A Reassessment of Dental Caries and Tooth Loss at CA-Ala-329

M.A. Thesis Proposal

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I. Abstract:

This study will reassess patterns of dental caries and antemortem tooth loss in a prehistoric population from CA-ALA-329, also known as the Ryan Mound. Previous studies at CA-ALA-329 use dental caries and antemortem tooth loss prevalence to make cross cultural comparisons and interpretations regarding diet, subsistence strategy and sexual division of labor (Bartelink 2006; Jurmain 1990). These studies relied primarily on the observation of inactive carious lesions, while assuming a direct, causal relationship between dental caries and antemortem tooth loss. Recent advancements in the understanding of dental caries and antemortem tooth loss etiology suggests the need to re-evaluate such methods.

Using data from CA-ALA-329, I will investigate the applicability and effectiveness of updated methodological approaches that incorporate inactive and active carious lesions. I will also investigate the relationship between dental caries and antemortem tooth loss by comparing patterns within the oral cavity. I hypothesize that this will result in new interpretations regarding diet, subsistence and sexual division of labor at CA-ALA-329. I also hypothesize that patterns of dental caries and antemortem tooth loss will be distinct, further supporting the argument for discrete etiology.

The results will expand upon the role of clinical research in bioarchaeological observation, analysis and interpretation. Providing evidence for what, if any, multi-disciplinary research integration is necessary for accurate and effective bioarchaeological research.

II. Introduction:

The purpose of this study is to reassess patterns of dental caries and antemortem tooth loss at CA-ALA-329, also known as the Ryan Mound. This study will apply an updated methodology for dental caries observation and analysis, while also investigating the relationship between dental caries and antemortem tooth loss. I will calculate dental caries and antemortem tooth loss prevalence by age, sex and time period. I will also compare intra-oral patterns dental caries and antemortem tooth loss. My results will be compared to results from previous studies at CA-ALA-329, conducted by Robert Juramin (1990) and Eric Bartelink (2006). This comparative analysis will provide insight into the importance of multi-disciplinary research in bioarchaeological investigations, specifically

the integration of recent clinical research in bioarchaeological methodology, analysis sand interpretation.

Oral pathology is an important topic in bioarchaeological research due to the fundamental role of human behavior in disease pathogenesis (Bignozzi et al. 2014; Dietrich et al. 2007). Therefore, bioarchaeologists have used oral disease, such as dental caries and antemortem tooth loss prevalence as evidence of certain behaviors in the past, such as dietary patterns (Cucina et al 2011; Lukacs and Largaespada 2006), food processing techniques (Griffin 2014; Lanfranco and Eggers 2010) and subsistence strategies (Array-de-la-Rosa et al 2009; Cuellar 2013; Fabra and Gonzalez 2015; Gagnon and Wiesen 2013; Rick 2011; Scopacasa 2014; Mayes 2015; Walker and Erlandson 1986).

More recent bioarchaeological investigations have attempted to take this interpretation a step further, using dental caries and antemortem tooth loss prevalence as evidence for differential access to resources based on sex and social status (Cucina et al. 2011; Cucina and Tiesler 2003; Graham and Burkart 1976; Lukacs 2011; Novak 2015; Scopacasa 2014), sexual division of labor (Lanfranco and Eggers 2010; Mayes 2015; Novak 2015;) and environmental adaptations (Cucina et al. 2011; Kennett et al 2007; Pilloud 2006; Temple 2015; Walker and Erlandson 1986; Weiss 2002).

Two important studies looking at dental pathology at CA-ALA-329 include Robert Jurmain (1990) and Eric Bartelink (2006). CA-ALA-329 is a prehistoric site in the San Francisco Bay Area and offers a unique opportunity to investigate oral pathology due to its well preserved skeletal and archaeological material (Leventhal 1993). Research by Robert Jurmain (1990) and Eric Bartelink (2006) calculated dental caries and antemortem

tooth loss prevalence by age, sex and time period. The resulting interpretations made conclusions regarding disease interaction, dietary patterns and subsistence strategies at CA-ALA-329 (Bartelink 2006; Jurmain 1990)

This bioarchaeological research using dental caries and antemortem tooth loss to interpret the past relies on accurate bioarchaeological methods of observation and interpretation. Methods that are built on the accurate integration of clinical research. Yet, not only is methodological standardization rare for dental caries and antemortem tooth loss observation and interpretation (Hillson 2001), often the integration of current clinical research is delayed. This means that many bioarchaeological studies continue to use methods of observation and interpretation based on out dated clinical research. This suggests the need to re-assess past research. Therefore, I will investigate how this delay in integration impacts bioarchaeological research.

Using data collected using a new protocol (Griffin 2014), I will analyze dental caries and antemortem tooth loss at CA-Ala-329. My results and interpretations will then be compared to Robert Jurmain's (1990) and Eric Bartelink's (2006) findings. This comparison will indicate how slight changes in observational methodology and analysis impact bioarchaeological interpretations. I will also critically evaluate the applicability of the 'caries correction factor' method used by Bartelink (2006) and introduced by Lukacs (1995). This method was commonly used in bioarchaeological research (Bartelink 2006; Lukacs 1992, 1996) and can dramatically impact statistical results and interpretation. Therefore, this research will ultimately investigate the role and importance of clinical research in bioarchaeological investigations.

I hypothesize that due to changes in observational methodology, my calculated dental caries prevalence will vary when compared to Jurmain (1990) and Bartelink (2006). While the rates of antemortem tooth loss will likely remain the same, I hypothesize that the interpretations will vary due to increased etiological understanding. I also hypothesize that the 'caries correction factor' method is no longer applicable in bioarchaeological research. If this is found to be true, then previous studies will need to be re-visited. I also hypothesize that recent advances in clinical research will offer new interpretative avenues when studying dental caries and antemortem tooth loss in past populations; this including behavioral, biological and environmental factors. Ultimately, this these will expand upon the role of clinical research and a multi-disciplinary approach in bioarchaeological research.

III. Background:

Dental caries and antemortem tooth loss are both complex processes that occur in the oral cavity. In order for bioarchaeological research to accurately interpret disease prevalence, a comprehensive understanding of disease etiology is necessary. Dental caries is an infectious disease found in the oral cavity. This disease is caused by an imbalance of microflora, resulting in an increase in pathogenic bacteria (Featherstone 2004). The increase in pathogenic bacteria causes increased acidification and eventual enamel demineralization (Featherstone 2004; Selwitz, Ismail and Pitts 2007; Takahashi and Nyvad 2011). At any given time, there are over 20,000 microorganisms in the oral cavity, including over 700 species of bacteria (Bjarnsholt 2013; Zaura et al. 2009). Initially, these bacteria are platonic, single celled and independent (Bjarnsholt 2013).

These bacteria can occur naturally in the oral cavity, or can be introduced through the consumption food (Zaura et al. 2009).

Eventually, these bacteria form bacterial aggregates with additional micro-organisms to form oral biofilm (Bajarnsholt 2013; Hoiby 2014). The official definition of oral biofilm is a microbial community composed of bacteria, fungi, proteins, acids and poolysaccharidges (Lee and Tan 2014; Palmer 2014; Selwitz, Ismail, Pitts 2007). This oral biofilm plays a fundamental role in dental caries pathogenesis. (Allison and Gilbert 1995; Bjarnsholt 2013; Jenkinson 2011; Palmer 2014; Wilson 2005).

Before oral biofilm research, it was often assumed that dental caries was caused by specific, extremely cariogenic bacteria. The most well-known cariogenic bacteria include *Streptococci (*Merrit and Qi 2009) and *Lactobacilli* (Byun et al. 2004; Caufield 2015), which are both aciduric and acidogenic bacterium (Ding et al. 2014; Takahashi and Nyvad 2008; Caufield et al. 2015). This means they have the capacity survive increasingly acidic biofilm environments while contributing to this acidification by metabolizing carbohydrates into acidic waste (Dame-Texier et al. 2016; Ding et al. 2014; Hujoel 2009; Takahashi and Nyvad 2008; Merrit and Qi 2009). The importance of carbohydrate consumption and dental caries formation in bioarchaeological research was provided by this key interaction.

With the introduction of oral biofilm research, the importance of microbial interaction and biofilm ecology took precedence over independent bacterial studies. While the role of *Streptococci* and *Lactobacilli* remained important in dental caries formation, it was no longer the actions of independent bacteria that resulted in enamel demineralization. Instead, it was the interaction between the bacteria, fungi and proteins that cause

environmental change, acidification, and enamel demineralization. This interaction includes competition, inter-dependence, genetic transfer and bacterial suicide and altruism (Jenkinson 2001; Kreth, Merrit and Qi 2009).

There are multiple behavioral and biological factors that contribute to the increase in pathogenic and decrease in non-pathogenic bacteria (Bignozzi et al. 2014; Dietrich et al. 2007; Bjarnsholt 2013; Kreth, Merritt and Qi 2009; Selwitz, Ismail and Pitts 2007). Important behavioral factors include diet, consumption patterns, medication use, dental hygiene and smoking habits (Bignozzi et al. 2014; Dietrich et al. 2007). Biological considerations include genetic predisposition affecting salivary flow, salivary composition, enamel thickness and tooth eruption (Bjarnsholt 2013; Kreth, Merritt, and Qi 2009; Bignozzi et al. 2014; Selwitz, Ismail and Pitts 2007). Additional biological processes can affect salivary flow and composition, including sex hormones and the process of aging (Shaffer et al. 2015; Silveira et al. 2016). Therefore, dental caries pathogenesis goes beyond merely carbohydrate consumption and offers opportunity for expanded bioarchaeological interpretations.

The physical manifestation of dental caries is a carious lesion (Selwitz 2007). This can occur on the crown or root of the tooth (Emilson, Klock and Sanford 1988; Hillson 2001). There are multiple stages of carious lesion formation, ranging from initial discoloration to late stage pit formation. Depending on the activity of the carious lesion, the texture of the lesion can be soft and rough to the touch, or hard and smooth (Hillson 2001; Featherstone 2004; Fejerskov 2004). This is because carious lesion formation is not a linear process, in fact, demineralization is often interrupted by periods of inactivity and remineralization (Cordoso 2016; Hujoel 2013; Moynihan 2005).

Identifying the multi-stage, reversible process of carious lesion formation is difficult in the bioarchaeological record due to postmortem techonomic changes (Hilson 2001). Therefore, bioarchaeologists are often conservative in carious lesion observation, only counting a carious lesion as present if there is a definable pit with a rough or sticky texture (Hilson 2001; Buikstra and Ubelaker 1994). Therefore, bioarchaeological investigations predominately rely on the observation of late stage, active carious lesions (Bartelink 2006; Hillson 2001; Griffin 2014; Temple and Larson 2007). Many of the bioarchaeological methods acknowledge that this dramatically reduces dental caries prevalence, but to do otherwise would inaccurately attribute postmortem taphonomic changes to an increase dental caries prevalence (Buikstra and Ubelaker 1994; Wasterlain et al. 2009).

Over the past decade, clinical studies have focused on the process of demineralization and remineralization (Burwell et al. 2012; Deyhle et al. 2013; Featherstone 2004; Hujoel 2013; Iwami et al. 2008; Karayasheva et al. 2016; Nyvad and Fejerskov 1997; Moynihan 2005; Shaffer et al. 2015; Vieira et al. 2014). This has resulted in a more comprehensive understanding of carious lesion formation. This in conjunction with unique intra-oral patterns of dental caries prevalence and susceptibility, offers an opportunity to better observe carious lesions in the archaeological record (Burwell et al. 2012; Iwami et al. 2008). The data that I will use in this study applied such a methodology, allowing observation of both active and inactive carious lesions in multiple stages of formation. While isolated discoloration on tooth enamel remaines difficult to identify as a carious lesion, this updated methodology allows observation of early pit lesion formation, inactive carious lesions, as well as lesions on teeth with very little enamel.

Similar to dental caries, antemortem tooth loss is a multi-factorial disease process that occurs in the oral cavity. Unlike dental caries, antemortem tooth loss is often attributed to other pathological conditions (Baelum and Fejerskov 1986; Bignozzi et al. 2014; Buckwald et al 2013; Gilmore 2013; Olsson 1978; Wu et al. 2016). This includes dental caries, periodontal disease, extreme wear and attrition as well as trauma. Similar to Jurmain (1990), multiple bioarchaeological investigations have attributed antemortem tooth loss to either periodontal disease or dental caries (Bartelink 2007; Hillson 2001; Lukacs 1992, 1995, 1996, 2007). Yet, the current clinical research suggests a more complex etiology.

Initially, the loss of a tooth is due to the breakdown of periodontal tissues, including the structures of the tooth, periodontal ligaments, gingiva and alveolar bone (Ali et al. 2011; Bath-Balogh 1997; Kandray 2007). This breakdown is associated with multiple inflammatory conditions. While this includes the introduction of inflammatory bacteria associated with dental caries and periodontal disease (Baelum and Bignozzi et al. 2014; Buckwald et al. 2013; Fejerskov 1986; Wu et al. 2016), it also includes inflammation caused by systemic inflammatory diseases such as heart disease, obesity and diabetes (Bole et al. 2010; Buckwalt et al. 2013; Muluke et al. 2016). All of these conditions cause a general inflammatory response throughout the body that affects the periodontal tissues.

There are behavioral and biological processes that impact this inflammatory response (Bole et al. 2010; Kim et al. 2015; Muluke et al. 2016; Tak et al. 2014). Behavioral considerations include those associated with dental caries and periodontal disease pathogenesis, such as diet, food consumption patterns, hygiene and medication use (Ali et al. 2002; Bignozzi et al. 2014; Delima et al. 2002; Dietrich et al. 2007). Biological

processes that influence antemortem tooth loss include genetic predisposition regarding inflammatory response and hormone levels associated with sex and aging (Bole et al. 2010; Kim et al. 2015; Muluke et al 2016; Tak et al. 2014). Additionally, individuals can also be predisposed to quicker and more severe inflammation in the periodontal tissues, which directly affects antemortem tooth loss susceptibility (Giannobile 2010). Therefore, much like dental caries, antemortem tooth loss is more than just a process that results from periodontal disease or dental caries.

Unfortunately, very little research has been conducted that looks at the interaction between dental caries and antemortem tooth loss. Antemortem tooth loss in modern populations is usually caused by the physical extraction of the tooth. Therefore, even fewer studies have been conducted looking at how severe carious lesions naturally results in antemortem tooth loss. The initial literature review suggests a differential etiology, and a weak association between dental caries and antemortem tooth loss without tooth extraction. This brings the 'caries correction factor' method, used by multiple bioarchaeologists into question (Bartelink 2006; Lukacs 1995, 1996). The applicability of the caries correction factor method and those similar will therefore be a main topic of discussion and research in this research project. Through the comparison of intro-oral patterns of disease pathogeneses and etiological research, I will investigation such analytical bioarchaeological methodology.

The analysis of dental caries and antemortem tooth loss will look at a skeletal collection from CA-ALA-329, also known as the Ryan Mound. CA-ALA-329 is a large earthen mound site located in the southeastern Bay Area (Coberly 1973; Leventhal 1993). It is one of four mound sites that make up the Coyote Hills Complex, including CA-

ALA-12, CA-ALA-13 and CA-ALA-328 (Bickel 1976; Leventhal 1993). CA-ALA-329 dates to the end of the Middle Period (200BC-AD900) through the Late Period Phase II (AD1500-AD1769) (Leventhal 1993), and is located in an environmentally diverse area that includes prairie grasslands, tidal marshes and mudflats (Hylkema 2002).

Nels Neslon first located and documented CA-ALA-329 in 1909 (Nelson 1909). Since then, multiple phases of excavation have taken place at CA-ALA-329 from the 1940's to the 1960's (Coberly 1973; Wilson 1993). Over these multiple field seasons, a total of 427 burials were excavated (Leventhal 1993). While a portion of these remains have been repatriated with the Ohlone Muwekma, 176 individuals with permanent dentition were available for this research.

CA-ALA-329 is culturally associated with the Muwekma Ohlone tribe. Kroeber (1925) explained that the Muwekma Ohlone people were part of a broader Costanoan tribe. This tribe extended from the South San Francisco Bay to the Monterey Coast (Bocek 1990) and were comprised of multiple tribelets (Bocek 1990; Kroeber 1925; Milliken 1991). The cultural practices associated with the Ohlone tribelets, including diet, subsistence strategies and ceremonial practices temporally and geographically distance. Therefore, the archaeology at CA-ALA-329 provides unique insight into the past lifeways specifically at CA-ALA-329, while also offering comparative data for the greater San Francisco Bay archaeology.

Due to the well preserved skeletal and archaeological remains at CA-ALA-329, multiple archaeological and bioarchaeological investigations have expanded our understanding of life at CA-ALA-329 from the Middle Period to the Late Period Phase I. This includes investigations into increased violence due to resource competition (Jurmain

2001; Jurmain and Bellifemine 1997; Jurmain et al. 2009), disease prevalence (Atwood 2008; Blake 2010; Jurmain 1990; Necheyev 2007), and the role of CA-ALA-329 as a ceremonial center (Leventhal 1993). CA-ALA-329 is also important in understanding San Francisco Bay Area and California archaeology as a whole (Bartelink 2006; Lightfoot and Luby 2002).

A common research topic includes the impact of a climatic event, known as the Medieval Climatic Anomaly (MCA), that occurred AD700-1100 (D'Oro 2009; Leventhal 1993; Lightfoot and Luby 2002;) Studies looking at the impact of the MCA in the San Francisco Bay have varied; some argue that the Bay remained largely unaffected by the MCA due to the diversity of resources (Bartelink 2006, 2009), while others argue that skeletal and archaeological remains suggest the MCA was characterized by increased violence and seasonal movement (Leventhal 1993; Lightfoot and Luby 2002; D'Oro 2009). This research has been important in the greater discussion of shell mound use in the Bay Area (Leveanthal 1993; Lightfoot and Luby 2002), as well as behavioral changes due to climate change (D'Oro 2009; Hyklema 2002). These discussions are ongoing in San Francisco archaeology today (Bryne and Byrd 2009; Whitaker, Byrd and Darcangelo 2013).

Dental pathology is an important element in this research. Robert Jurmain (1990) and Eric Bartelink (2006) analyzed oral pathology, including the observation and analysis of dental caries and antemortem tooth loss analysis at CA-ALA-329. Jurmain (1990) and Bartelink (2006) compared disease prevalence by age, sex and time period, while also looking at differential tooth involvement and intra-oral patterns of disease pathogenesis. Jurmain's (1990) research focused on collecting data on all oral pathology at CA-ALA-

329, including attrition, dental caries, periodontal disease, abscesses and antemortem tooth loss. While the focus of his research and interpretations looked at disease interaction, he did suggest that the low rates of dental caries suggests a hunter-gatherer diet over and agricultural diet (Juramin 1990).

Jurmain suggested a hunter-gatherer lifeway due to the extremely low rate of dental caries (Jurmain 1990). Jurmain only observed 21 carious lesions on 10 individuals, all of which were under the age of thirty. This is an extremely low rate of dental caries, even for a coastal population. He also found a moderate prevalence of antemortem tooth loss and attributed this loss primarily to periodontal disease. Unfortunately, his methods for observation of dental caries and antemortem tooth loss were not explicitly stated, but he did provide percentages by age, sex and time period, as well as the tooth elements disproportionately affected (Jurmain 1990).

Eric Bartelink's research was conducted on a different curated skeletal population than Jurmain (1990). Similar to Jurmain (1990), Bartelink (2006) observed low dental caries prevalence at CA-ALA-329. Bartelink compares these findings with other coastal California populations that also have low rates of dental caries and antemortem tooth loss, such as Santa Barbara Island (Walker and Erlandson 1986). Bartelink (2006) also found different rates of dental caries and antemortem tooth loss between males and females, age, and time period. He uses these results as evidence for differential sex based diets, and change in subsistence strategy through time. Ultimately, Bartelink (2006) argues that people at CA-ALA-329 likely consumed a higher percentage of marine resources when compared to terrestrial plants, but that this trend changed through time

due to environmental changes and drought-like conditions in the bay (Bartelink 2006; Bartelink 2009).

The observational and analytical methods were explicit in Bartelink's research (2006). He used Buikstra and Ubelakers (1994) *Standards for Data Collection* for examination and data collection on dental caries and antemortem tooth loss. Dental caries was marked as present only if there was a minimum of 2mm enamel on 50% of the crown. Bartelink used Hillson's (2001) method for carious lesion observation. Based on Hillson (2001) a carious lesion is only present if there is a definable pit with noticeable textural changes when using a dental probe. In addition to observational methods, Bartelink also explained that a key method is his analysis was the application of the 'caries correction factor', which was first introduced by Lukacs (1995). The 'caries correction factor' estimates the number of antemortem tooth loss due to different pathological conditions. The differences in dental caries and antemortem tooth loss prevalence were only statistically significant when the 'caries correction factor' method was applied (Bartelink 2006).

The use of dental caries and antemortem tooth loss as evidence of dietary patterns, subsistence strategy and differential access to resources is based on a clinical understanding that emphases late-stage carious lesion observation and carbohydrate consumption (Hillson 2001; Hujoel 2009). Recent clinical research suggests that the etiological processes associated with dental caries pathogenesis, carious lesion formation and antemortem tooth loss is more complex (Abdulbaqi, Himratul-Aznita and Baharuddin 2016; Hujoel 2009; Schroth et al. 2014; Takahashi and Nyvad 2008; Wilson 2005). Dental caries and antemortem tooth loss are multi-factorial diseases that occur in the oral cavity, where biological, behavioral and the environmental processes interact

(Avlund et al. 2003; Baelum and Fejerskov 1986; Hillson 2001). Therefore, the observation, analysis and interpretation of dental pathology in the bioarchaeological record should incorporate this complexity.

IV. Methods:

The data for this study were collected by the author and Dr. Mark Griffin during the summer of 2016. Each dentition was examined macroscopoically and a dental inventory was collected. This includes the presence or absence of dental caries and antemortem tooth loss. Collecting this information by dental element allows for more flexibility during analysis (Hillson 2001; Buikstra and Ubelaker 1994). The methods used for dental caries observation combine previously established bio archaeological and clinical methods for dental caries observation. The earliest bioarchaeological method was provided by Corbett and Moore (1976), which calculates caries frequency by tooth type and by the total number of surfaces susceptible to carious lesion formation. Buikstra and Ubelaker (1994) expanded this to include additional data regarding non-observable dentition, lesion location and lesion size. Frequency was therefore calculated by dividing total diseased teeth by total number of observable teeth. Buikstra and Ubelaker's (1994) method remains the most used methodology for dental inventory and dental caries observation in the bioarchaeological record.

There are three main methods for dental caries identification in the clinical literature; Nyvads 6-stage system (Nyvad, Machiulskiene and Baelum 1999; Nyvad and Fejerskov 1997) and the World Health Organizations standardization IICADS-II and IICADS-LAA (Ismail et al. 2007). Each method is distinct and applies different clinical research

looking at carious lesion formation. Nyvad's 6-stage system (NY) provides classification for inactive carious lesions, but is limited to three carious lesions stages (Nyvad, Machiulskiene and Baelum 1999). The ICDAS-II provides more lesion formation stages, but lacks identification possibilities for active versus inactive carious lesions (Braga et al. 2016; Iwami et al. 2008). Finally, ICDAS-LAA provides multiple stages of lesions formation, while also accounting for activity status (Braga et al. 2016; Ismail et al. 2007). Yet, ICDAS-LAA applies a complex, numbering system that also requires extensive training and practice and can be difficult to fully integrate into bioarchaeological analysis. While none of the clinical methods were directly used in data collection at CA-ALA-329, methods and understanding required for the above methods was integrated into the detection of inactive and active carious lesions.

A combination of the above methodologies was used in the collection and analysis of dental caries at Ca-ALA-329. Based on Buikstra and Ubelaker (1994) methods, both the location on the tooth and size of the lesion were recorded. Dental caries was considered present if at least one carious lesion was noted on the tooth. Based on the clinical methods, both inactive and active carious lesions were included in the analysis. Previously, a carious lesion was only identified as present if there was an identifiable pit with textural changes (Hillson 2001; Buikstra and Ubelaker 1994). Due to the integration of both active and inactive carious lesions in this analysis provided by recent clinical research (Afroughi et al. 2010; Burwell et al. 2012; Iwami et al. 2008), a carious lesion was identified as present if there was a definable pit, with or without textural changes. If textural changes were absent, additional evidence of demineralization or mineralization

was required. This includes discoloration within or surrounding the pit, as well as color or textural changes in adjacent teeth.

Antemortem tooth loss was considered present only if there was evidence of alveolar healing. This can range from early stage inflammatory response that results in the placement of woven bone, or late stage bone loss and morphological changes to the alveolar bone. It can be difficult at times to distinguish between postmortem and antemortem tooth loss, where evidence of healing is crucial. Early stage woven bone placement results in a porous look inside the tooth socket and around the enamel cementum juncture. Once time has passed and bone resorption and remodeling takes place, horizontal and vertical reduction of the alveolar is also observed. If woven bone and alveolar remodeling is absent, it was counted as postmortem tooth loss.

While the data collected by Mark Griffin includes the location and size of the carious lesion, this information will not be used in my analysis. However, due to the importance of sex and age in dental caries and antemortem tooth loss analysis, methods for sex and age estimation are also important. Sex was determined using a combination of methods to identify morphological changes to the ilium caused by the formation of the birth canal (Phenice 1969; Buikstra and Ubelaker 1994). While the ilium remains the most accurate source for sex determination (Ubelaker 2002), if the ilium was damaged or missing, methods observing morphological changes to the skull were used for sex determination (Buikstra and Ubelaker 1994). Sex was recorded as indeterminate, female or male.

Age at death was estimated and recorded in years using methods that observe morphological changes on the auricular surface of the ilium (Lovejoy et al. 1985) and pubic symphyseal face metamorphosis (Todd 1920; Meindle et al. 1985). If the auricular

surface or public symphysis was damaged, supplementary methods for age estimation included epiphyseal union (Krogman and Iscan 1986) and dental eruption (Ubelaker 1989) to distinguish between sub-adult, adolescent and adult. I will initially bin the population into 10 year age categories, but this may be adjusted based on sample size and statistical analysis.

Due to high rates of attrition and tooth loss, I will pool all left with right dentition for each individual, to aid in the statistical analysis and comparison for CA-ALA-329 (Bartelink 2006; Hillson 2001). I will then calculate frequencies for both dental caries and antemormtem tooth loss. Dental caries frequency will be calculated by dividing the total number of diseased teeth by the total number of observed teeth. While antemortem tooth loss will be calculated by dividing the total number of teeth lost antemortem by total number of sockets. These frequencies will be calculated for all dentition as well as for appropriate dental subgroups (Table 1) Due to differential susceptibility, it is common for bioarchaeological research to separate anterior and posterior teeth, or maxillary and mandibular dentition during analysis (Cucina et al. 2011; Griffin 2014; Turner 2015; Temple 2011; Sheihem and Sabbah 2010). Calculating prevalence by similar subgroups will allow for increased research comparability (Härkänen et al., 2002; Hillson 2001; Sheiham and Sabbah 2009).

The frequency results for both dental caries and antemortem tooth loss will then be compared by sex, age group and time period using statistical analysis. Since this data is categorical in nature, only chi-square tests for independence are appropriate. Using SPSS, I will test for association between dental caries and antemortem tooth loss, sex, age and time period. I will also use chi-square tests of independence to test for association

between dental caries and antemortem tooth loss sub-groups. This will test for statistically different intra-oral patterns of disease pathogenesis. If the Pearson's two-tailed t-test is less than p=0.05, the test will be considered significant. A post-hoc test will then be run and residuals with greater than 1.96, the critical value for a two-tailed analysis with p=0.05, will be counted as statistically significant. Fishers exact tests will be used if and when the sample size falls below 5 for 25% of the categories.

Using loglinear analysis, I will also test for levels of association between three categorical variables simultaneously. This includes dental caries, sex and time period. This will be completed for both antemortem tooth loss and dental caries and can provide information on association if chi-square and fishers exact tests do not show any statistically significant interactions.

Finally, I will the compare all results and associated interpretations with previous investigations at CA-ALA-329. This comparative analysis will provide insight into how the integration of current clinical research and the associated changes in bioarchaeological methodology impacts bioarchaeological investigations.

Table 1.
Dental Subgroup Categories for Analysis

Dental Subgroups	**Dentition Included**
Anterior Maxillary	(URC, URI2, URI1)
Anterior Mandibular	(LRC, LRI2, LRI1)
Posterior Maxillary	(URP3, URP4, URM1, URM2, URM3)
Posterior Mandibular	(LRP3, LRP4, LRM1, LRM2, LRM3)
Dental Element	LRM1
	LRM2
	LRM3
	URM1
	URM2
	URM1

V. Expected Findings:

I expect to find that my results differ from previous studies due to the change in methodology that incorporates active and inactive carious lesions. I also expect to find that the intra-oral patterns of dental caries and antemortem tooth loss will be distinct due to differential etiology. For the population specific results, I expect that there will be statistically significant differences in dental caries and antemortem tooth loss between males and females. I also expect to find statistically significant differences in prevalence in both dental caries and antemortem tooth loss by age group due to changes associated with hormones, saliva and bone mineral density as individuals age. I also hypothesize that there will be statistically significant changes in prevalence through time due to changes in diet and subsistence strategy associated with climatic changes at CA-ALA-329. All of my research hypotheses are presented in Table 2.

Therefore, my null hypotheses include the following: that there will be no difference in interpretive results due to change in methodology, no statistical differences in the intra-oral patterns of dental caries or antemortem tooth loss, no statistically significant differences in dental caries and antemortem tooth loss prevalence between the dental subgroups, biological sex, age or time period. All null hypotheses are listed below in Table 3.

Table 2.

	Research Hypotheses
1	I hypothesize that my results will differ from previous studies due to a change in observational methodology.
2	I hypothesize that the intra-oral patterns of dental caries and antemortem tooth loss will be distinct due to differential etiology.
3	I hypothesize that there will be statistically significant differences in dental caries and antemortem tooth loss prevalence between the different dental subgroups.
4	I hypothesize that there will be statistically significant differences in dental caries and antemortem tooth loss prevalence between males and females, suggesting an increased susceptibility for females.
5	I hypothesize that there will be statistically significant differences in prevalence of dental caries and antemortem tooth loss between age categories, suggesting an increase in susceptibility with an increase in age.
6	I hypothesize that there will be a statistically significant change in prevalence through time (MP, LP1, LP2)

Table 3.

	Null Hypotheses
1	There will be no statistically significant differences between dental subgroups.
2	There will be no statistically significant differences between biological sex, age or time period.
3	There will be no difference in interpretation due to a change in methodological approach.
4	There will be no difference between the intra-oral patterns of dental caries and antemortem tooth loss

VI. Schedule:

I plan to start writing my thesis during the Fall of 2017, focusing first on the literature review and methods section, which I have already researched. I will then simultaneously complete data analysis and interpretation from November to December 2017. I plan to finish the final sections of my thesis at the beginning of 2018, focusing on edits and corrections over the Spring 2018 semester. I will finish my thesis by April 2018, with final submittal in May 2018.

VII. Committee:

Mark Griffin – Major Advisor
Cynthia Wilczak – 2nd reader

Literature Cited

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