6.67)         | 17 (11.77)        | 34 (5.88)       | —                    | —                    | 13 (38.46)           | 15 (6.67)            | 7 (0)                | —                    | —                    | —          | —                    | —                   | —                   | —                   | — |
| ant. condylar canal double   | 78 (25.64)        | 20 (30.00)        | 46 (28.26)      | 35 (37.14)           | 48 (29.17)           | 10 (50.00)           | 11 (36.36)           | 2 (0)                | 133 (30.83)          | 26 (30.77)           | 20 (30.00) | 28 (50.00)           | 23 (30.43)          | 38 (10.53)          | 24 (37.50)          | — |
| zygomatico-facial foramen    | 82 (86.59)        | 22 (81.81)        | 43 (86.05)      | 35 (77.14)           | 52 (7.69)            | 12 (0)               | 12 (8.33)            | 9 (11.11)            | 135 (15.56)          | 26 (73.08)           | 20 (85.00) | 29 (17.24)           | 23 (91.30)          | 36 (77.78)          | 24 (70.83)          | — |
| acc. infraorbital foramen    | 36 (16.67)        | 10 (20.00)        | 18 (11.11)      | 36 (41.67)           | 52 (55.77)           | 11 (27.27)           | 11 (27.27)           | 7 (28.57)            | 135 (48.89)          | 26 (42.31)           | 19 (31.58) | 29 (48.28)           | 22 (18.18)          | 38 (52.63)          | 24 (4.17)           | — |

<sup>1</sup>Suchey 1975

<sup>2</sup>Donahue 1993

The statistics commonly used to analyze nonmetric trait variation do not take into account the existence of inter-trait correlations and depend on the assumption that the traits used are not statistically correlated (Kennedy 1981). For this reason the dental and cranial traits chosen for this analysis were tested separately in pair-wise combinations via two-way contingency tables and Tau-b correlation coefficients. These analyses were performed in order to detect statistically significant and strong inter-trait correlations.

A number of strong correlations were observed between traits in this study. Without exception, these correlations result from the nature of the traits. That is, in each case either the traits involved occur on multiple teeth (i.e., field effects) or the traits are different manifestations of the same complex. An example of the latter would be central incisor curvature and central incisor double-shoveling. By definition, these traits vary inversely. Therefore, they are strongly negatively correlated.

## **Biological Distance**

The objective of conducting this biodistance analysis is to estimate the genetic similarity of the population sample from CCO-18/548 with other samples from close geographic and/or temporal proximity. The estimates of population distance will help put this sample into biological perspective in regard to previously examined sites in California. A number of different statistical procedures are employed to compare these groups. Among these procedures are: estimations of mean measures of divergence (Green and Suchey 1976; Sofaer et al. 1986), cluster analysis (Aldenderfer and Blashfield 1984); and multidimensional scaling (Kruskal and Wish 1978).

### *Distance Statistics*

Assessment of biological distance is best achieved by expressing the degree of dissimilarity between populations with a single numerical value, rather than trying to evaluate relationships on a trait by trait basis using univariate statistics (Cybulski 1975; Molto 1983). The single numerical value is calculated using multivariate statistics and is derived from the sum of the squared differences between corresponding variates of two population samples (Smith 1972). When the populations are similar, the coefficient value should be small, and when the populations are dissimilar, it should be large. Dissimilarity in population studies is equated with biological distance. The distance for population models is usually defined in terms of Euclidean distance. In other words, populations are plotted relative to one another in terms of their values for a given set of variables. Euclidean distance is the distance between the plotted positions. In the simplest case, one could describe two populations in terms of two variables, X and Y. Plotting the populations two dimensionally and measuring the distance between the two points on the graph gives one the Euclidean distance between the populations. As more variables are added, calculation of Euclidean distance becomes more complex and requires the use of distance coefficients.

Most of the distance coefficients used for nonmetric morphological data are based on C.A.B. Smith's Mean Measure of Divergence (Grewal 1962). This statistic uses an angular transformation of the original trait frequencies for each population sample being compared. The angular transformation stabilizes the variance so that sampling error does not distort the estimation of distance. This is necessary with dichotomous traits because the variance of the *sample* proportion is a function of the *population* proportion (Molto 1983; Sjøvold 1977). The mathematical foundation of the Mean Measure of Divergence as an appropriate distance measure using nonmetric data has been substantiated by Sjøvold (1977). This is the most widely utilized statistic for estimating population distance for nonmetric data (Molto 1983; Sjøvold 1977). However, use of the Mean Measure of Divergence without corrective statistics on small population sample sizes has been cautioned against (Green and Suchey 1976; Sjøvold 1977). There are a number of transformations currently used to remove the effects of small sample size (e.g., Anscombe 1948; Freeman and Tukey 1950). The usefulness of each technique is dependent on how quickly and effectively they stabilize the variance (Molto 1983).

The transformation devised by Smith (in Grewal 1962) has been widely used (Birkby 1973; Berry 1974; Buikstra 1972; Corruccini 1974; Cybulski 1972; Finnegan 1972; Gaherty 1970; Jantz 1970; Kellock and Parsons 1970a, 1970b; Lane and Sublett 1972; McWilliams 1974; Pietruszewsky 1969, 1971; Rightmire 1972). Green and Suchey (1976) have demonstrated that this transformation produces inflated variances for small sample sizes combined with small trait frequencies. Thus, the variance is not adequately stabilized and tests of significance between samples are unreliable.

Other researchers have proposed the use of alternate transformations (de Souza and Houghton 1977; Green and Suchey 1976; Sjøvold 1977). The best are those of Freeman and Tukey (1950) and Anscombe (1948). According to Molto (1983), there is little empirical difference between the two transformations. However, the Freeman and Tukey transformation is slightly more efficient at stabilizing the variance of very small proportions which are common in archaeological samples (Green and Suchey 1976; Sjøvold 1977).

The Mean Measures of Divergence (MMD) for this study were calculated using the Freeman and Tukey (1950) transformation and the method suggested by Green and Suchey (1976). The angular transformation for each trait was carried out using the formula suggested by Freeman and Tukey (1950). The standard deviation of the Mean Measures of Divergence was calculated using the method suggested by Sofaer et al. (1986). A mean measure of divergence equal to or greater than twice its standard deviation is considered to be statistically significant at the  $p < 0.05$  level (Molto 1983). When two samples have identical frequencies of each variant or sample sizes are small, the mean measure of divergence assumes a negative value (Turner and Bird 1981). As Constandse-Westermann (1972:3) points out, "lack of significance usually does point to a close association of populations". However, non-significant distance does not necessarily mean that the samples being compared are drawn from the same population (Constandse-Westermann 1972; Hiernaux 1972; Rightmire 1972; Sjøvold 1977). It is equally misleading to interpret statistically significant distances as indicating samples from different populations. As Grüneberg (1952, 1963) has noted, distances between populations may increase at a constant rate over generations due to random genetic drift.

The primary objective of this study was to estimate biological distances between population samples. This objective was best achieved by expressing the degree of divergence between the populations with a single numerical value, rather than trying to evaluate relationships on a trait by trait basis using univariate statistics (Cybulski 1975; Molto 1983). The single numerical value chosen for this analysis is the mean measure of divergence based on the method of Green and Suchey (1976) and standardized mean measures of divergence using the method of Sofaer et al. (1986).

Standardized mean measures of divergence were calculated by dividing each mean measure of divergence by its standard deviation. The standardized mean measures of divergence are more appropriate for comparison of distances among groups of populations with greatly varying sample sizes (Sofaer et al. 1986). In order to be considered statistically significant at the  $p$  is less than point zero five level, the mean measure of divergence must be at least twice its standard deviation. An examination of the distance matrix for cranial traits (Table 94) reveals that the majority of the mean measures of divergence are statistically significant. The statistically significant measures are noted in grey and constitute 61.1% of the comparisons (129/211). It is especially notable that all of the distance measures for CCO-18/548 are statistically significant. None of the comparison samples show a close affinity with CCO-18/548 regardless of time period (sites located closer to CCO-18/548 in the matrix) or geographic location. This indicates that in regard to cranial nonmetric morphology the sample from CCO-18/548 appears to be genetically dissimilar to the other comparison populations.

TABLE 94. Mean Measures of Divergence Derived from Cranial Nonmetric Data\*

|         | CCO 548 | SJO 68  | SAC 60  | SAC 66  | SAC 99  | SAC 127 | SAC 151 | SJO 17  | SJO 91  | SOL 2C  | ALA 328 | SAC 29  | SAC 43  | CCO 139 | YOL 13  | ALA 329 | SRI 83  | SOL 2L  | CCO 138 | COL 1   | LAN 264 |
|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|
| CCO 548 | —       | 4.3063  | 6.4931  | 3.8230  | 2.6072  | 6.9376  | 2.5476  | 5.7152  | 2.5470  | 2.9666  | 3.1333  | 2.2909  | 3.8348  | 4.3262  | 4.7870  | 3.4462  | 5.7823  | 1.7759  | 6.4333  | 3.2635  | 3.4633  |
| SJO 68  | 11.0701 | —       | 3.6088  | 2.2772  | 0.6730  | 4.5758  | 1.3390  | 1.4892  | 0.6000  | -0.1228 | 0.3480  | 1.0111  | 0.8531  | 0.4738  | 3.0777  | 0.0536  | 3.0699  | 0.1742  | 4.0513  | -0.0807 | 1.1501  |
| SAC 60  | 14.3535 | 7.7611  | —       | 1.0590  | 2.9831  | 1.4418  | 1.0539  | 0.4929  | 2.3767  | 1.8963  | 2.3170  | 2.7652  | 1.6538  | 1.0617  | 1.6434  | 3.0121  | 7.4976  | 1.6422  | 3.7996  | 1.4874  | 2.9984  |
| SAC 66  | 10.9986 | 6.3981  | 2.5074  | —       | 1.5055  | 3.1337  | 0.8601  | 1.6300  | 0.7762  | 0.9451  | 1.0131  | 1.7808  | 0.1867  | 0.3302  | 2.5996  | 1.4054  | 4.3583  | 0.8345  | 3.6276  | 0.3755  | 1.4150  |
| SAC 99  | 5.1509  | 1.2911  | 5.0756  | 3.1463  | —       | 1.7853  | 0.6261  | 1.5919  | -0.6256 | 0.1504  | -0.3758 | 0.3983  | -0.0531 | -0.4126 | 0.5142  | 0.1293  | 2.3104  | -0.3099 | 1.1790  | 0.2004  | 1.0460  |
| SAC 127 | 16.8752 | 10.8603 | 2.9578  | 8.2834  | 3.2831  | —       | 1.3954  | 1.3649  | 1.5582  | 2.2509  | 2.7095  | 4.7544  | 1.6159  | 1.7117  | 0.2223  | 4.0798  | 9.4484  | 1.6936  | 1.4526  | 3.2497  | 3.6107  |
| SAC 151 | 6.5249  | 3.3383  | 2.2545  | 2.4004  | 1.1955  | 3.2950  | —       | 1.0645  | 0.4570  | 0.1355  | 0.6052  | 1.1151  | 0.4731  | 0.3548  | 1.0768  | 1.3081  | 6.4097  | -0.1656 | 2.6244  | 0.0097  | 1.3917  |
| SJO 17  | 15.3160 | 3.8797  | 1.0945  | 4.7788  | 3.1409  | 3.3581  | 2.7554  | —       | 1.5371  | 0.3399  | 0.8373  | 1.9825  | 0.6713  | 0.2549  | 0.9174  | 2.1638  | 5.7632  | 0.3379  | 2.0100  | 0.2875  | 1.5494  |
| SJO 91  | 5.2635  | 1.2019  | 4.2008  | 1.7001  | -1.0054 | 2.9870  | 0.9109  | 3.1698  | —       | 0.2149  | 0.2214  | 1.3072  | 0.0981  | -0.0484 | 1.0569  | 0.7880  | 4.1042  | -0.1210 | 1.6032  | 0.5356  | 1.5237  |
| SOL 2C  | 7.8673  | -0.3158 | 4.1648  | 2.7308  | 0.2939  | 5.4721  | 0.3462  | 0.9079  | 0.4386  | —       | -0.5505 | 0.2232  | -0.2308 | -0.5116 | 1.2542  | -0.0934 | 3.3845  | -1.1725 | 1.8777  | -0.2683 | 0.0582  |
| ALA 328 | 8.7946  | 0.9469  | 5.3373  | 3.1176  | -0.7660 | 6.9466  | 1.6353  | 2.3701  | 0.4724  | -1.5368 | —       | 0.0643  | 0.0416  | -0.4477 | 1.2093  | -0.0787 | 2.3415  | -0.7511 | 1.5155  | -0.3904 | -0.1726 |
| SAC 29  | 5.1033  | 2.1857  | 5.2273  | 4.2412  | 0.6805  | 9.8038  | 2.3981  | 4.4256  | 2.3201  | 0.4928  | 0.1490  | —       | 1.5586  | 0.0068  | 2.4843  | 0.2898  | 2.4108  | 0.3857  | 3.3816  | -0.6212 | 1.6503  |
| SAC 43  | 10.5805 | 2.2964  | 3.7778  | 0.5679  | -0.1075 | 4.1027  | 1.2656  | 1.8831  | 0.2080  | -0.6383 | 0.1223  | 3.5808  | —       | -0.0945 | 1.3440  | 0.4355  | 4.0569  | -0.2823 | 2.1012  | 0.1036  | 0.3691  |
| CCO 139 | 9.8734  | 1.0541  | 2.0580  | 0.8115  | -0.7216 | 3.6281  | 0.7852  | 0.5867  | -0.0880 | -1.1640 | -1.0704 | 0.0133  | -0.2236 | —       | 0.4899  | -0.0710 | 3.2638  | -0.3488 | 1.3019  | -0.7553 | 0.4628  |
| YOL 13  | 12.1584 | 7.6216  | 3.4960  | 7.2023  | 0.9772  | 0.5217  | 2.6513  | 2.3593  | 2.0961  | 3.1844  | 3.2462  | 5.3141  | 3.5697  | 1.0777  | —       | 2.2041  | 6.6999  | 0.7042  | 0.1056  | 1.2778  | 2.3461  |
| ALA 329 | 10.8160 | 0.1644  | 7.6761  | 4.9629  | 0.2881  | 11.7052 | 3.9829  | 6.9484  | 1.8461  | -0.2953 | -0.2667 | 0.7430  | 1.4582  | -0.1884 | 6.6573  | —       | 2.2687  | 0.1417  | 3.2793  | 0.0045  | 0.8061  |
| SRI 83  | 13.7872 | 7.0833  | 15.0038 | 11.1594 | 4.1531  | 20.7358 | 14.7080 | 13.7606 | 7.6815  | 7.9853  | 5.8126  | 4.8467  | 9.9938  | 6.7440  | 15.2856 | 6.2846  | —       | 3.8632  | 5.9727  | 2.9691  | 3.3287  |
| SOL 2L  | 3.6146  | 0.3432  | 2.8607  | 1.7951  | -0.4915 | 3.1957  | -0.3247 | 0.6851  | -0.1989 | -2.3534 | -1.5745 | 0.6746  | -0.5879 | -0.6248 | 1.3740  | 0.3257  | 7.1201  | —       | 1.7096  | 0.1650  | -0.3364 |
| CCO 138 | 26.1869 | 16.3855 | 12.1176 | 17.7477 | 3.1869  | 5.3855  | 10.5171 | 8.6443  | 4.6070  | 7.9060  | 7.0028  | 10.8682 | 9.5565  | 4.3687  | 0.4188  | 18.7882 | 21.1737 | 4.7981  | —       | 2.3579  | 2.9658  |
| COL 1   | 6.4380  | -0.1547 | 2.5275  | 0.7833  | 0.3109  | 5.9659  | 0.0185  | 0.5665  | 0.8598  | -0.5235 | -0.7947 | -1.0599 | 0.2094  | -1.3188 | 2.4244  | 0.0100  | 5.3350  | 0.2615  | 6.3592  | —       | 0.2399  |
| LAN 264 | 8.5381  | 2.7600  | 6.2042  | 3.7821  | 1.9380  | 8.2189  | 3.3200  | 3.8514  | 2.9435  | 0.1430  | -0.4469 | 3.4321  | 0.9478  | 0.9900  | 5.5649  | 2.3416  | 7.3689  | -0.6396 | 11.1724 | 0.4440  | —       |

\* Mean measures of divergence above diagonal, standardized mean measures of divergence below diagonal



## *Taxonomic Statistics*

Interpreting biological relationships from a large matrix of distance coefficients can be a quite confusing task. In order to make interpretation easier, two related taxonomic statistical techniques have traditionally been employed (Lukacs and Hemphill 1990; Molto 1983). These two techniques are cluster analysis and multidimensional scaling. Both of these procedures express biological dissimilarity in terms of Euclidean distance (Molto 1983).

*Cluster analysis.* Affinity of the groups is assessed using cluster analysis, a metric approach leading to the establishment of clusters of similar groups (Aldenderfer and Blashfield 1984; Anderberg 1973; Blashfield 1976; Everitt 1974). The object of the analysis is: given a sample of  $n$  objects, each of which has a score on  $p$  variables, devise a scheme for grouping the objects into classes so that similar ones are in the same class. The objects here are the population samples and the scores are the arcsine transformed trait frequencies. The method must be completely numerical and, unlike discriminate function analyses, the number of classes is not known.

There are a number of hierarchical methods available for cluster analysis. These methods operate on a distance matrix to construct a dendrogram which illustrates the relationships among the population samples. Agglomerative hierarchical methods in cluster analysis start with the calculation of the distances of each individual to all other individuals. Groups are then formed by a process of agglomeration. All objects start by being alone in groups of one. Close groups are then gradually merged until finally all individuals are in a single group. Of the methods available, Ward's minimum variance provides the most accurate results for the type of data used here (Blashfield 1976; Molto 1983). His method is designed to generate clusters so that the variance within clusters is minimal (Ward 1963). The procedure uses an error sum of squares function which computes the sum of squares of the distance from each point to its parent cluster. At each step, it combines those two clusters which result in the least increase in the within group sum of squares objective function. A cluster formed by this method can, therefore, be defined as a group of entities such that the error sum of squares among the members of each cluster is minimal (Blashfield 1976).

For the cluster analyses, arcsine transformed trait frequencies were used as input for a cluster analysis program (Cluster, SPSS 1997a). This program is designed to construct dendrograms in Euclidean space based on Ward's Minimum Variance method (Ward 1963). The results of cluster analyses are reported in Figures 130 and 131. The results of the cluster analyses in large part mirrors the distance statistics calculated from mean measures of divergence. That is, none of the population samples show particular affinity for one another. All of the joining distances (indicated by the relative length of the joining lines) are relatively large. As with the MMD analysis, the indication from this analysis is of a group of populations that show marked dissimilarity to one another. It is especially notable that CCO-18/548 is once again quite morphologically distinct from the majority of the rest of the samples. It is equally notable however that as Suchey (1975) noted in her analysis of nonmetric cranial traits in the Central Valley, the sites overall do not cluster systematically by time period or geographic location.

*Multidimensional Scaling.* The next procedure used to illustrate population distances is multidimensional scaling (Kruskal and Wish 1978; Schiffman et al. 1981; Torgerson 1952). Multidimensional scaling is a technique that attempts to position objects in space according to distance measures rather than classify them as in cluster analysis. The "objects" in this case are the population samples and the distances used are the standardized mean measures of divergence derived with the Freeman-Tukey transformation and using the method of Sofaer et al. (1986). A point is usually specified in terms of its coordinate location in reference to a set of axes. An axis defines a direction of movement and the number of axis defines the dimensionality of the space. The reference axes are assumed to be at right angles to one another and can be referred to as a Cartesian coordinate system (Molto 1983).

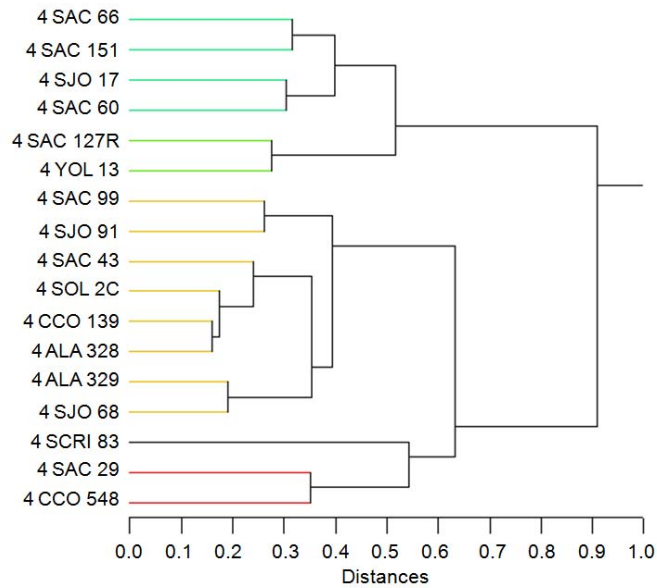


FIGURE 130. Cluster Analysis for Nonmetric Cranial Traits (Early, Middle, and Middle/Late Horizon Sites).

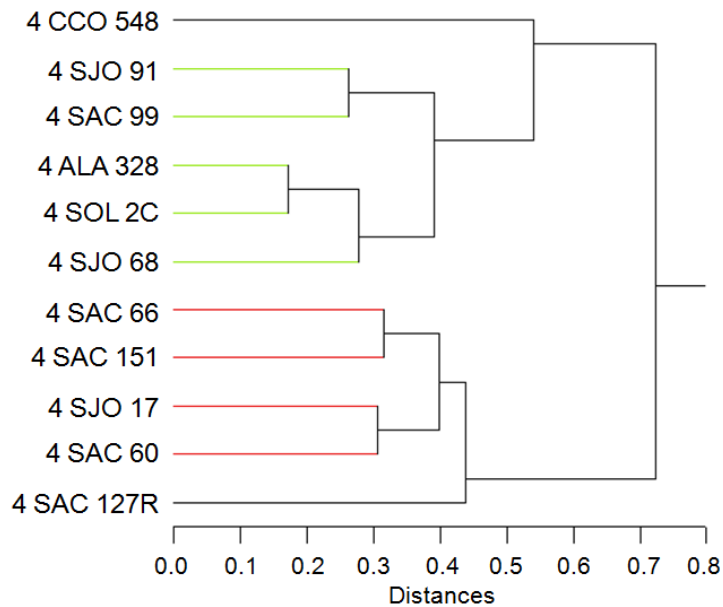


FIGURE 131. Cluster Analysis for Nonmetric Cranial Traits (Early and Middle Horizon Sites).

The procedure for multidimensional scaling is iterative and the groups are moved around within a space of specified dimensionality in order to find a monotone function expressing the original distances and the distances in the configuration. The fit between the two distances and a monotone function is expressed as a measure referred to as "stress" (Kruskal 1964a, 1964b). The stress is computed as the square root of the sum of the squared deviations of the distances in the configuration space from the monotone function divided by the sum of the squares in the configuration space (Kruskal 1964a, 1964b). This statistic has a theoretical range from 0 to 1 with the larger the value the weaker the fit of the data to a given configuration. After a series of iterations has produced a configuration of minimal stress in some number of dimensions the procedure is terminated. Generally, increasing the number of dimensions improves the fit of the data to a configuration. However, beyond three dimensions, interpretation becomes problematic.

In multidimensional scaling, the position of the objects in space can be described in one dimension (if the objects fall on a line), in two dimensions (if the objects lie on a plane), in three dimensions (if the objects can be represented by points in space), or in a higher number of dimensions (in which case an immediate geometrical representation is not possible). In this study, the representations will be in three dimensions because interpretation of more dimensions is problematic at best.

For the multidimensional scaling analysis, arcsine transformed trait frequencies were utilized in a mean measure of divergence analysis. The standardized mean measures of divergence were used as input for a multidimensional scaling analysis (MDS-Guttman, SPSS 1997a). Guttman's (1968) coefficient of alienation was used. Each analysis was stopped when a level of stress in fitting the coordinate points to the monotonic function dropped below a stress level of zero. The output from this program results in a table of three-dimensional co-ordinates in Euclidean space for each sample. These co-ordinates are then plotted in three-dimensional space giving a representation of the relative distances between populations (SPSS 1997b). The results of multidimensional scaling analyses are reported in Figures 132 and 133. The results of multidimensional scaling largely correspond to the previous taxonomic technique. That is, the marked dissimilarity between the population samples produces a widely dispersed plot with very few close relationships indicated. Examination of Figure 132 indicates this same pattern. However, a somewhat different pattern is revealed when only population samples within a thirty-mile radius of CCO-18/548 are included (Figure 133). The distribution of sites in Figure 133 indicates that while the population samples are all morphologically dissimilar compared to one another, sites that are geographically contiguous tend to cluster more closely together. This may support the probability of mate exchange between nearby communities.

## **Discussion**

Tremendous biological variability has long been attributed to the early Native American inhabitants of the Central Valley (Fenenga 1939; Newman 1957; McHenry 1969; Suchey 1975). Fenenga (1939) first suggested the presence of significant differences between population samples from different cultural horizons based on his analysis of cranial and nasal indices. Newman (1957) came to similar conclusions based on other metric analyses of the cranium. McHenry (1969) confirmed both author's previous findings using Mahalanobis  $D^2$  and multivariate discriminate analysis. Both Newman and McHenry attributed the distinctive dissimilarity between the samples to in-migrations of new people to the area, however neither went so far as to suggest the complete replacement of earlier populations. Suchey (1975) also found marked diversity between central California population samples based on the analysis of nonmetric cranial data. She found marked diversity not only between cultural horizons but also between sites attributed to the same horizon. She attributed some of this diversity to isolation by distance due in part to the sedentary life style of central California populations. Suchey asserts that the hunting and gathering subsistence system that persisted up to European contact was so efficient that it essentially prevented the diffusion of agriculture into the region and supported a very high population density. The maintenance of sedentary population centers with well-defined family ownership of local territories was

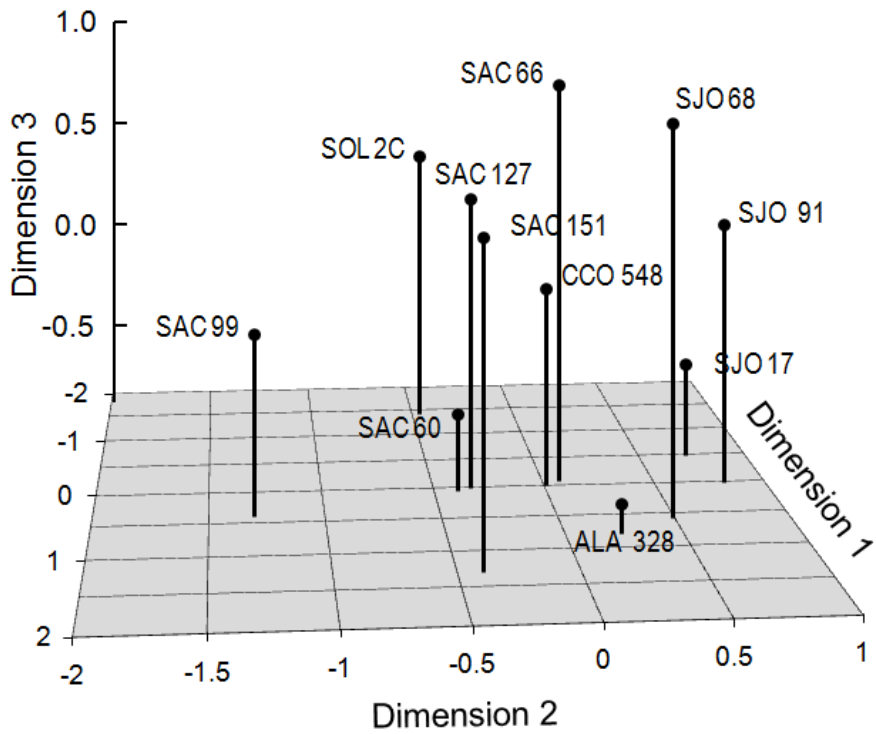


FIGURE 132. Multidimensional Scaling Analysis for Nonmetric Cranial Traits (Early and Middle Horizon Sites).

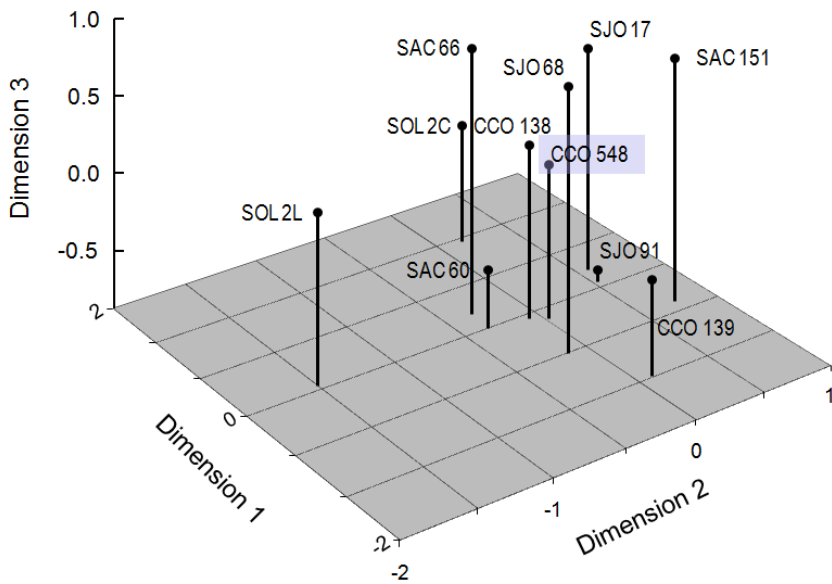


FIGURE 133. Multidimensional Scaling Analysis for Nonmetric Cranial Traits (Sites within 30-mile Radius of CA-CCO-18/548).

documented by Kroeber (1925). This pattern would have encouraged long periods of endogamous marriage that would inevitably result in the formation of population isolates due to random mutation and random genetic drift. Similar to Newman and McHenry, Suchey (1975) also suggested that an appreciable portion of the diversity seen in central California is likely due to episodes of the Founder Effect with the influx of new populations from other areas.

The results of the present biological distance analysis in large part mirrors results found in previous studies. The estimated distances between populations is quite large and taxonomic diagrams based on those distances reveal very few close relationships. This may lend further support to the contention that precontact California Native American groups practiced a relatively strict form of endogamy. However, another possibility must also be considered. Genetic drift is not just a phenomenon of geographic separation but also can be due to temporal separation. The difficulties presented by the inadequate California chronologies force researchers to place population samples in extremely long time sequences with little meaningful internal separation of those large temporal associations. In other words, different populations are grouped together in time brackets of several thousand years. It is inevitable, when dealing with population samples at opposite ends of the time bracket, that morphological change will occur due to the combination of small population size and considerable temporal depth. Both of these result in genetic drift and complicate the delineation of biological relationships. This complication can only be resolved by carefully correlating precise dates to individuals within sites and using population samples controlled in this manner for examinations of biological distance.

Griffin MC, Snyder J, Balabuszko R, Entriken K, Wiberg R. 2010. Demography, health, and regional biodiversity. In: Wiberg R, editor. Archaeological investigations at CA-CCO-18/548: Final report for the Vineyards at Marsh Creek Project, Contra Costa County, California. San Francisco: Holman and Associates Archaeological Consultants p 355-408.

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