Biocultural Implications of Oral Pathology in an Ancient Central California Population

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ABSTRACT   Bioarchaeologists have long noted two unusual trends in the dentitions of prehistoric Native Californian populations: high rates of wear and low prevalence of caries. The Central California site of CA-CCO-548 offers a unique opportunity to examine the relationship between oral pathology and extreme dental wear in a large ($n = 480$), ancient (4,300–3,100 BP), and temporally well-defined population sample. This study specifically examines three interrelated processes of the oral cavity in this population: dental wear, dental caries, and periodontal disease. The results show high levels of dental wear (average of 6.1, Smith system), low frequencies of carious lesions (2.5%), low frequencies of periodontal disease (17.8%), and high frequencies of periapical abscesses (10.7%). The pathological processes examined here have complicated multifactorial etiologies. However, they all share the common primary etiological agents of facultative pathogenic bacteria proliferation in the oral biofilm. Integration of the current etiological explanations for infections of the oral cavity, information from the ethnographic record pertaining to subsistence and activity patterns in Native Californian populations, and statistical analysis of specific disease and wear patterns leads to a novel explanation for the observed pattern of oral pathology in this population sample. Specifically, the introduction of antibacterial compounds through dietary items and non-alimentary tooth use is suggested as the most likely explanation for the unusually low prevalence of dental caries and periodontal disease. Am J Phys Anthropol 154:171–188, 2014.   © 2014 Wiley Periodicals, Inc.

Two pervasive features noted among precontact California Native American populations are extreme dental wear (Leigh, 1928; Molnar, 1971a and b; Walker, 1978; Reinhardt, 1983; Jurmain, 1990; Bartelink, 2006; Grant, 2010; Blake, 2011) and remarkably low prevalence of dental caries (Schulz, 1981; Walker and Erlandson, 1986; Hoffman, 1987; Jurmain, 1990; Bartelink, 2006). Precontact individuals from California sites routinely exceed the highest score for the various dental wear systems (Reinhardt, 1983; Jurmain, 1990). Reinhardt (1983) noted that dental wear in precontact California individuals is so extreme that it often obliterates the crown of the tooth. This creates a condition where the tooth roots are functioning in occlusion. Individuals exhibiting this wear pattern continued to masticate using the remaining root stubs, frequently wearing them down to the alveolar margin. Interpretations vary as to the causative factors involved in this extreme wear. Explanations include the unintentional inclusion of grit and stone from food processing (Jurmain, 1990), food sources with natural abrasive inclusions (Walker, 1978), and non-alimentary dental wear from producing baskets and cordage (Molnar, 1971b, 1972; Schulz, 1977; Grant, 2010; Blake, 2011).

Extraordinarily low prevalence of dental caries is also a ubiquitous feature of precontact California Native populations (Schulz, 1981; Walker and Erlandson, 1986; Jurmain, 1990; Bartelink, 2006). This observation is remarkable considering the shift in many parts of California to a carbohydrate-rich, plant-intensive economy in the Middle Holocene (Rosenthal et al., 2007). Precontact California populations never adopted agriculture as was seen in many parts of precontact North America. The transition in California was more of a move from hunting-gathering to a pattern of foraging and "tending."

That is, while agricultural plants were never adopted in California, the careful management of acorn producing oak groves was a dominant feature of the culture beginning around 4500 BP (Basgall, 1987; Beaton, 1991; McCarthy, 1993; Rosenthal et al., 2007). The catalyst for this transition was likely the increasing aridity of the environment caused by the Altithermal. The increasing aridity would have in turn caused populations that retained a hunter-gatherer economy to exceed the carrying capacity of their environment (Basgall, 1987). Acorns provided a more reliable alternative to other available resources (Tushingham and Bettinger, 2013).

Extreme wear has been suggested as a possible contributory cause for the low prevalence of caries in precontact California populations (Jurmain, 1990; Bartelink, 2006), as well as low caries prevalence observed in populations outside of California (Powell, 1985; Maat and Van der Velde, 1987; Kerr et al., 1990; Newman, 1999; Caglar et al., 2007; Buzon and Bombak, 2010). Researchers however, have come to differing conclusions regarding the relationship between wear and caries. Some researchers have suggested that the elimination of occlusal fissures on the posterior dentition

through excessive dental wear reduces the prevalence of caries (Powell, 1985; Newman, 1999). These fissures are a primary location of the oral biofilm, which in turn is one of the essential etiological components of caries (Macek et al., 2003; Ferreira Zandona et al., 2012). Other research has indicated a synergistic relationship between wear and caries (Hardwick, 1960; Miles, 1969; Silverstone et al., 1981). Increased attrition has the capacity to expose the softer dentin to cariogenic bacteria and therefore predispose individuals to higher frequencies of carious lesions. Alternatively, Meiklejohn and coworkers (1992) concluded that there is no causal relationship between caries and dental wear. They suggest that the processes are independent of one another.

The aim of this study is to examine the relationship between extreme dental wear and oral pathology in the ancient California population sample from CA-CCO-548. This will be done by assessing the prevalence of dental caries and periodontal disease in this sample and statistically comparing prevalence of these oral pathologies to levels of dental wear. Patterns of oral pathology are assessed in the context of recent advances in the understanding of oral disease etiologies. CA-CCO-548 offers the unique opportunity to examine the potential interaction between extreme dental wear and caries prevalence in a well-documented precontact California population. Research at the site has yielded extensive information pertaining to skeletal biology, paleobotany, zooarchaeology, material culture, and environmental context (Grant, 2010; Wiberg, 2010; Guidara, 2012; Eerkens and Bartelink, 2013). Based on previously reported patterns of dental wear and caries in precontact California, it is hypothesized that there will be a statistically significant relationship between high levels of wear and low prevalence of dental caries.

ORAL PATHOLOGY AND DENTAL WEAR

It is estimated that only 10% of the cells in the human body are mammalian (Marsh et al., 2011). The remaining 90% are primarily comprised of the resident human microflora that live in and on our body (Wilson, 2005). Most of the nearly 1200 indigenous species of microflora that inhabit the human oral cavity are commensal (Jenkinson, 2011). That is, most of our oral microfloras are not harmful and some are actually beneficial. These microorganisms live in normally stable microbial communities of viruses, mycoplasma, bacteria, Archaea, fungi and protozoa collectively referred to as biofilm (Meurman et al., 2004; Marsh and Martin, 2009; Palmer, 2014). A biofilm is a three-dimensional structured microorganism community with a complex web of fluid channels for transport of substrate, waste products, and signal molecules (Costerton, 1999; Scheie and Peterson, 2004). The oral microflora are initially introduced through salivary transmission associated with intimate human contact and establish themselves on the hard, non-desquamating surfaces of the oral cavity by 19–31 months of age (Caufield et al., 1993). In the normal individual there is a delicate balance between commensal and facultative pathogenic microflora of the oral cavity (Liljemark and Bloomquist, 1996; Kidd and Fejerskov, 2004; Palmer, 2014). Oral microorganisms dynamically adapt to changes in their immediate environment by using various mechanisms such as altering their gene expression (Forng et al., 2000). This capacity allows these protean communities to maintain a synergistic

homeostasis. Disruptions of the oral environment that exceed this adaptive capability allow facultative pathogenic species to proliferate resulting in disease processes. Once the disease process commences, pathogenic microorganisms have the capacity to make the process self-sustaining. Pathogenic oral microorganisms increase their fitness advantage over commensal microflora by using mechanisms like altering their gene expression, regulating conditions of the local environment, and releasing metabolic products that kill or suppress competitive bacterial species (Merritt and Qi, 2012; Corcuera et al., 2013; Koo et al., 2013). Two disease processes that are the result of this type of microbial disruption are dental caries and periodontal disease.

Caries etiology

Dental caries results from three interrelated contributors: (1) disturbance of the balance between commensal and facultative pathogenic oral microflora, (2) frequent consumption of fermentable carbohydrates, and (3) host susceptibility mediated by genetic and non-genetic influences (Lingström et al., 1994; Marsh, 1995). There are more than forty different species of *Streptococcus* (Innings et al., 2005), many of which colonize the human oral cavity shortly after birth (Law et al., 2007). Many of these species are commensal, some are pathogenic. Seven pathogenic species of *Streptococcus* have been implicated in human caries and endocarditis (Coykendall, 1989). Collectively these are known as the mutans streptococci. The two most common mutans streptococci in humans are *Streptococcus mutans* and *Streptococcus sobrinus* (Coykendall, 1989). Although mutans streptococci are the most frequently implicated in caries etiology, research demonstrating the frequent presence of caries in the absence of these organisms indicates that it is the disturbance of oral microbiotic balance that is more important in caries etiology than the presence of a particular organism (Kleinberg, 2002; Giacaman et al., 2010; Simón-Soro et al., 2013). When pathogenic bacteria proliferate in the oral cavity and consume carbohydrates, they produce metabolic wastes which contribute organic acids and proteases to the oral fluids (Liljemark and Bloomquist, 1996; Featherstone, 2004). These metabolic products make the oral environment more favorable for the pathogenic organisms by lowering pH and in sufficient concentration, can dissolve the mineral component of dental enamel producing a carious lesion. Left unchecked, proteolytic decay can completely destroy a tooth and enter the circulatory system exposing extra-oral sites to infection. Oral bacteria, especially those connected with dental caries, have been shown to have a major role in the development of serious extra-oral diseases including cardiovascular disease, rheumatoid arthritis, inflammatory bowel disease, colorectal cancer, and respiratory tract infections (Meurman et al., 2004; Han and Wang, 2013).

Periodontal disease etiology

Periodontal diseases are multifactorial infections of the sulcular region between the tooth surface and the gingival margin (Holt and Ebersole, 2005). At least 400 of the more than 1200 bacterial species that colonize the oral cavity inhabit subgingival sites (Paster and Dewhirst, 2009). Up to 150 different species are present in any individual's subgingival plaque (Buduneli and Kinane, 2011). Similar to dental caries etiology, most of the

bacterial species in subgingival biofilms are commensal. Fewer than twenty are considered to be major periodontal pathogens (Socransky and Haffajee, 2005). The initiation of periodontal disease commences with a shift in subgingival bacterial colonies from ones predominated by gram-positive bacteria to communities dominated by anaerobic gram-negative bacteria (Slots, 2010). The triggers that cause the overgrowth of these normally commensal species and the virulence determinants associated with them are not understood (Holt and Ebersole, 2005).

The pathogenic bacterial species primarily implicated in periodontal diseases are *Tannerella forsythia, Porphyromonas gingivali*s, *Aggregatibacter actinomycetemcomitans* (Laine et al., 2013). The predominance of these bacterial species has been clearly linked to chronic periodontal disease (Dentino et al., 2013). Tissue destruction resulting from chronic periodontal disease is due to collateral damage from the host immune response rather than destruction directly caused by the pathogenic bacteria (Taubman et al., 2005; Dentino et al., 2013; Cekici et al., 2014). The overgrowth of pathogenic species stimulates the release of host produced inflammatory cytokines, chemokines, and mediators (Silva et al., 2007). Host leukocytes responding to infection do not discriminate between the invading bacteria and host tissue. The result of this failure to discriminate pathogen from host tissue is the collateral destruction of gingival and skeletal tissues adjacent to the infection site (Liu et al., 2010; Preshaw and Taylor, 2011). With continued infection there is progressive destruction of gingiva, the periodontal ligament, and the underlying alveolar bone tissue. Unabated, periodontal disease will cause sufficient alveolar recession to result in tooth exfoliation (Oliver and Brown, 1993; Thomson et al., 2013). The bacteria associated with periodontal disease have also been linked to increased risk for cardiovascular diseases (Kebshull et al., 2010; Reyes et al., 2013; Schenkein and Loos, 2013) and other serious extra-oral infections (Han and Wang, 2013).

Periapical periodontitis etiology

More than 450 bacterial species have been identified in endodontic infections (Siqueira and Rôças, 2009). This is by far the most diverse assemblage of oral pathogens in all of the diseases of the oral cavity. Most endodontic infections are polymicrobial, involving various combinations of the most prevalent bacterial species (Brauner and Conrads, 1995; Dahlén, 2002). These include microaerophilic streptococci (*S. anginosus*, *S. constellatus*, *S. intermedius*), anaerobic streptococci (*Peptostreptococcus anaerobius*, *P. micros*), gram-positive anaerobic rods (*Eubacterium* spp., *Actinomyces* spp., *Propionibacterium* spp.), and gram-negative anaerobic rods (*Porphyromonas* spp., *Prevotella* spp., *Bacteroides* spp., *Campylobacter* spp., *Fusobacterium* spp., *Treponema* spp.). Endodontic infection involves an opportunistic infection of the dental root canal system (Siqueira, 2002). Infection of the root canal requires the absence of vital pulp tissue (Siqueira and Rôças, 2009). Pulp tissue deficiency results from either pulp necrosis which occurs as part of a disease sequela (caries, trauma, periodontal disease) or pulp removal as part of a medical procedure (Abbott, 2004). In response to the proliferation of pathogenic microorganisms, the body mounts an immune response involving the same inflammatory cytokines, chemokines, and

mediators found in other periodontal diseases (Silva et al., 2007). The resulting abscess is a localized collection of the immune responders and disintegrated tissues (pus). Drainage of the cellular debris is accomplished through the formation of a fistula. In the case of periodontal abscesses, the drainage is most likely through the periodontal pocket because that is the path of least resistance (Dahlén, 2002). The sequelae of chronic infection include tooth exfoliation as well as bacterial diffusion to sinuses and other facial spaces to form cellulitis (Siqueira and Rôças, 2009)

Dental wear etiology

The age-related phenomenon of dental enamel reduction (dental wear) occurs in all human populations and is recognized as a contributing factor in oral disease risk (Lee et al., 2012). Dental wear results from three contributors: (1) attrition results from tooth-on-tooth contact, (2) abrasion is produced by the friction of foreign substances on the enamel, and (3) erosion is the chemical dissolution of enamel not due to biofilm (Kaidonis et al., 1998). Dental wear is dependent on two major variables, diet and age of the individual. Dental wear in populations that have a more abrasive diet (e.g., hunter-gatherer subsistence) tends to be more rapid and severe than in those that have a softer, more highly processed diet (e.g., agricultural subsistence) (Deter, 2009). Not only does subsistence system affect the overall level of wear but it also has an effect on the specific pattern of wear on the teeth. Given the fibrous nature of many hunter-gatherer diets, the wear pattern generally tends to be flat across the occlusal surface (Smith, 1984). The softer texture of the agricultural diet tends to produce a "scooped out" appearance to the occlusal surface (Smith, 1984). Severe wear or wear resulting from specific, repetitive alimentary and non-alimentary activities can result in wear patterns that significantly deviate from these generalized patterns (Kieser et al., 2001; Kaifu et al., 2003).

The pathological and non-pathological physiological processes discussed above all have the capacity of altering the oral environment. Recent clinical research indicates that pathological processes in the oral cavity are the result of disturbances in the normal balance of our native microbiotic communities rather than invasion by exogenous organisms. Dental wear also has the capacity of altering the balance of the microbiotic community through both disruption and facilitation. That is, dental wear in general disrupts the microbial environment through biofilm removal, while excessive wear offers pathological species access to normally protected areas. Based on the etiological foundations of the four processes examined here, it is hypothesized that high levels of dental wear will correspond to low prevalence of oral pathology in the CA-CCO-548 population sample. Specifically, the consistent removal of biofilm through heavy dental wear should prevent the proliferation of pathogenic microorganisms in the oral cavity.

MATERIALS AND METHODS
The population sample

The population sample examined in this study derives from CA-CCO-548, an ancient Native American mortuary site in the Central Valley of California. Human occupation of the Central Valley can be traced back to
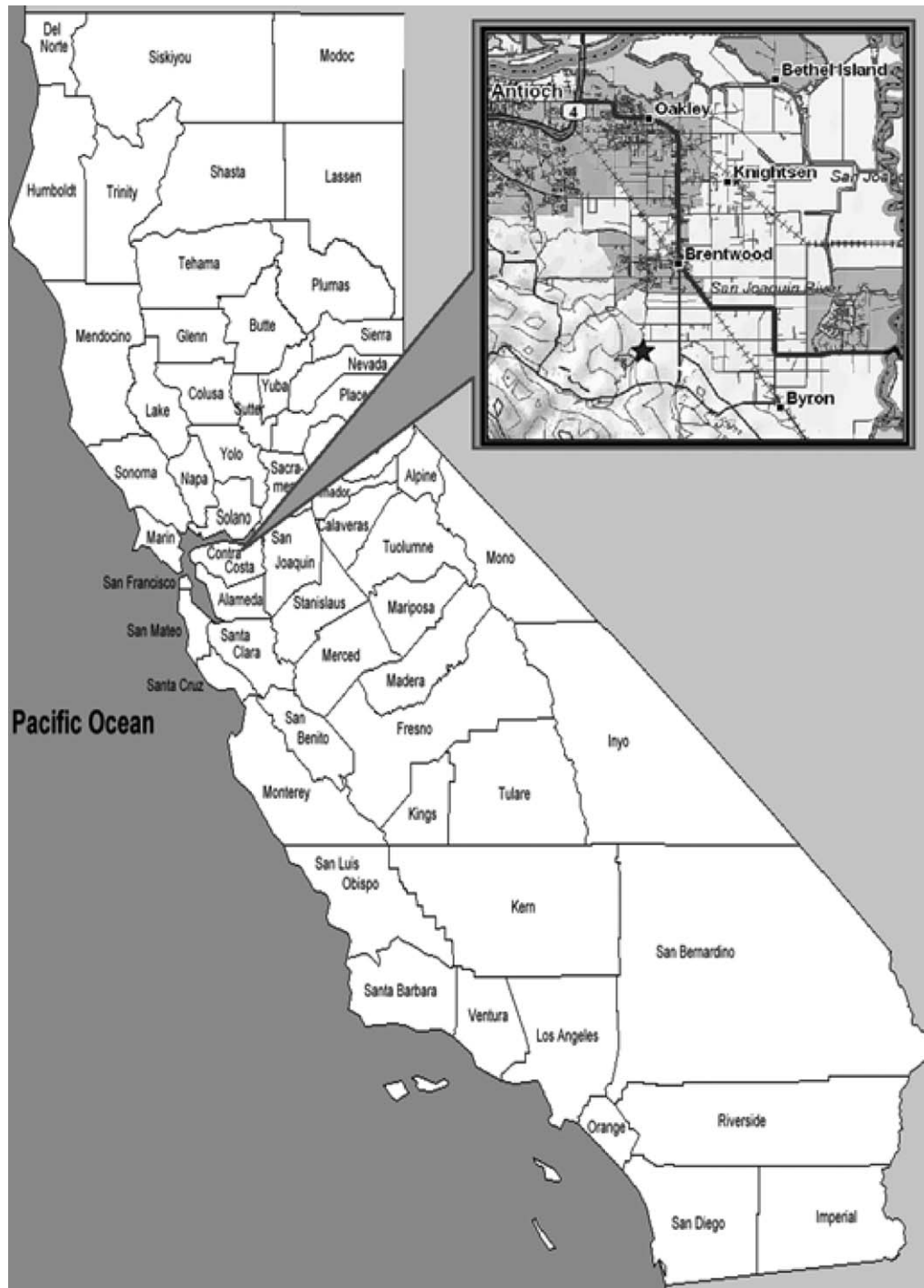
Fig. 1. Geographic location of the Vineyards Site (CA-CCO-548).

around 13,500 BP (West et al., 1991). Occupation during the Paleo-Indian (13,500-10,500 BP) and Lower Archaic (10,500-7,500 BP) is represented by isolated finds (Rosenthal et al., 2007). Occupation in the valley during the Middle Archaic (7,500–2,500 BP) is represented by numerous archaeological sites scattered across the entire region. Despite the presence of numerous sites, extensive human skeletal samples from the Early Period of California (11,000–3,000 BP) are rare (Walker, 2006). This trend holds true for the early Middle Archaic of the

Central Valley (ca. 7,500–4,000 BP) where few cultural deposits have been found due to obliteration or burial by alluvial action (Rosenthal et al., 2007).

The archaeological deposits at CA-CCO-548 represent a rare example of a stratified, multi-component site from the Middle Archaic (Fig. 1). The human skeletal sample, comprised of at least 480 individuals, is the largest for this pivotal time period in California. Geoarchaeological analysis has identified four discrete stratigraphic temporal components for the site, spanning a time from 7000

TABLE 1. Age and sex distribution for CA-CCO-548

	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

TABLE 2. Frequencies of antemortem tooth loss (AMTL) and postmortem tooth loss (PMTL) in maxillary and mandibular dentitions

	Total sample			Female			Male		
	N	% AMTL	% PMTL	N	% AMTL	% PMTL	N	% AMTL	% PMTL
Maxilla									
I1	252	9.1	26.2	79	3.8	34.2	116	16.4	21.5
I2	273	4.8	20.1	88	3.4	21.6	128	7.8	19.5
C	293	2.4	9.2	91	0.0	11.0	133	5.3	6.8
P3	289	3.1	5.9	90	2.2	3.3	136	5.1	4.4
P4	291	7.9	5.1	89	7.9	2.2	132	9.8	6.8
M1	296	11.5	0.7	86	5.8	1.1	128	19.5	0.0
M2	294	6.8	2.0	89	3.4	3.4	135	11.1	2.2
M3	239	7.9	8.4	68	4.4	11.8	120	10.0	7.5
Total	2227	6.6	9.4	680	3.8	10.7	1028	10.5	8.4
Mandible									
I1	263	8.4	19.8	82	11.0	20.7	122	8.2	16.4
I2	301	5.3	14.3	96	5.2	16.7	139	6.5	11.5
C	331	3.3	15.7	96	7.3	16.7	151	2.6	15.2
P3	343	4.9	10.5	102	9.8	7.8	152	3.9	12.5
P4	361	6.1	6.9	112	11.6	2.7	163	6.5	7.4
M1	379	6.1	4.2	112	13.4	5.3	164	3.6	0.6
M2	373	7.5	1.9	114	14.0	1.7	168	7.1	1.2
M3	307	11.4	7.5	85	16.5	12.9	149	11.4	4.0
Total	2658	6.5	9.5	799	11.1	9.9	1208	6.2	8.2
Site total	4885	6.6	9.5	1479	7.8	10.3	2236	8.2	8.3

to 3100 cal BP (Meyer, 2010). All but five of the burials were recovered from the components dating between 4,300 and 3,100 cal BP. The majority ($n = 304/480$) of the mortuary features were from the component that dates from 3,600 to 3,400 cal BP. Sex determination could be ascertained for 191 individuals (Table 1). Among the individuals for which sex could be determined, 41.9% ($n = 80/191$) are female and 58.1% ($n = 111/191$) are male (Griffin et al., 2010). Age at death could be estimated for 335 individuals (Table 1). Subadults account for 10.1% ($n = 34/335$) of the sample for which age-at-death could be estimated and 89.9% ($n = 301/335$) are adults (Griffin et al., 2010). It is notable, especially for the pathological conditions that are age-progressive, that 49% ($n = 164/335$) of the sample are older than 40 years of age and 29% ($n = 97/335$) are older than 45 years of age.

Carious lesions

To examine frequencies of carious lesions in the sample from CA-CCO-548, each tooth was examined with $3.5\times$ dental loupes, a 3-mm Microlux Transilluminator™ fiber optic diagnostic light, and stainless steel dental probes. Fiber optic transillumination offers a superior method of lesion detection compared to other macroscopic methods (Davies et al., 2001; Pretty, 2006). Carious lesions were only counted with the presence of a definable pit (cf., Lukacs, 1989; Buikstra and Ubelaker, 1994; Cucina et al., 2011; Halcrow et al., 2013). Tooth discolorations alone were not counted as carious lesions. Wasterlain and coworkers (2011) suggest that this method underestimates the actual number of carious

teeth, however, all of the comparative studies used here use the minimum criteria of a definable pit for lesion identification. Lesions were recorded with regard to their location on the tooth (occlusal, cervical, buccal, lingual, mesial, and distal). Maximum lesion size was measured in millimeters using a Paleo-Tech Helios type needle point caliper. The data reported here have been reduced to lesion presence and absence because the comparative studies for the region do not include details regarding numbers of lesions on particular teeth, lesion sizes, or lesion locations. To facilitate comparison with other sites, left and right antimeres were pooled while maintaining separate counts for each tooth classification in the maxillary and mandibular dentitions. Maintaining counts for separate tooth classifications allows for the detection of intra-oral patterns of disease (cf., Hillson, 2001; Caglar et al., 2007; Temple and Larsen, 2007; Wasterlain et al., 2009; Cucina et al., 2011). Correction factors to account for antemortem tooth loss have been suggested for the examination of caries rates (e.g., Lukacs, 1995; Duyar and Erdal, 2003). Correction factors are not used here because none of the comparative studies used such factors. Antemortem tooth loss (Table 2) is very low in this population sample (overall 6.6%; $n = 322/4885$) and there are no significant differences in AMTL between sexes.

Periodontal disease and periapical abscesses

Clinical researchers that have measured alveolar bone loss in dry skulls consider distances of 1–3 mm from the cemento-enamel junction (CEJ) to the alveolar crest

Fig. 2. Examples of teeth with stage 9 wear.

(AC) to be normal (Davies et al., 1969; Tal, 1985). Most bioarchaeological studies consider CEJ to AC measurements in excess of 2 mm to be potentially pathological (Clarke et al., 1986; Wasterlain et al., 2011; DeWitte, 2012). Bioarchaeological research has shown that accurate documentation of periodontal disease in skeletal populations depends on CEJ to AC measurement and corroborating inspection of the AC for pathologic activity (Klaus and Tam, 2010; Wasterlain et al., 2011; Vodanović et al., 2012). This method accounts for the phenomenon of continuous eruption. The human dentition continues to erupt beyond the time when teeth initially come into occlusion (Tal, 1985; Whittaker et al., 1985; Kaifu et al., 2003; Haytac et al., 2013). The process is age-progressive and is not directly dependent on other factors such as wear (Varrela et al., 1995), although excessive wear can accelerate the process (Newman, 1999). For this study, CEJ to AC measurements were taken for each tooth and the adjacent AC was macroscopically examined for porosity. Teeth with CEJ to AC measurements in excess of 2 mm that also exhibited adjoining alveolar bone porosity were considered pathological.

Periapical abscesses can be associated with periodontitis (periodontal abscess); however, the vast majority of periapical abscesses are of endodontic origin (Dahlén, 2002). Other research has suggested that most periapical cavities in the archaeological record may be the result of periapical granuloma and cysts (Dias and Tayles, 1997). This view is not supported by the clinical research that indicates that granuloma and cysts are relatively rare, accounting for only 15% of periapical lesions (Shear, 1992; Nair, 1997; Nair, 2003). Differential diagnosis of the two conditions is relatively straightforward. Granuloma and cysts are closed pathological cavities (Nair, 2003) that measure less than 3 mm (Dias and Tayles, 1997). Periapical abscesses are a localized collection of pus with a well-defined sinus tract (fistula) which generally exits through the periodontal pocket (Dahlén, 2002) and measure greater than 3 mm (Dias and Tayles, 1997).

To assess the frequency of periapical abscesses, each tooth position was examined macroscopically for the presence of periapical alveolar defects. Defects were recorded by specific location and size. Defects that measured in excess of 3 mm and exhibited a well-defined sinus tract were considered to be periapical abscesses.

For purposes of intersite comparison, left and right maxillary and mandibular tooth positions were pooled.

Dental wear

While the systematic observation of dental wear has long been a standard part of bioarchaeological studies, the method of measurement has been far from standardized. Various scoring systems have been proposed attempting to account for variations in dentin exposure (Molnar, 1971b; Scott, 1979; Molnar et al., 1983; Richards, 1984; Dreier, 1994), reduction of crown height (Tomenchuk and Mayhall, 1979), and angle of wear plane (Molnar, 1971b; Hall, 1976; Smith, 1984). Unfortunately, standardizing a method of recording dental wear is nearly impossible because the way in which human dentitions wear is far from standard and depends on a number of complex biological and behavioral factors.

Each tooth in the CA-CCO-548 sample was scored for dental wear using the Smith wear system (Smith 1984). The Smith system was used here because of its simplicity, which reduces inter- and intra-observer error. A considerable number of teeth in precontact California Native populations exceed stage eight in the system. Following the recommendation of Reinhardt (1983), a stage 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 (Fig. 2). 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. Pooling the antimeres is less than ideal for accurately recording specific wear patterns. However, published comparison data for populations of appropriate geographic location and time period mostly consists of pooled data.

RESULTS

Examination of carious lesion prevalence in the CA-CCO-548 sample (Tables 3 and 4) reveals that the overall lesion frequency is quite low. The frequency of teeth with carious lesions is 2.5% for the maxillary dentition ($n = 44/1782$) and 2.6% for the mandibular dentition ($n = 56/2175$). Compared with other population samples from Central California (Tables 3 and 4) the overall frequencies of carious lesions in this population sample are

TABLE 3. Carious lesion frequencies in Central California sites

Tooth	CCO-548 N	CCO-548 %	SJO-17[a] N	SJO-17[a] %	SJO-154[b] N	SJO-154[b] %	SRI-41[c] N	SRI-41[c] %	SRI-2A[c] N	SRI-2A[c] %	SRI-2B[c] N	SRI-2B[c] %
Maxilla												
I1	153	0.6	36	2.8	26	7.7	82	1.2	16	0	36	0
I2	190	0	35	2.8	32	3.1	107	6.5	20	0	54	0
C	236	0.8	52	7.7	35	14.3	124	16.9	22	0	77	2.6
P3	248	0.4	59	7.7	32	9.4	111	16.2	16	0	73	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	21	33.3	70	34.3	14	28.6	43	18.6
Total	1782	2.5	417	9.3	231	12.5	806	17	143	11.9	531	7.7
Mandible												
I1	186	0.5	33	0	30	3.3	105	0	8	0	39	0
I2	231	0.4	34	0	33	6.1	126	1.6	12	0	46	0
C	258	0	38	7.9	33	6.1	129	3.9	18	5.6	52	0
P3	276	0.7	39	2.6	39	15.4	126	7.9	15	6.7	53	0
P4	296	0	40	0	40	10	120	10.8	16	0	52	0
M1	340	1.8	45	4.4	35	14.3	107	22.4	14	14.3	63	12.7
M2	339	5.3	41	4.9	32	15.6	118	19.5	14	21.4	53	7.5
M3	249	11.2	41	17.1	33	30.3	81	17.3	11	27.3	45	13.3
Total	2175	2.6	311	4.8	275	12.7	912	10	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

[a] Molnar 1971b.
[b] Hoffman 1987.
[c] Walker and Erlandson 1986.

extraordinarily low. Furthermore, the combined lesion frequencies for CA-CCO-548 are more than 50% lower than any of the other comparative population lesion frequencies. Clinical research has established that there is a general gradient of caries risk which progressively increases from anterior tooth positions to posterior ones (Afroughi et al., 2010; Sheiham and Sabbah, 2010). Second molars consistently have the highest frequencies of carious lesions due to their complex fissure patterns (Ferreira Zandona et al., 2012). Given these relationships, it is important to establish that combined lesion frequencies are not the result of over-representation of one tooth type when reporting overall frequencies of carious lesions. The difference in proportion of anterior teeth to posterior teeth in this sample is statistically significant ($\chi^2 = 111.37$, P-value = 0.0001). However, considering that the higher proportion of teeth is contributed by the posterior dentition (68.3%; $n = 2703/3957$) and the highest proportion of a tooth class is contributed by just the molars (41.6%; $n = 1646/3957$), the low overall lesion frequencies become more notable. With a high proportion of molar teeth, one would expect resulting higher overall frequencies of lesions.

Considering the molar teeth separately, the overall low frequencies of carious lesions in this sample become more remarkable. The frequency of carious lesions in maxillary molars is 5.4% ($n = 39/718$) and 5.6% ($n = 52/928$) for mandibular molars. These are far lower than the frequencies reported for the molar teeth in most of the comparative samples. The only comparative site with frequencies this low is the Channel Islands site of CA-SRI-2B, reporting frequencies of 9.5% ($n = 21/221$) for the maxillary molars and 5.6% ($n = 28/501$) for the mandibular molars. The frequencies of molar lesions in the CA-CCO-548 sample are less than half that observed for most other sites and less than one quarter of that seen at some of the comparative sites.

When assessing frequencies of carious lesions, the age-progressive nature of the disease is always a concern. This is especially true of the molars. The age structure of CA-CCO-548 is of particular concern given that 49% of the sample are estimated to be older than 40. Table 5 shows the distribution of carious lesions in seven age groups for the molar series. The differences in frequency of carious lesions between the age groups does not show a consistent age progression and are not statistically significant ($\chi^2 = 9.699$, P-value = 0.138). Similar to previous research in other population samples, females in the CA-CCO-548 sample exhibit higher frequencies of carious lesions for most teeth (Fig. 3). However, only two teeth (lower M2 and M3) have differences between males and females that are statistically significant ($\chi^2 = 7.602$, P-value = 0.022; $\chi^2 = 7.897$, P-value = 0.006).

Examination of the average wear across the entire CA-CCO-548 sample (Fig. 4) indicates a population exhibiting considerable dental wear. The average overall dental wear score for the sample is 6.1 with more than 30% of the sample exhibiting average wear scores of eight or above. Comparing composite wear scores for samples can be somewhat deceiving when individuals are wearing their teeth in unusual ways. The mean wear separated by tooth class shows that the overall wear in this population sample is relatively uniform in terms of degree of wear (Fig. 5). Comparison of male and female average wear scores (Fig. 5) reveals very little difference in wear levels between the two sexes. None of the differences are statistically significant. A comparison of CA-CCO-548 with two other Central California pre-contact sites for average wear scores in each tooth class of the upper and lower dentition (Fig. 6) reveals a striking similarity in amount and pattern of wear. All three sites exhibit considerable wear, with the highest wear values on the upper and lower first molars.

Comparing frequencies of the three separate pathological conditions of the periodontal region reveals a

TABLE 4. Carious lesion frequencies in females and males for Central California sites

Tooth	CCO-548 Female N	%	CCO-548 Male N	%	SJO-17[a] Female N	%	SJO-17[a] Male N	%	SJO-154[b] Female N	%	SJO-154[b] Male N	%	SRI-41[c] Female N	%	SRI-41[c] Male N	%	SRI-2A[c] Female N	%	SRI-2A[c] Male N	%	SRI_2b[c] Female N	%	SRI_2b[c] Male 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
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	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	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	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	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	42	9.5
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	42	19.0
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	19	10.5
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	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	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	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	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	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	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	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	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	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	163	4.9
Site Total	1102	3.5		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	404	6.4

[a] Molnar 1971b.
[b] Hoffman 1987.
[c] Walker and Erlandson 1986.

TABLE 5. Carious Lesions in Adult Molars by Age Group (N, %)

				Age-at-death				
	21–25	26–30	31–35	36–40	41–45	46–50	51–55	
Tooth[a]								Total
UM1	20 (0.0)	21 (4.7)	34 (8.8)	52 (15.4)	52 (3.8)	48 (6.2)	8 (0.0)	**235** (7.7)
UM2	19 (0.0)	21 (9.5)	29 (3.4)	55 (3.6)	52 (1.9)	62 (0.0)	8 (0.0)	**246** (2.4)
UM3	10 (0.0)	15 (13.3)	22 (9.1)	41 (7.3)	44 (2.3)	56 (12.5)	5 (20.0)	**193** (8.3)
LM1	24 (0.0)	28 (0.0)	37 (5.4)	59 (0.0)	81 (0.0)	80 (5.0)	12 (0.0)	**321** (1.8)
LM2	24 (16.7)	31 (3.2)	35 (11.4)	62 (1.6)	79 (3.8)	78 (2.6)	11 (9.1)	**320** (5.1)
LM3	20 (15.0)	25 (8.0)	21 (23.8)	44 (15.9)	72 (9.7)	54 (3.7)	8 (25.0)	**244** (11.5)
Total	**117** (6.0)	**141** (5.7)	**178** (9.5)	**313** (7.0)	**380** (3.7)	**378** (4.8)	**52** (7.7)	**1559** (5.8)

[a] Antimeres are combined.
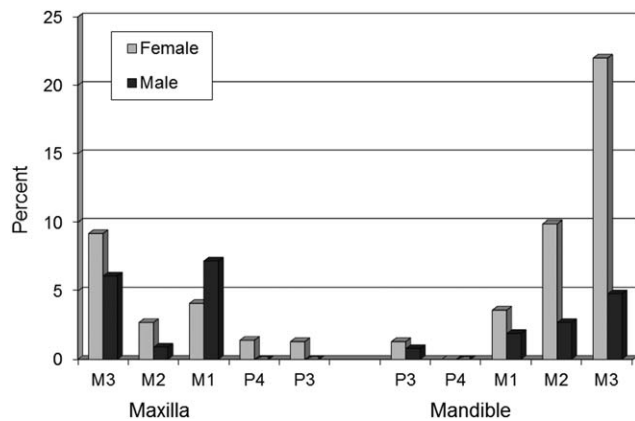
Fig. 3. Frequencies of carious lesions in maxillary and mandibular teeth of females and males.

Fig. 4. Frequencies of Smith (1984) composite dental wear scores for CA-CCO-548.

dynamic interplay between unique features of subgingival microenvironments and cultural factors. Examination of periodontal recession levels for each tooth position (Fig. 7) shows a population with very moderate amounts of alveolar bone resorption. Considering the upper and lower dentitions separately, the upper dentition shows 59.9% ($n = 1001/1671$) of the teeth with CEJ to AC measurements greater than 2 mm and 27.2% ($n = 454/1671$) greater than 3 mm. The lower dentition exhibits 76.9% ($n = 1657/2154$) of the teeth with CEJ to AC measurements greater than 2 mm and 56.3% ($n = 1213/2154$) greater than 3 mm. Females in this sample generally have greater amounts of periodontal recession than males, however the difference in frequencies is not statistically significant ($\chi^2 = 0.120$, P-value = 0.729). The overall frequency of individuals that exhibit periodontal disease (CEJ to AC measurements greater than 2 mm and evidence of alveolar bone porosity) is 17.8%.

Examination of periodontal disease frequencies reported in Table 6 shows that unlike most other population samples, females in the CA-CCO-548 sample have overall frequencies of periodontal disease that are higher than males (20.3% vs. 16.6%). The difference is statistically significant ($\chi^2 = 10.41$, P-value = 0.001). Examination of the frequencies of periapical abscesses reported in Table 6 reveals a relatively high number of lesions across the entire sample, especially in the region of the maxillary and mandibular first and second molars. The overall frequency of periapical abscesses is 10.7% ($n = 409/3825$). Females show a higher frequency of periapical abscesses than do males (14.0% vs. 10.8%). The
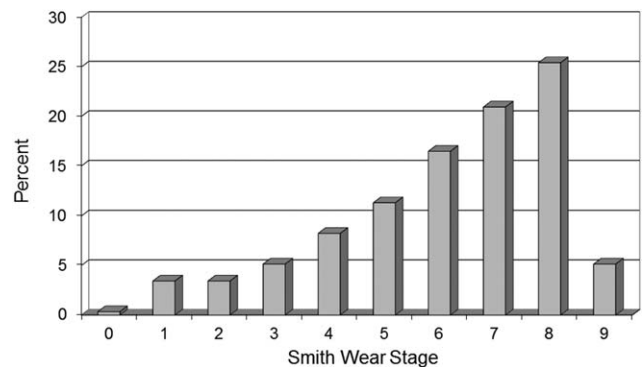
difference is statistically significant ($\chi^2 = 7.92$, P-value = 0.0049).

Taken together, the frequency and pattern of periodontal recession and periapical abscesses observed in this population is one that cannot be fully explained by disease. That is, while the rates of periodontal recession in excess of 2 mm are high at 69.5% ($n = 2658/3825$), less than one-third of these tooth positions exhibit signs of periodontal disease. In fact, the teeth with the highest overall rates of recession (lower I1-P4) have some of the lowest frequencies of periodontal disease. The most likely explanation for the levels of recession in these teeth is super-eruption due to excessive wear. Super-eruption of the dentition (continuous eruption) has been suggested as a physiological compensatory mechanism for horizontal enamel loss in humans (Newman, 1999; Haytac et al., 2013). Continuous eruption of this type increases the distance between the CEJ and the AC (Newman, 1999). Continuous tooth eruption has been documented in populations that exhibit excessive dental wear (Levers and Darling, 1983; Whittaker et al., 1985; Varrela et al., 1995). While continuous eruption has been shown to occur in humans irrespective of wear, it does seem to be correlated with the availability of interocclusal space (Levers and Darling, 1983; Newman, 1999).

DISCUSSION

Four notable features emerge from the statistical analyses of the CA-CCO-548 dentitions. The sample exhibits 1) extraordinarily low frequencies of carious lesions, 2) very low frequencies of periodontal disease, 3) high
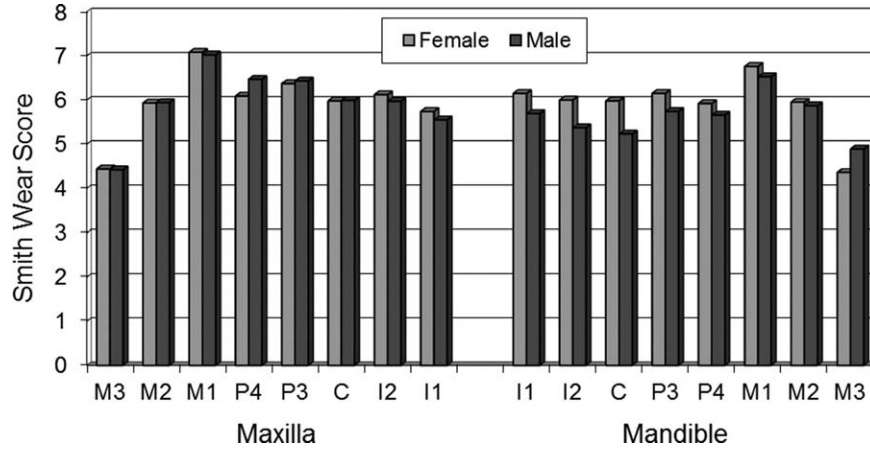
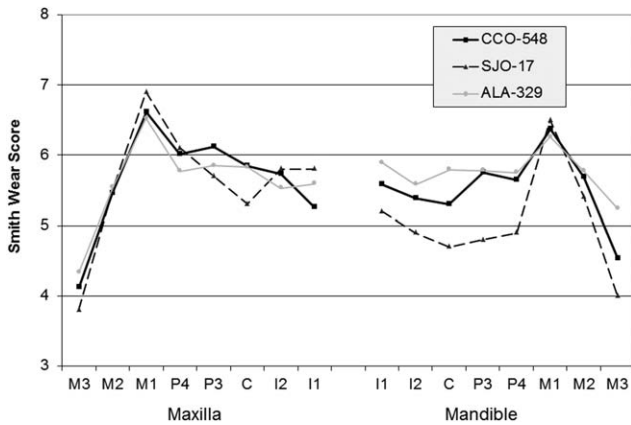**Fig. 5.** Mean dental wear by tooth class and sex.

Fig. 6. Mean dental wear by tooth class and sex for CA-CCO-548 compared to two other sites, CA-SJO-17 and CA-ALA-329 (comparative data from Molnar 1971 and Jurmain 1990).

frequencies of periapical abscesses, 4) and remarkably high levels of dental wear. Because the etiologies of the pathological conditions are interrelated, it is logical to examine the relationship and potential influence of each condition on the others. For instance, both caries and the periodontal diseases have as their primary etiological agent a shift of the oral microflora from commensal to facultative pathogenic in the diseased individual (Marsh and Devine, 2011; Takahashi and Nyvad, 2011; Dentino et al., 2013). Another key shared risk contributor for the pathologic processes is poor oral hygiene (Stabholz et al., 2010; Rodrigues et al., 2011; Dentino et al., 2013). Dental wear also has a potential influence on all three of the pathologic processes (Newman, 1999; Caglar et al., 2007; Buzon and Bombak, 2010).

Dental caries has been used in bioarchaeological studies as an indicator of overall dental health and dietary changes in past populations (Larsen et al., 1991; Sciulli, 1997; Temple and Larsen, 2007; Šlaus et al., 2011; Williams and Murphy, 2013). Much of the literature pertaining to the influence of dietary change on overall health has focused on the adoption of agriculture (Larsen et al., 1991; Lukacs, 1992; Temple and Larsen, 2007; Watson, 2008; Cucina et al., 2011; Halcrow et al., 2013). The conclusion of this body of research is that the increase in carbohydrate consumption with the transition

to agriculture precipitates an increase in caries prevalence. Clinical research indicates that the relationship between complex carbohydrates and caries is more complicated than much of the bioarchaeological research suggests (Lingström et al., 1994; Beighton et al., 1996; Lingström et al., 2000; Hujoel, 2009; Moynihan, 2012).

Clinical investigation has found that the relationship between consumption of low molecular weight (LMW) carbohydrates (white flour and white sugar) and caries is rather direct (Lingström et al., 2000; Moynihan and Kelly, 2014). In addition to being fermentable, LMW carbohydrates such as sucrose act as a substrate for the synthesis of polysaccharides in dental plaque (Bowen, 2002). Long-term consumption of LMW carbohydrates therefore lowers oral pH and shifts the balance of microflora from predominantly commensal to more cariogenic (Paes Leme et al., 2006; Parisotto et al., 2010). Nonhuman research has found a similar synergistic relationship between LMW carbohydrates and subgingival pathogenic bacteria associated with periodontal disease (Aguirre et al., 2012; Seneviratne et al., 2013). The relationship between complex carbohydrate consumption and caries rates is far less conclusive (Lingström et al., 2000). In fact, some research has shown that the consumption of starches enhances oral calcification of tooth enamel (Hidaka and Oishi, 2007). Other research has shown that starch encourages the proliferation of commensal bacteria found in the oral cavity at the potential expense of pathogenic species (Nikitkova et al., 2012).

In vitro biofilm and nonhuman caries studies have shown that complex carbohydrates are substantially less cariogenic than sucrose or glucose (Duarte et al., 2008; Thurnheer et al., 2008). *In vivo* human research has also shown that complex carbohydrates that have not undergone extensive processing generally do not offer sufficient bioavailability of the sugars necessary for cariogenic bacterial proliferation (Lingström et al., 2000) or do so at a significantly reduced rate (Moynihan, 2012). Research has shown that the manner in which the carbohydrate is processed is far more important than the amount or type of the carbohydrate consumed (Grenby, 1990; Harper et al., 1985). Modern commercial processing methods used for complex carbohydrates, such as high-temperature extrusion, explosion puffing, or "instantization," render starches with far higher glycemic and hydrolytic indices than their non-commercially processed counterparts (Brand et al., 1985; Ross et al.,
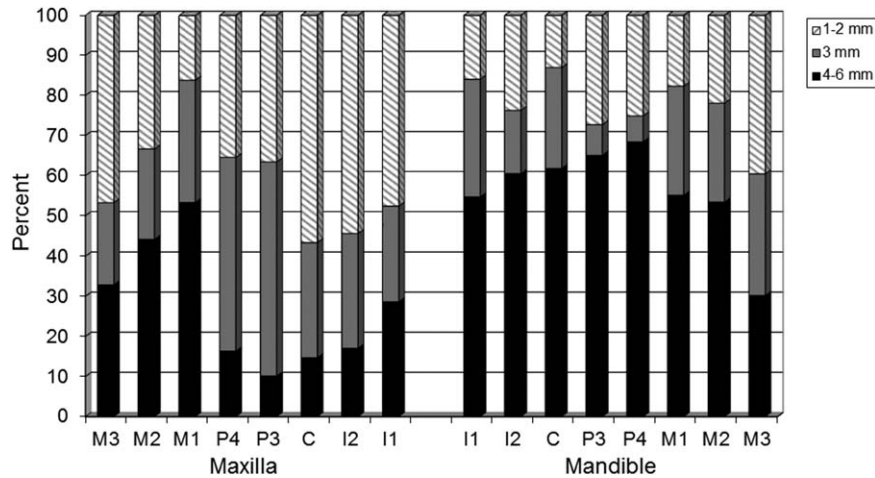
Fig. 7. Mean periodontal recession by tooth class for CA-CCO-548.

TABLE 6. Frequencies of periodontal disease (PD) and periapical abscesses (AA) in maxillary and mandibular dentitions

	Total sample			Female			Male		
	N	% PD	% AA	N	%PD	%AA	N	%PD	%AA
Maxilla									
I1	185	14.6	9.7	52	17.3	11.5	101	12.9	9.9
I2	193	9.8	6.7	59	13.6	8.5	102	6.9	5.9
C	224	13.8	8.9	69	15.9	10.1	110	11.8	8.2
P3	227	7.5	8.4	67	7.5	8.9	106	5.7	8.5
P4	226	11.9	9.3	67	23.9	16.4	106	9.4	8.5
M1	236	44.9	34.7	71	43.7	40.8	109	41.3	38.5
M2	213	27.2	12.2	60	26.7	18.3	105	19.1	13.3
M3	167	11.4	5.4	43	4.6	2.3	89	13.5	9.0
Total	1671	18.2	12.4	488	20.1	15.6	828	15.2	12.9
Mandible									
I1	201	4.5	1.5	61	8.2	3.3	96	4.2	1.0
I2	228	6.6	3.5	73	11.0	9.6	106	2.8	0.9
C	254	11.4	5.5	77	18.2	13.0	115	5.2	0.9
P3	275	10.2	6.2	88	19.3	12.5	121	1.7	1.7
P4	295	8.1	5.1	88	11.4	6.8	135	5.9	5.2
M1	323	41.8	23.8	91	52.8	30.8	150	46.0	24.7
M2	320	31.3	15.3	91	25.3	15.4	148	41.2	18.9
M3	258	14.3	7.4	71	8.5	5.6	126	18.3	10.3
Total	2154	17.5	9.4	640	20.5	12.8	997	17.7	9.0
Site total	3825	17.8	10.7	1128	20.3	14.0	1825	16.6	10.8

1987). In other words, starches that have been gelatinized and partially degraded during processing are far more cariogenic than starches processed in other manners (Lingström et al., 2000). Another important finding is that the combination of sucrose and starch vastly amplifies the cariogenic potential of sucrose alone (Campain et al., 2003; Duarte et al., 2008; Moynihan, 2012). The observations concerning the cariogenic propensity of complex carbohydrates versus LMW carbohydrates from current clinical research highlight an important finding for bioarchaeological research. That is, the cariogenic capacity of complex carbohydrates found in the modern diet is a direct result of the means and methods of manufacture and not an inherent characteristic of the food items found in preindustrial diets. Analysis of microbiota in dental calculus for populations spanning the last 10,000 years confirms this association (Adler et al., 2013). That is, shifts in the microbiota from predominantly commensal to predominantly pathogenic did not occur until the Industrial Revolution.

Dietary analysis for CA-CCO-548 provides important evidence for the cariogenic potential of the subsistence regimen at this site. Isotopic analysis for CA-CCO-548 shows that the majority of the diet derived from terrestrial and freshwater resources with a very small contribution from marine resources (Bartelink et al., 2010). Despite the close proximity of CA-CCO-548 to San Francisco Bay, the abundance of local terrestrial and fresh water aquatic resources encouraged the exploitation of resources predominantly from the immediate vicinity rather than the more distant marine resources. Analysis of stable isotopes and paleobotanical evidence from CA-CCO-548 indicates the substantial inclusion of acorns in the diet (Bartelink et al., 2010; Wohlgemuth, 2010). Acorns have a very high carbohydrate content (Soni et al., 1993; Correia et al., 2009; Yoo et al., 2012) with an amylose content similar to apples (Stevenson et al., 2006). The amylose and amylopectin content of acorns is considerably higher than that of corn, wheat, rice, and all other A-type starches (Soni et al., 1993; Stevenson
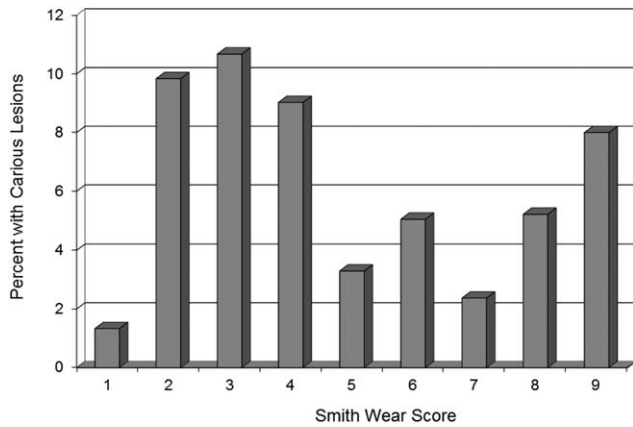
Fig. 8. Frequency of carious lesions for each wear score in the molar series.
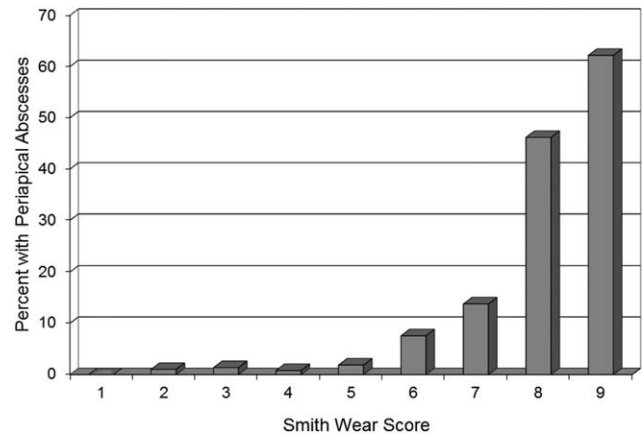

Fig. 9. Frequency of periapical lesions for each wear score in the molar series.

et al., 2006; Yoo et al., 2012). The high tannin content of acorns necessitated an intensive processing and cooking procedure which approximated some of the techniques of modern LMW starch production (Basgall, 1987; Margolin, 1989; Jacknis, 2004). For instance, the repeated grinding, sifting, and drying involved in the traditional preparation of acorns is similar to modern techniques of mechanical reduction which make starches highly susceptible to gelatinization (Lingström et al., 2000). In addition, acorns have a very low temperature of gelatinization (Stevenson et al., 2006) compared with other starches (Ashogbon and Akintayo, 2013). High water-starch ratios significantly decrease the temperature at which gelatinization occurs (Lingström et al., 2000). Water-leaching to remove tannins and hot water meal cooking techniques contributed to high water-starch ratios in traditional acorn preparation (Basgall, 1987; Margolin, 1989; Jacknis, 2004). Considering the high amylose and amylopectin content of acorns, the intensive processing techniques associated with acorn consumption, and the demonstrated relationship between a high carbohydrate diet and prevalence of caries, one would expect a high prevalence of caries in the CA-CCO-548 sample. However, not only is caries prevalence very low at CA-CCO-548, it is one of the lowest recorded in precontact Central California. Recent clinical research regarding the etiologies of oral pathologies offers possible explanations for the low prevalence of caries in a population consuming a carbohydrate-rich diet.

One possible explanation for the exceptionally low frequencies of carious lesions in this sample is excessive dental wear. The results reported here for CA-CCO-548 show an inverse relationship between wear and frequencies of carious lesions. That is, while the overall frequency of carious lesions is remarkably low at 2.5%, the overall average Smith wear score is notably high at 6.1 out of nine. Isolating the comparison to only the molars, the teeth most susceptible to caries, the relationship is statistically significant ($\chi^2 = 30.301$, P-value = 0.000). However, the relationship between dental wear and carious lesions is only weakly correlated (Tau-b = −0.052). Examination of the distribution of carious lesion frequencies for each wear score reveals why the relationship is very weakly correlated (Fig. 8). While the inverse relationship does generally hold true for comparison of wear scores 2 through 7, teeth with the wear scores of 1, 8, or 9 do not follow the overall trend. Results of this

analysis do not provide strong support for the cariostatic influence of dental wear.

While a causative relationship between degree of wear and prevalence of caries is not supported here, a relationship between dental wear and periapical lesions is supported. Examination of dental wear scores for each tooth position and the corresponding frequency of periapical lesions for that position show a positive correlation. Isolating the comparison with the molars, the teeth most susceptible to periapical lesions, low wear scores correspond to low frequencies of lesions (Fig. 9). Likewise, as the wear progresses, frequencies of periapical abscesses increase. The relationship is statistically significant ($\chi^2 = 378.88$, P-value = 0.000) and relatively strong (Tau-b = 0.386). Researchers have noted the likely interaction between excessive dental wear and frequency of periapical lesions (Clarke et al., 1986; Jurmain, 1990; Clarke and Hirsch, 1991). Excessive dental wear causes compressive injury of the dental pulp which releases inflammatory mediators leading to inflammation of the periodontal ligament (Torobinejad and Bakland, 1980). Clinical research has demonstrated a direct relationship between physiological compressive force on the periodontal ligament and the stimulation of inflammation related genes (El-Awady et al., 2013). Bioarchaeological research has shown that even in the relative absence of other evidence of periodontal disease, populations that show excessive wear also exhibit high frequencies of periapical abscesses (Clarke et al., 1986; Hall et al., 1986; Clarke and Hirsch, 1991). Statistical analysis in this population sample shows a significant and strong positive correlation between severe wear and periapical lesion frequency.

Another possibility connecting the low frequencies of carious lesions to the high levels of attrition is revealed by examining not only the extent of wear but also the specific patterns of wear. In particular, when the corresponding maxillary and mandibular dentitions are articulated in anatomical position, a revealing pattern of wear emerges. In the majority of intact dentitions from this site, the maxillary and mandibular teeth are not capable of direct occlusion due to the wear facets that have developed (Fig. 10). The pattern of wear has been shown to most likely result from non-alimentary behavior such as basket-making or cordage manufacture (Grant, 2010). Dietary items and non-alimentary

Fig. 10. Examples of non-alimentary dental wear in CA-CCO-548 (photos by Dave Grant).

material consistently in contact with the oral cavity have the capacity to alter the microbial environment, potentially disrupting the ability of pathogenic bacteria to displace the commensal bacteria. Specifically, phenolic compounds found in dietary and non-dietary plant materials have antimicrobial properties (Lattanzio et al., 2008; Dai and Mumper, 2010).

Many naturally occurring phenolic plant compounds have antimicrobial properties specific to human pathogens (Chung et al., 1998; Puupponen-Pimiä et al., 2005; Friedman, 2007). Salicylic acid is a naturally occurring phenolic plant hormone that has been used medicinally for more than 2,000 years (Vlot et al., 2009). The primary natural source of salicylic acid is the willow tree (*Salix sp.*). Willow was a ubiquitous material used in Central California Native American basketry, as well as a wide range of household applications including the manufacture of bows, arrows, women's skirts, mats, cordage, and the construction of dwellings (Elsasser, 1978; Farmer, 2010; Shanks and Shanks, 2010). While basket making was largely confined to females, construction of other household items using willow was not exclusively a female occupation. The traditional method of fiber and willow bark preparation (still practiced modernly) is via oral modification and manipulation of the materials (Merriam, 1967; Newman, 1974; Bocek, 1984; Shanks and Shanks, 2006). In plants, salicylic acid plays critical roles in development and metabolism (Vlot et al., 2009). It also serves a key function in plant disease resistance by inducing the production of pathogenesis-related proteins (Kawano and Furuichi, 2007). These proteins offer resistance to viral, bacterial, and fungal pathogens. When used medicinally in humans and *in vitro* experiments, salicylic acid has been found to inhibit the bacterial colonization of pathogenic organisms including species of *Staphylococcus*, *Streptococcus*, *Porphyromonas*,

and *Salmonella* (Kupferwasser et al., 2003; Bandara et al., 2004; Guinta et al., 2011; Rivero-Cruz et al., 2011). Although the specific mechanism of bacterial inhibition is not fully understood, the effect has been documented in a wide variety of contexts including specific intra-oral inhibition of *Streptococcus mutans* (Muroi and Kubo, 1993).

Willow bark would have been introduced to the oral cavity on a daily basis through the manufacture of basketry and other household materials. Because willow baskets were used in the preparation and boiling of acorns (Mayer, 1976; Bibby, 2012), salicylic acid would have been inadvertently added to the diet via this route as well. Another phenolic compound introduced into the oral cavity was tannic acid. Paleobotanical evidence from CA-CCO-548 shows a substantial reliance on acorns as well as other nuts (e.g., buckeye) with a tannic acid component (Wohlgemuth, 2010). Tannic acid, a compound found in significant quantities in acorns, has been shown to inhibit the growth of many pathogenic bacteria including *Streptococcus mutans* (Chung et al., 1993; Serrano et al., 2009). While the traditional preparation methods extracted most of the tannic acids before consumption (Gifford, 1936; Mayer, 1976), some of the acid remained after processing. The combined effect of daily introduction of these phenolic plant substances into the oral cavity inevitably would be the lifelong suppression of pathogenic oral bacteria.

The pattern of periodontal pathology observed in the CA-CCO-548 sample is also consistent with the interpretation of oral microbial suppression. Comparison of periodontal disease frequencies between archaeological populations is difficult because of the general lack of standardization in data collection. Studies from archaeological populations do exist (Clarke et al., 1986; Delgado-Darias, 2006; Eshed et al., 2006; Klaus and Tam, 2010;

DeWitte and Bekvalac, 2011; Wasterlain et al., 2011; DeWitte, 2012; Vodanović et al., 2012) as does abundant data from modern populations (e.g., Baelum and López, 2013; Genco and Borgnakke, 2013; Haytac et al., 2013). In all available comparisons, the frequencies of periodontal disease reported for the CA-CCO-548 sample are exceptionally low. While periodontal disease has a multifactorial etiology, the primary agents are facultative pathogenic bacterial species, which if permitted to proliferate will initiate disease (Dentino et al., 2013). Suppression of these pathogenic bacteria is the key to preventing or controlling the disease process (Dentino et al., 2013). The primary etiological agents associated with periapical abscesses are also facultative pathogenic bacterial species (Siqueira and Rôças, 2009). Because the infection originates in the root canal system (Siqueira and Rôças, 2009), clinical treatment of periapical lesions requires mechanical debridement to gain access to the infected endodontic space followed by systemic antibiotic treatment (Dahlén, 2002). Therefore, pathogenic bacteria that proliferate in endodontic spaces are protected from antibacterial agents in the oral cavity. The bacterial suppression effect that would apply to caries and periodontitis therefore would not apply in the same way to risk of periapical lesions.

CONCLUSIONS

The results of this study show a population sample that exhibits extraordinarily low frequencies of carious lesions and periodontitis, high frequencies of periapical lesions, and remarkably high levels of dental wear. Wear was examined as a causative agent for the low prevalence of caries; however, statistical analysis shows a very weak association between the two. Statistical analysis does show a significant and strong correlation between wear and prevalence of periapical lesions. A more cogent explanation for the low prevalence of caries and periodontitis is provided by examination of the likely cause of the excessive dental wear rather than its extent. The individuals that comprise the CA-CCO-548 population sample exhibit an unusual wear pattern that is the likely result of non-dietary abrasion, specifically the manufacture of basketry. Materials used for Native Californian basketry contain naturally high levels of antimicrobial compounds. In addition, staple constituents of the documented dietary regime for this population also contain high levels of antimicrobial compounds. At least two of these antimicrobial compounds, salicylic and tannic acids, have been clinically documented to inhibit the primary bacterial species implicated in dental caries and periodontal disease.

This research offers a novel explanation for the remarkably low frequencies of dental caries that have been noted by virtually every bioarchaeological study of California Native American populations. The evidence presented here suggests an explanation integrating the current etiological explanations for infections of the oral cavity from the clinical literature, information from the ethnographic record pertaining to subsistence and activity patterns in Native Californian populations, and statistical analysis of specific disease and wear patterns in a large and temporally well-defined population sample. This study shows a correlation between lowered prevalence of caries and periodontitis, elevated level of dental wear and prevalence of periapical lesions, and cultural practices that introduced antibacterial compounds into the oral cavity.

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